Skin Manifestations in Response to Local Pressure and Microclimate in Healthy Adults

Submitted by

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Statement of Authorship

This thesis consists primarily of work by the author that has been published as described in the text. Except where reference has been made in the text of the thesis, this thesis contains no other material published elsewhere or extracted in whole or in part for a thesis submitted for the award of any other degree or diploma. No other person's work has been used without due acknowledgement in the main text of the thesis. This thesis has not been submitted for the award of any degree or diploma in any other tertiary institution.

Signed:

Caroline Jacqueline Borzdynski

Date: 28th September 2022

In memory of my Grandmother, Zofia Sworobowicz

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Publications by the Candidate relevant to this Thesis

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Statement of Authorship and Contribution for Article

Caroline Borzdynski	Overall contribution 85% Conception and design of the study Analysis and interpretation of the data Drafted the manuscript Contribution of knowledge
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Associate Prof. Bill McGuiness	Overall contribution 5% Conception and design of the study Made some critical revisions to the manuscript Contribution of knowledge

Ethics Approval

Ethical approval for the research undertaken in this thesis was obtained from two separate applications to the La Trobe University Human Ethics committee (College of Science, Health and Engineering) prior to the recruitment of study participants; ethics approval numbers HEC-18467 (Appendix A) and HEC-20300 (Appendix B). All participants involved in this research initially reviewed the participant information statements (Appendices C and D) and provided informed written consent.

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Abbreviations

ACSQHC: Australian Commission on Safety and Quality in Health Care
AIHW: Australian Institute of Health and Welfare
BMI: Body Mass Index
COF: Coefficient of Friction
DFU: Diabetic Foot Ulcer
DTI: Deep Tissue Injury
EPUAP: European Pressure Ulcer Advisory Panel
HAPI: Hospital-acquired Pressure Injury
ICC: Intraclass Correlation Coefficient
LMM: Linear Mixed Model
NICE: National Institute for Health and Care Excellence
NPUAP: National Pressure Ulcer Advisory Panel
NRAS: Norton Risk Assessment Scale
NSQHS: National Safety and Quality Health Service
PI: Pressure Injury
PPA: Pressure Prone Area
PPPIA: Pan-Pacific Pressure Injury Alliance

PUPPS: Pressure Ulcer Point Prevalence Survey

QOL: Quality of Life

RAS: Risk Assessment Scale

TEWL: Trans-epidermal Water Loss

VLU: Venous Leg Ulcer

VSA: Visual Skin Assessment

WHO: World Health Organization

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Abstract

Background

The skin microclimate has been theorised as an indirect contributor to pressure injury (PI) development. Several disruptions to the skin microclimate can be reflected by manifestations at the skin surface. As such, an appraisal of the skin at pressure-prone areas of the body represents a focal point of PI risk assessment. To date, contemporary methods of skin appraisal used in PI risk assessment are subjective and fraught with challenges. Biophysical instruments—that have the capacity to quantitatively appraise skin properties associated with PI development—warrant exploration for their application in PI risk assessment. Limited in-vivo research has examined how biophysical skin properties change over time under different skin microclimate conditions and in response to brief intermittent off-loading of pressure-prone areas of the body.

Aim

The primary aim of this research was to examine the skin microclimate effects on clinical manifestations often attributed to PI development in healthy adults. To address this aim, a program of research was undertaken in two phases and involved three inter-related studies. The first research phase sought to examine skin parameter changes at common pressure-prone areas during intermittent pressure off-loading every 10 minutes for 60 minutes, and during continuous pressure-loading for 60 minutes. The second research phase sought to assess for skin parameter changes in response to heat and moisture introduced at the skin-support surface interface.

Method

Three studies were undertaken. Studies I and II employed a descriptive-correlational design. Non-invasive biophysical measures of erythema, melanin, stratum corneum hydration and skin temperature were obtained at the elbows, heels and sacrum of healthy adults. Measurements were taken comparing results before and after 60 minutes in two conditions; (a) without pressure off-loading, and (b) with pressure off-loading every 10 minutes to enable measurement. Measures were gathered while participants lay supine and then semi-recumbent. Study III was a quasi-experimental study. Participants advanced through three conditions; (a) controlled condition, (b) heat intervention, and (c) moisture intervention. In the heat intervention, participants lay on a heated blanket. In the moisture intervention,

participants' sacral skin was exposed to a pad soaked with normal saline. All the anatomical testing sites were off-loaded briefly to enable each measurement. Between- and within-subject comparisons with repeated measures were performed, yielding both descriptive and inferential data. A 15 minute washout interval ensured sufficient time for skin parameters to return to baseline after the loading periods.

Data Analyses

In Studies I and II, intraclass correlation coefficients (ICC) were calculated to indicate test-retest reliability and absolute agreement of results between the two methods (brief intermittent off-loading versus continuous pressure-loading). Analyses were performed using the IBM Statistical Package for the Social Sciences (SPSS) Statistics (Version 25.0; IBM Corp., 2017) and STATA Statistical Software: Release 13 (StataCorp, 2013). In Study III, linear mixed models (LMMs) were used to compare skin parameters, over time, and between control and intervention (heat and moisture) conditions. All LMM analyses were performed using IBM SPSS Statistics (Version 25.0; IBM Corp., 2017).

Results

The study samples were comprised predominately of females (68.3% in Studies I and II, and 70.8% in Study III), with a mean age of 27.9 years (*SD*=10.6) (Studies I and II) and 26.8 years (*SD*=9.5) (Study III). In all studies, large regional variation in skin measures emerged by condition and between participants. Studies I and II (n=41) detected no significant differences in skin parameter scores between the two methods. Strong agreement between the methods was observed, with ICCs ranging from 0.72 to 0.99 (supine) and 0.62–0.99 (semi-recumbent). As such, the method of data capture of skin parameters was deemed methodologically feasible and valid for Study III. Study III (n=48) identified that heat increased skin temperature at the heels ($p<.001$), elbows ($p=.005$) and sacrum ($p<.001$) and erythema at the sacrum ($p=.012$). The introduction of moisture at the sacrum significantly increased sacral stratum corneum hydration ($p<.001$). In none of the studies did any participant display non-blanching erythema as a subjective indicator of early skin injury.

Discussion and Implications for Clinical Practice

This thesis aimed to inform the understanding of how the controlled manipulation of skin microclimate conditions impacts measures of erythema, melanin, stratum corneum hydration and skin temperature at the skin overlying the bony prominences of the body in semi-recumbent healthy adults. The preliminary research phase tested the method of data

collection, which informed the feasibility and protocol of Study III. The results indicated that brief off-loading (every 10 minutes) to enable skin parameter measurement does not alter skin measures. Thus, when translating these findings into clinical practice, implementing a 10 minute off-loading regime for healthy patients lying continuously in the supine and semi-recumbent positions would not appear to clinically alter PI risk as indicated by changes in skin colour and skin microclimate properties.

Anatomical site variations in skin parameters may warrant customised assessment of skin condition. More frequent monitoring cycles for erythematous discolouration, moisture and thermal changes may be required at the sacral skin. Distinctive protocols in regard to skin-care regimes may be required for individual patients. This research has also strengthened the evidence supporting biophysical skin appraisal as a possible adjunct to current PI risk assessment methods employed within clinical practice. Overall, the findings of this thesis suggest clinical feasibility of biophysical skin monitoring as a detection system of important cutaneous changes manifested at pressure-prone areas of the body.

CHAPTER 1
Introduction

1.1 Chapter Overview

This chapter provides an outline of the background information relevant to the impetus for this thesis and an overview of the associated research studies. The chapter begins with a broad introduction to pressure injuries (PIs) and outlines their significance within healthcare. To frame the research in this thesis appropriately, a brief overview of the concept of skin microclimate, as a theoretical contributor to PI development, is provided. The chapter then considers current PI risk assessment guidelines pertaining to skin appraisal, and presents the notion of biophysical skin appraisal as a novel method that could enhance current skin appraisal methods. The rationale, purpose and significance of this research are then provided. The chapter concludes by outlining the thesis structure.

1.2 An Introduction to Pressure Injuries and their Significance

A pressure injury (PI)[1] (also referred to as a bed sore, pressure sore, pressure ulcer, or decubitus ulcer) is defined by the National Pressure Ulcer Advisory Panel (NPUAP) as a "localized injury to the skin and/or underlying tissue usually over a bony prominence, as a result of pressure, or pressure in combination with shear" (NPUAP 2019, p 12). PIs have a long-standing history of being recognised as debilitating chronic wounds (Reuler & Cooney, 1981). These wounds have a long history, with evidence of pressure ulceration found in Egyptian mummies, some dating back 5000 years (Agrawal & Chauhan, 2012). The affliction of PIs has continued to be evidenced throughout the millennia. In the 19th century, Jean-Martin Charcot, a French neurologist and professor of anatomical pathology, described a PI as a catastrophic occurrence, inevitably leading to infection and death (Levine, 2005). To date, the burden of PIs remains substantial and continues to constitute a significant global health concern (Bredesen et al., 2015; Chaboyer et al., 2018; Gunningberg et al., 2019; Miles et al., 2013).

PIs have been frequently reported to be detrimental to the quality of life of those affected (Charalambous, 2018; Gorecki et al., 2014; Thein et al., 2010) whilst imposing a

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[1] In 2012, the Pan-Pacific Pressure Injury Alliance (PPPIA)—representing healthcare contexts across the Australasian region—adopted the term *pressure injury* for sole use (AWMA, 2011). However, *pressure ulcer* and *pressure sore* continue to be used synonymously in the international literature (NPUAP, 2019).

substantial physical and emotional burden (Galhardo et al., 2010; Nguyen, Chaboyer & Whitty, 2015) and causing prolonged pain and social isolation (Gorecki et al., 2009; Latimer, Chaboyer & Gillespie, 2014). Additionally, many non-healing PIs have the propensity to result in life-threatening infections (Bodavula et al., 2015). Once a PI reaches a chronic stage, treatment options are often limited and expensive (Landi et al., 2007; White-Chu et al., 2011). The management of PIs extracts considerable national healthcare expenditure (Collier, 2008; Dealey et al., 2012; Glover, 2013). In Australia, PIs are one of the "top five" most expensive adverse events (Jackson, Nghiem & Rowell, 2011), costing AU$1.8 billion per annum (Nguyen et al., 2015) and increasing the duration of a hospital stay by up to 20 days (Padula & Delarmente, 2019).

The PI prevalence data are consistently high. These wounds remain a particularly significant problem within acute-care and long-term care hospital settings on a global scale. PIs have been reported to be most prevalent in the frail older adult population, typically occurring in the context of chronic illness and high-care dependency (Khor et al., 2014). It has been estimated that approximately 70% of all PIs occur in persons over 70 years of age (Asimus, Li & Kendall, 2011), with PI prevalence rates expected to rise concomitant with the ageing demographic of many developed countries (WHO, 2019).

Moore et al. (2019) conducted a systematic review to establish the prevalence of PIs based on published studies from Europe. Quantitative study designs, which explored prevalence data and/or the epidemiology of PIs in European countries, were considered. Across the reviewed studies, the median PI prevalence was reported to be 10.8% (*SD*=7%; range: 4.6-27.2%). The highest PI prevalence reported was from the Netherlands (27.2%; n=17,494), while the lowest was reported from Finland (4.6%; n=629). Other systematic reviews have similarly evidenced a wide range of PI prevalence rates among hospitalised patients: 6% in Australia; 3.1-30% in the United States; 1-54% in Europe, and 2.7-16.8% in Asia (Hahnel et al., 2017; Tubaishat et al., 2018).

1.3 Pressure Injury Development

PIs represent a breach of skin integrity and continuum of tissue damage of ischemic aetiology, typically occurring at the skin and soft tissue overlying the weight-bearing bony prominences of the body (Jiang et al., 2011; Kosiak, 1961; Shilo & Gefen, 2012). For ischemic tissue damage to develop, an individual must first be exposed to sustained mechanical loading (Bronnenberg et al., 2006; Källman et al., 2015) and high contact

interface pressures (i.e., the pressure measured between the body surface and an external support surface) (Linder-Ganz & Gefen, 2007). Mechanical loads can originate from bodyweight forces (i.e., the body mass pulled by gravity) or from the environment, for example, delivered by medical devices which apply continuous forces and deformations to skin and deeper tissues near the contact region. Under normal circumstances, the effects of continuous mechanical loading at a given anatomical region signal frequent small body movements to relieve the load. In the absence of pressure relief, the vessels extending to the dermal papillae collapse, leading to inadequate local tissue perfusion. Over time, inadequate tissue perfusion results in localised tissue ischemia which, ultimately, precipitates PI development (Gebhardt, 2002; NPUAP, 2019).

The time required to develop a PI is dependent on many factors, including pressure-loading time and the degree of interface pressure placed on the tissue (Gould et al., 2015). Higher interface pressures have been associated with a raised incline of the head-of-the-bed, for example, as observed in patients positioned semi-recumbent. Using full-body pressure mapping, Barnett and Shelton (1997) investigated interface pressures arising from backrest elevations of 0 to 30° and 45° in healthy human subjects. The findings showed that a higher backrest elevation was associated with increased loading of the pelvic region which, in turn, produced elevated interface pressures at the gluteal, sacral and coccygeal weight-bearing bony prominences. Peterson et al. (2008), also using pressure mapping in 14 healthy volunteers, found the lowest sacral pressures to occur with a 0° backrest elevation, and significantly greater pressures occurring with 30, 45, 60 and 75° backrest elevation. In patients positioned at an incline, internal structures such as bone and muscle are displaced downward due to gravity which, in the absence of pressure relief, can lead to tissue hypoxia as blood vessels become distorted or flattened (Mervis & Phillips, 2019). Thus, in clinical practice, it is recommended to avoid elevating the head-of-the-bed by more than 30° in bedbound patients to minimise the effects of interface pressure generated at the compressed bony prominences (Burk & Grap, 2018; Kim & Shin, 2021).

The mechanical forces of shear and friction, exerted at the skin surface, are two factors that can contribute to PI development through their impact on the mechanical boundary conditions (i.e., the mechanical load that is applied to the skin at the interface with the supporting surface) (NPUAP, 2019). Within the literature, shear and friction are conceptualised as mechanical boundary conditions that can considerably accelerate PI formation (Agrawal & Chauhan, 2012; Gefen, 2017; Hess, 2004; Stechmiller et al., 2008).

The combined effects of shear and friction and particularly deleterious when the skin is wet/moist (Derler et al., 2012; Goosens et al., 1997; Rotaru et al., 2013). As such, skin moisture represents a key factor that may influence the mechanical interactions between the skin and support surface. Within the literature pertaining to biomedical engineering, the parameter of skin moisture is frequently encapsulated by the notion of 'skin microclimate', whose role in the aetiology and evolution of PIs is becoming increasingly recognised (Bader et al., 2018; Clark et al., 2010; Gefen, 2011; Kottner et al., 2018; Yusuf et al., 2015).

1.3.1 Skin Microclimate in Pressure Injury Development

As demonstrated by a long-standing body of evidence, it is widely acknowledged that sustained mechanical loading, in combination with the synergistic effects of shear and friction, are the most important factors implicated in PI development. More recent evidence from key studies (Clark et al., 2010; Falloon et al., 2018; Gefen, 2011; Gefen et al., 2021; Kottner et al., 2018; Reger, Ranganathan & Sahgal, 2007; Schwartz & Gefen, 2020; Yusuf et al., 2015; Zeevi et al., 2018) has identified the skin microclimate as a relevant dimension to understanding PI development. The International Review on *Pressure Ulcer Prevention: Pressure, Shear, Friction and Microclimate in Context Consensus Document* describes the skin microclimate as "the moisture and skin temperature in a specific location" (Wounds International, 2010, p. 19). For the purposes of support surfaces, skin microclimate has traditionally been used to describe the interaction between skin moisture/relative humidity, airflow, and skin temperature in the vicinity of the skin-support surface interface (Clark & Black et al., 2011).

The microclimate parameter of skin moisture can refer to the moisture contiguous to the skin surface, or to the moisture within the deeper layers of the skin (Clark et al., 2011). Skin surface moisture—arising, for example, from perspiration, wound drainage, or urinary and/or faecal incontinence—can become problematic if continually 'trapped' between the patient's skin and the support surface (Dealey et al., 2015; Denzinger et al., 2020; Lachenbruch, 2005). If not managed effectively, the presence of continual moisture at the skin-support surface interface can lead to over-hydration of the stratum corneum, precipitating epidermal softening and maceration (Mayrovitz & Sims, 2001; Schnelle et al., 1997). These events have been documented to considerably impact the mechanical strength of the skin barrier (Brienza et al., 2015; Harrow & Mayrovitz, 2014), increasing its vulnerability to the compounding effects of pressure, shear and friction.

The microclimate parameter of skin temperature refers to the temperature at the surface of the skin (Clark et al., 2011). Empirical evidence suggests a relationship between elevations in interface skin temperature and a concurrent reduction in the tolerance threshold of skin and soft tissue to mechanical loading (Baldwin, 2001; Knox, 1999; Gefen, 2011; Kottner et al., 2018; Lechner et al., 2021). Elevations in interface skin temperatures may occur during direct and sustained contact of the skin with an external support surface, which creates an occlusive environment and precipitates further accumulation of heat and humidity. Observational (Yoshimura et al., 2015), quasi-experimental (Jan et al., 2009), descriptive (Knox, 1999; Sae-Sia et al., 2005) and cohort (Yusuf et al., 2015) studies demonstrate that the prolonged exposure of loaded skin to heat can initiate inflammatory processes and trigger vasodilation. These findings are congruent with the evidence-base that suggests that the signal that induces vasodilation of the underlying vasculature is inflammation, which clinically manifests as erythematous skin surface discolouration (Crawford & Pelle, 2004; Guzman-Sanchez et al., 2007; Yamasaki & Gallo, 2011). Subsequently, oxygen consumption, carbon dioxide production, and metabolic waste product excretion increases at the affected (compressed) area of the body. These processes further weaken the affected skin and soft tissue, increasing the individual's susceptibility to ischemic injury (Agrawal & Chauhan, 2012; Wu, van Osdol & Dauskardt, 2016). The direct contribution of skin temperature to PI development, however, remains less clear.

At present, there is limited in-vivo research evidence of the precise role of the skin microclimate in PI development. However, based on the available literature from animal studies (Boyce et al., 1996; Fluhr et al., 2003; Iazzio, 2004; Patel et al., 1999; Rawlings et al., 1995), in-vitro studies (Bronneberg et al., 2006; Gefen, 2011; Hashimoto-Kumasaka et al., 1993; Zeevi et al., 2018) and human-based studies (Bates-Jensen et al., 2007; Denzinger et al., 2020; Falloon et al., 2018; Kottner et al., 2015; Kottner et al., 2018; Lachenbruch, 2005; Lichterfeld-Kottner et al., 2021; Sae-Sia, Wipke-Tevis & Williams, 2005; Schwartz et al., 2020; Zhong et al., 2008), there are data to indicate that alterations in the local skin microclimate parameters of skin moisture and skin temperature may affect the mechanical boundary conditions and, thus, be indirectly attributed to PI formation in susceptible skin. As such, the clinician's early awareness of disruptions to the local skin microclimate may inform risk-mitigating interventions intended to prevent further undesirable microclimate effects at the skin surface.

1.4 Pressure Injury Assessment and Prevention

In the clinical setting, PIs represent significant adverse events (Gunningberg et al., 2019) with their incidence regarded as a quality of care indicator and a nursing-sensitive outcome (Ayello & Lyder, 2007; Ayello & Sibbald, 2019; Nakrem et al., 2009; Smith et al., 2016). Yet, the majority of PIs are considered to be largely preventable in nature (Bhattacharya, 2012; Dealey et al., 2013; Reddy, Gill & Rochon, 2006). Indeed, preventing PI has been an enduring nursing concern for many years. In 1859, Florence Nightingale wrote, "If he has a bedsore, it is generally not the fault of the disease, but of the nursing" (Nightingale, 1859, p. 8). Currently, the prevention of PIs is a multidisciplinary responsibility, however, it remains primarily a nursing duty (NPUAP, 2019). As such, the prevention of PIs has transcended into a key nursing priority.

The National Safety and Quality Health Service (NSQHS) Standards published by the Australian Commission on Safety and Quality in Health Care (ACSQHC) identify Preventing and Managing Pressure Injuries as part of Actions 5.21-5.23 of the Comprehensive Care Standard (ACSQHC, 2019). In line with this standard, healthcare service organisations are required to implement evidence-based strategies to prevent PI if screening identifies that a patient is 'at risk' (ACSQHC, 2019). Studies indicate that timely preventive care can significantly reduce the incidence of PIs (Jiang et al., 2014; Vanderwee et al., 2011). One of the major methods for PI prevention is the routine off-loading of pressure-prone areas (i.e., areas not well-padded with muscle or subcutaneous tissue) of patients with limited mobility. Furthermore, the *Prevention and Treatment of Pressure Ulcers Clinical Practice Guidelines* (EPUAP-NPUAP-PPPIA, 2019) recommend that bedbound patients be repositioned every two hours (EPUAP-NPUAP-PPPIA, 2019) in order to restore blood volume to the previously-compressed anatomical regions. Furthermore, each routine off-loading interval provides the clinician with an opportunity to inspect the skin for visual and tactile signs of superficial skin damage. To date, visual skin assessment (VSA) comprises a key element of PI prevention initiatives (EPUAP-NPUAP-PPPIA, 2019).

1.4.1 Skin Appraisal at Pressure-Prone Areas

A continual appraisal of an individual's skin overlying the bony prominences is a focal point of PI risk assessment and prevention (NPUAP, 2019). Specifically, assessors are required to make subjective interpretations of an array of skin parameters with the aim of identifying signs of pressure-induced tissue distress (NPUAP, 2019). Skin erythema—denoted by the descriptor of 'erythematous'—has been well-established as one of the most

important early indicators of soft tissue responses associated with early ischemic injury (Coleman et al., 2013; Hagisawa, 1994; Keller et al., 2002; McElhinny & Hooper, 2008; NPUAP, 2019). In lightly-pigmentated skin, an erythematous area will typically present as reddened discolouration, and as blue/purple discolouration in deeply-pigmented skin (Vanderwee, Grypdonck & Defloor, 2007).

In clinical practice, the detection of erythema at a given bony prominence is routinely evaluated with a blanch test (NPUAP, 2019). An erythematous area that does not blanch in response to brief application of pressure is used to signal ischemic damage associated with Stage I PI (Fletcher, 2010; Romanelli et al., 2006; Sussman & Bates-Jensen, 2007; Vanderwee et al., 2007). However, provided that the erythematous area is promptly off-loaded, non-blanchable erythema is generally reversible (Edsberg et al., 2014; Romanelli et al., 2006; Shi et al., 2020). Thus, early identification of erythema is of high clinical importance as it allows instigation of interventions to prevent possible progression of further damage, followed by a review of the patient's pressure off-loadng regime (Gillespie et al., 2020; NPAUP, 2019). The detection of non-blanchable erythema may be difficult to discern in darker skin tones due to the masking effect of melanin pigment (Sprigle et al., 2003).

In conjunction with an appraisal of erythema, a fundamental component of PI risk screening (NPUAP, 2019) is the visual appraisal of the microclimate parameter of skin moisture. As previously outlined in this chapter, the presence of continual moisture contiguous to the skin surface acts as a critical factor for diminished epidermal barrier function, particularly in patients who are immobile and bedbound (Agrawal & Chauhan, 2012; Harrow & Mayrovitz, 2014). In clinical practice, the detection of pronounced skin moisture at the skin support surface interface is typically followed by interventions aimed at 'microclimate management' to ensure that the epidermal barrier does not become over-hydrated. Such interventions typically include keeping the skin dry and allowing recovery periods between phases of occlusion (NPUAP, 2019).

Finally, the NPUAP (2019) recommends that clinicians use touch and palpation when inspecting the skin over pressure-prone areas to detect for the presence of elevated skin warmth. There is evidence to suggest that localised areas of pronounced skin warmth at the skin surface result from local sub-epidermal inflammatory responses (Armstrong & Lavery, 1996; Bach et al., 2015; Gefen, 2021; Lachenbruch, 2005; Oerlemans et al., 1999). Inflammation has been reported as the earliest sign of impending ulceration (Bates-Jensen,

2007; Schubert & Fagrell, 1989; Sprigle, Linden & Riodan, 2003; Wong et al., 2011), with erythema manifested at the skin surface regarded as a cardinal sign of inflammation (Bates-Jensen et al., 2007). Additionally, studies indicate that an elevation in skin temperature can contribute to impaired tissue tolerance (Baldwin, 2001; Jan et al., 2009; Gefen et al., 2021; Knox, 1999; Posada-Moreno et al., 2011; Sae-Sia et al., 2005; Soares et al., 2019; Yoshimura et al., 2015; Yusuf et al., 2015). However, whether elevated skin temperature acts as a direct contributor to PI development still remains unclear.

1.4.2 Limitations of Visual Skin Appraisal

Contemporary approaches to PI prevention rely on the clinician's appraisal of erythema, moisture and skin temperature of the skin overlying the bony prominences of the body. These parameters have emerged as important factors in the theories and evidence pertaining to PI development. There may, however, be limitations with regard to the current methods used to appraise these skin parameters in clinical practice. Such limitations can be observed in the case of skin colour appraisal, specifically in finer hues of erythema. As outlined earlier in this chapter, the clinician's visual recognition of erythematous hues at the skin surface overlying a bony prominence can provide a measure of soft tissue responses associated with early ischemic injury. However, these observations neither allow quantitative description of tissue responses nor are they effective for patients with deeply-pigmented skin (Bennett, 1995; Bethall, 2003; Sprigle et al., 2003). Thus, the subjective nature of current PI risk appraisal methods may preclude an accurate classification of important cutaneous parameters attributed to PI development.

Further challenges of visual skin appraisal pertain to the classification of the microclimate parameter of skin moisture. Currently, an assessment of skin moisture is featured as a sub-scale component of three well-recognised standardised PI risk assessment scales: the Braden Scale (Bergstrom & Braden, 1987), Waterlow Scale (Waterlow, 1985) and Norton Scale (Norton, 1987). Yet, there may be a theoretical limitation in regard to the manner in which skin moisture is represented by these scales. The scales tend to rely on a broader representation of skin moisture; that is, moisture is measured by the presence of 'dampness' or 'wetness' at the skin surface. However, the scales do not consider a 'direct' measure of moisture, such as the moisture within the deeper layers of the stratum corneum. Studies report that stratum corneum moisture may be associated with maintaining the mechanical properties of the skin barrier (Berardesca, 1997; Clarys et al., 2021; Fluhr et al., 1999; Warner et al., 1999) and, therefore, may be a critical factor implicated in PI

development. However, given that stratum corneum moisture is a biophysical skin property (Clarys et al., 2012), it can only be evaluated by capacitance or conductance-measuring devices (Clarys & Barel, 1995). As such, it can be argued that current methods of skin moisture appraisal, as guided by PI risk assessment scales, may often fail to recognise what is happening 'below' the skin surface.

Limitations may also exist in reference to the measurement of the microclimate parameter of skin temperature in a PI risk assessment context. It is challenging to quantify and interpret variations in relation to skin temperature merely by the touch of the hand; this technique is highly interpretive and only as sensitive as the hand of the assessor; a cold hand will indicate a warm skin surface that, at the same time, a warm hand will detect as a cooler skin surface (Pompei, 1996). Ultimately, skin temperature should be considered as a continuum measured in small units. However, from the perspective of a subjective assessment, skin temperature assessment, at best, serves as a gross measurement.

Overall, it can be argued that an appraisal of the parameters of erythema, skin moisture and skin temperature is critical to guide informed decision-making regarding appropriate PI prevention actions. Underpinning these actions, however, is a reliance on the assessor's diagnostic accuracy of skin appraisal itself, which is deeply subjective and may be limited. As such, the initiation of PI prevention measures may be hindered by the fact that skin appraisal may not always yield reliable and accurate results. In view of these limitations, there is an opportunity to augment appraisal accuracy with objective techniques. There are biophysical skin-analysis instruments with the capacity to generate objective measures of erythema, stratum corneum hydration and skin temperature. Theoretically, these instruments could have the potential to overcome the challenges associated with subjective appraisal methods, and may offer a more 'precise' indication of important skin changes preceding early onset PI development.

1.5 Biophysical Skin-Analysis Instruments

There is a range of commercially available non-invasive biophysical skin-analysis instruments that enable the instant quantification of erythema (irrespective of skin colour), stratum corneum hydration, and skin temperature. At present, the application of these instruments is largely limited to dermatological and cosmetic practice. However, the reliability and validity of some of these instruments—in the context of PI risk assessment—have been previously investigated in three independent studies (Borzdynski, McGuiness &

Miller, 2016; Huimin et al., 2017; Rowledge et al., 2016). The reliability and validity of the investigated instruments was found to be exceptionally high with respect to the assessment of erythema and stratum corneum hydration at the peri-wound skin of venous leg ulcers (Huimin et al., 2017) and diabetic foot ulcers (Rowledge, et al., 2016), as well as intact skin at pressure-prone areas of the body (Borzdynski, McGuiness & Miller, 2016). Furthermore, one of these collective studies (Borzdynski, McGuiness & Miller, 2016) indicated that the finer measures afforded by biophysical skin-analysis instruments in the assessment of the subtle red hues displayed in erythematous skin may provide an additional advantage over traditional clinician assessment.

A recent study by Fletcher, Milne, and Moore (2018) investigated the reliability of a biophysical skin-analysis instrument, the SEM scanner, in its measurement of sub-epidermal moisture beneath the skin surface. The findings demonstrated that the SEM scanner is an effective instrument in detecting early changes in sub-epidermal moisture levels, and can pre-empt the development of PIs earlier than the standard practice of visual skin appraisal alone. Studies report that sub-epidermal moisture represents an inflammatory biophysical marker that has been found to precede the appearance of visible or palpable skin changes by approximately 3 to 10 days (Bates-Jensen et al., 2009; Gefen & Gershon, 2018; Moore et al., 2017). Budri et al. (2020) state that sub-epidermal moisture is the product of plasma that leaks as a response to local inflammation mediated by pressure-induced damage. Thus, based on the results of Fletcher, Milne, and Moore (2018), it can be argued that the SEM scanner may serve as a useful diagnostic tool capable of capturing physiological changes in tissue damage in ‘real time’, that is, when pressure-associated damage is occurring.

Within dermatological and cosmetic practice, there are biophysical skin-analysis devices that can also enable quantitative measures of melanin; a naturally-occurring skin pigment (Mackintosh, 2001). At present, there is some evidence to suggest an association between the amount of melanin pigment and skin barrier function (Baumgarten et al., 2004; Maranduca et al., 2019; Reed, Ghadially & Elias, 1995); however, the clinical significance of melanin as a prognostic factor for PI development is yet to be elucidated.

Overall, the clinician’s early recognition of clinically concerning cutaneous changes manifested at pressure-prone areas of the body is critical for enabling timely interventions to protect tissue viability. At present, visual skin appraisal is routinely used to identify signs of cutaneous factors proposed to be implicated in PI development (EPUAP, 2019). A cutaneous

factor routinely appraised by the clinician includes erythema. Furthermore, the literature postulates that skin moisture and skin temperature may serve as subjective indicators of skin status, with elevations in any of these parameters signaling the potential skin and soft tissue compromise. As such, an appraisal of skin moisture and skin temperature is routinely performed by the clinician in individuals with a predisposition to PI development. However, there are limitations in regard to the clinician's accuracy in capturing PI-associated skin parameters when relying merely on visual skin appraisal alone. Opportunities exist to quantify PI-associated skin parameters using objective modalities which, in turn, may help to strengthen skin appraisal accuracy. This has provided the research ground for this thesis. The next sections detail the rationale, purpose and significance of this thesis.

1.6 Thesis Rationale

To date, there continues to be an inadequate understanding of the construct of the skin microclimate as a direct contributor to PI development. This understanding is essential given that evidence suggests that alterations in the skin microclimate are implicated in the processes which give rise to PI. Studies have theorised that the inextricable interaction between the skin microclimate parameters of skin moisture and skin temperature indirectly contribute to PI formation. Yet, there continues to be an evident lack of foundational knowledge of an 'optimal range' for skin microclimate in human skin. The possible upper and lower thresholds for skin moisture and skin temperature at pressure-prone predilection areas, based on in-vivo studies, are currently unknown (Kottner et al., 2018). Additionally, there is an evident need to better understand the concept of skin colour, given that changes in skin colour have been associated with early onset PI (Bates-Jensen et al., 2007). In order to facilitate a more comprehensive understanding of these concepts, a clear interpretation and range for 'normal' and 'clinically significant' parameters of skin colour (erythema and melanin), skin moisture and skin temperature at pressure-prone areas needs to be established.

There are commercially available skin-analysis instruments that have the capacity to yield quantitative measurements of skin parameters (erythema, melanin, stratum corneum hydration and skin temperature). However, there is currently a lack of sufficient evidence to confirm the clinical value of quantitative appraisal of skin manifestations when applied to PI risk assessment. To the researchers' best knowledge, there are currently no standardised thresholds for biophysical measures of erythema, melanin, stratum corneum hydration and skin temperature proceeding skin and soft tissue damage at PI predilection areas. Although there are published research studies reporting biophysical skin colour and microclimate data,

this research is largely limited to animal-based studies (Boyce et al., 1996; Fluhr et al., 2003; Iazzio, 2004; Patel et al., 1999; Rawlings et al., 1995), in-vitro studies (Bronneberg et al., 2006; Gefen, 2009; Hashimoto-Kumasaka et al., 1993) and cosmetic studies (Conti et al., 1995; Edwards et al., 2005; Heinrich et al., 2003; Horii et al., 1989). Thus, the results of these studies may not be directly transferrable to a PI risk assessment context. Some human-based studies exist that report biophysical measures of skin colour (Bates-Jensen et al., 2007; Bates-Jensen et al., 2012; Borzdynski, McGuiness & Miller, 2016; Denzinger et al., 2020; Huimin et al., 2017; Lindgren et al., 2004) and microclimate data (Bates-Jensen et al., 2012; Denzinger et al., 2020; Falloon et al., 2018; Kottner et al., 2015; Kottner et al., 2018; Lachenbruch, 2005; Lichterfeld-Kottner et al., 2021; Sae-Sia, Wipke-Tevis & Williams, 2005; Schwartz et al., 2020; Zhong et al., 2008) at pressure-prone areas using immobilisation protocols in healthy adults. However, at present, no studies that have collectively measured these parameters—erythema, melanin, stratum corneum hydration and skin temperature—nor considered how these parameters change in response to disruptions to the skin microclimate. This paucity in the literature provides the impetus for this research.

In essence, this research is justified by the evident need to generate new knowledge regarding changes in skin colour and skin microclimate variables gathered at PI predilection areas of healthy semi-recumbent healthy adults. Translating biophysical skin measures of erythema, stratum corneum hydration and skin temperature into clinically meaningful indicators of impending PI is certainly promising, and could potentially enhance the identification of PI risk status in various patient cohorts. Further investigation is required to determine how biophysical skin measures of erythema, stratum corneum hydration and skin temperature are influenced by the compounding effects of interface pressure and loading time, as well as extrinsic factors of heat and moisture. It is within this context that the current study was developed.

1.7 Purpose of this Research

The purpose of this research is to explore possible skin manifestations attributed to local pressure and skin microclimate in individuals positioned in a supine and semi-recumbent position over one hour. This research seeks to measure biophysical skin properties of erythema, melanin, stratum corneum hydration and skin temperature in response to local pressure-loading and controlled manipulation of the skin microclimate. As such, this research builds on the literature related to skin microclimate changes in healthy adults, and contributes to the emerging evidence-base relating to quantitative skin appraisal.

1.8 Significance of this Research

PIs represent significant adverse events, with these wounds being progressive in nature and having the potential to develop into life-threatening complications. The management and treatment of PIs can impose substantial financial burdens for both the patient and the healthcare system. Prevention is the best strategy for managing PIs. Regular inspection of patients' skin condition is a key step in PI prevention, and is dependent on the clinician's diagnostic appraisal of cutaneous changes manifested over pressure-prone areas of the body. However, current methods of PI skin appraisal tend to be largely subjective, heavily reliant on assessor's clinical judgment and interpretation. To date, the accurate detection of important cutaneous manifestations preceding PI development represents a pernicious clinical challenge. This challenge provides an opportunity to objectify current methods of skin appraisal employed in PI risk assessment.

This research is anticipated to make a significant contribution to the literature in four ways. First, this research will enable the establishment of a descriptive library of objective (quantitative) measures of erythema, melanin, stratum corneum hydration and skin temperature, gathered at the skin surface of pressure-prone areas of healthy adults lying in the supine (0° incline) and semi-recumbent (45° incline) position over one hour.

Second, the gathered data will not only provide a baseline for biophysical skin parameters measured under normal conditions, over time (one hour), during brief periods of cyclic loading, but will also consider how these parameters change individually in controlled conditions simulating alterations in the skin microclimate at pressure-prone areas. As such, in view of the paucity of detailed in-vivo studies considering skin colour and skin microclimate changes over time, this thesis contributes longitudinal inferences; albeit limited to one hour as an initial excursion to ensure participant safety.

Third, this research will enable regional anatomical comparisons of erythema, melanin, stratum corneum hydration and skin temperature data. These findings, in turn, will strengthen reference values for erythema, melanin, stratum corneum hydration and skin temperature at common PI predilection areas of the body. This information is of particular clinical relevance in view of the lack of benchmark data for the aforementioned skin parameters at anatomical regions prone to PI development.

Finally, this research is distinctive in that it aims to contribute to an improved understanding of the utility of biophysical skin-analysis instruments in the examination of cutaneous manifestations often attributed to PI. In this way, this thesis makes a unique contribution to the literature; it is anticipated that the findings will assist in the establishment of the potential value of quantitative technologies as innovative methods to capture important changes proposed to be implicated in PI development.

1.9 Thesis Outline (Organisation of Chapters)

This thesis is presented in the form a traditional thesis and is organised into ten chapters, and includes one published work. Chapter 1 provides an overview of the thesis, and introduces the key background concepts necessary to understand this research in context. The chapter additionally states the rationale, purpose and significance of this research. It concludes by presenting an overview of the thesis layout and structure.

Chapter 2 of this thesis provides a comprehensive overview of PIs and their significance within the healthcare system. A discussion pertaining to PI aetiology is provided, and factors contributing to PI development are identified.

Chapter 3 explores the literature relevant to the construct of skin microclimate as a theoretical contributor to PI formation. In doing so, the parameters of skin moisture and skin temperature are considered as contextual microclimate elements that may influence PI development.

Chapter 4 presents a review of the current clinical methods employed in the appraisal of the skin in a PI risk assessment context, and considers technology that could enhance current appraisal methods. The chapter concludes with an articulation of the research gaps and reinforces the aims, questions and objectives specific to Studies I-III.

In Chapter 5, the first phase of this research (Studies I and II) is presented as a publication, as published by the *Journal of Tissue Viability* and titled "Brief intermittent pressure off-loading on skin microclimate in healthy adults – A descriptive-correlational pilot study" (Borzdynski, Miller, Vicendese & McGuiness, 2021). This publication represents an important milestone in the progression of the understanding of skin changes, over time, in response to local pressure-loading at the bony prominences of the body. The results and

conclusions emerging from this preliminary research phase were used to guide the experimental research design of the final phase of this research (Study III).

Chapter 6 details the methodological basis and the methods used to undertake data collection and analysis in Study III. In doing so, the chapter details the research aims, questions, objectives, and hypotheses specific to Study III. The research design, participant eligibility criteria, recruitment processes and the measured variables are provided. The data collection instruments and their measurement principles are also detailed. The chapter then outlines the relevant data collection procedures, and provides a description of the statistical analyses that were used to address the Study III research aim and objectives.

In Chapter 7, descriptive data for the Study III sample are presented. The chapter provides an overview of the screening and preparation of data, followed by a presentation of the participant sample with respect to their demographic and health data profile. Descriptive statistics are provided to profile the investigated skin parameters, as gathered over time and across the study conditions.

Chapter 8 of this thesis reports the linear mixed model (LMM) analyses results for the Study III sample. The chapter begins with an overview of the screening and treatment of the data related to LMM analyses for repeated measures, including the extent to which the data met assumptions of linearity as well as bivariate distribution. The results of LMM analyses are reported for each skin parameter variable by anatomical location for the participant sample.

In Chapter 9, the results of this research are closely evaluated and interpreted and the significance to published literature is discussed. The clinical implications of the study results are considered as they relate to the study hypotheses and, more broadly, to the research aims and objectives. The strengths and limitations are identified, and recommendations are proposed in relation to clinical practice and further research.

Chapter 10 summarises the research as a whole and its contribution to practice. The chapter includes a final discussion and concluding remarks, which summarise the research findings and potential clinical implications. The chapter concludes with a summary of the implications for both clinical practice and future research based on the evidence generated.

1.10 Chapter Summary

This chapter has provided an overview of the research studies conducted as part of this thesis, and has introduced the reader to the concept of PIs and their significance. A brief overview of the key factors implicated in PI development has been provided, and the theoretical contribution of the skin microclimate to PI development has been highlighted as a focal point of forthcoming chapters. The purpose of skin appraisal as part of PI risk assessment processes has been explained, and the notion of biophysical instruments as a potential method of objectifying current, subjective methods of skin appraisal has been considered. The rationale, purpose and significance of this research have been presented. Finally, forthcoming thesis chapters have been summarised. In the next chapter, a comprehensive discussion is provided pertaining to the significance of PIs and factors contributing to PI development in order to build upon the background provided in Chapter 1.

CHAPTER 2

Background of Pressure Injuries and Factors Contributing to their Development

2.1 Chapter Overview

Chapter Two provides an expanded review of the current literature exploring pressure injuries (PIs) and factors contributing to their development. The chapter begins with a brief historical perspective of PI. An overview of the significance of PIs and their prevalence rates is subsequently detailed. The current classification system for PI is provided, and the anatomical locations most susceptible to PI occurrence are identified. The chapter concludes with a detailed overview of PI aetiology, and identifies key factors that contribute to PI formation.

2.2 Historical Perspective of Pressure Injuries

PIs appeared in the medical literature more than 5000 years ago; detailed reports date back to Ancient Egypt with evidence of pressure ulceration found in Egyptian mummies (Agrawal & Chauhan, 2012). Hippocrates (460-370 B.C), the Greek physician of the Age of Pericles, described PIs in association with paraplegia with bladder and bowel dysfunction (Adams, 1946). PIs continued to be recognised as a disease entity throughout the centuries. During the Renaissance, Ambrose Paré, 16th century French army surgeon and founding father of medical surgical practice, wrote in his autobiography about a wounded French aristocrat developing a PI. Paré described a PI as an incurable disease which could be treated with rest and adequate nutrition (Bansal et al., 2005). In the same century, Hieronymus Fabricius, an Italian pioneering anatomist and surgeon, had associated PIs with the disease of the nervous system. Similarly, in the 19th century, Jean-Martin Charcot, a French neurologist and professor of anatomical pathology, associated the occurrence of a PI with damage to the central nervous system, with its presence signalling imminent patient death (Bansal et al., 2005). Charcot recognised that rapid atrophy of the tissues contributed to PI, as did immobility. He also recognised the role of pressure but considered it secondary. Charcot tried to alleviate the PI by turning the patient regularly. He stressed that PIs could appear in two days and carried an ominous prognosis, referring to them as ‘ominous sores’. He described PIs as infected and dangerous, commonly giving rise to fever in the acute stage, and relapsing fever in the chronic stage (Bader, 1990).

As such, it is clear that PIs are not a new phenomenon. From the beginning of recorded history, PIs have been reported as one of the most constant problems plaguing the disabled, debilitated and chronically ill. To date, the impact of PIs is significant, extending beyond hospital walls. While the majority of PIs are argued to be largely 'preventable', they represent an ongoing challenge on a global scale (Asimus et al., 2011; Dealey et al., 2015; Ebi et al., 2019; Lyder et al., 2012; Moore, Johansen & van Etten, 2013; Nguyen et al., 2015; NPUAP, 2019).

2.3 Significance of Pressure Injuries

The impact of PIs on patients has been well-documented. Hay et al. (2014) described PIs as one of the most "severe dermatological diseases" contributing to a substantial loss of health. PIs have a detrimental impact on all domains of the quality of life of those affected (Benbow, 2009; Chaboyer & Gillespie, 2014; Coleman et al., 2014; Essex et al., 2009; Fox, 2002; Gorecki et al., 2009; Langemo et al., 2000) including physical and psychosocial wellbeing (Coleman et al., 2013; Latimer, Chaboyer & Gillespie, 2014). The physical consequences associated with a PI can range from mild tissue damage or a chronic wound requiring major surgical intervention and the possibility of permanent disfigurement. Hopkins (2006) identified the disfigurement caused by chronic non-healing PIs to be associated with diminished self-image and social isolation due to a restriction to one's normal lifestyle and undesired dependence. Hopkins (2006) further reported that alongside the personal impact of acquiring a PI, those affected had expressed their concerns for the burden placed on their family members having to become caregivers.

Numerous quantitative and qualitative studies have identified pain as the dominating physical factor in the lives of patients with an existing PI (Beitz & Goldberg, 2005; Galhardo et al., 2010; Gorecki et al., 2014; Rastinehead, 2006). PI pain has been described as both constant and transient (Girouard et al., 2008). Beitz and Goldberg (2005) reported that the intensity of pain associated with PIs significantly interrupts daily activities including ambulation and normal sleep patterns. Additionally, Ahn, Stechmiller, and Horgas (2013), in their longitudinal study of 56,577 nursing home residents, found that residents with a PI reported to have 24% to 68% more severe pain than residents without a PI.

Many early stage PIs, if left untreated, can rapidly progress to more severe and complex lesions of the skin and underlying tissue. Chronic PIs are particularly morbid; once a PI reaches a chronic advanced stage, it usually becomes resistant to even the best medical

and nursing therapies available today (Dealey, Posneet & Walker, 2012; Jaul et al., 2018). Chronic PIs have been reported as difficult and, in numerous cases, impossible to heal, with estimates of complete healing as low as 10% in some aged-care facilities (Palese et al., 2015). Furthermore, it has been estimated that as many as 54% of chronic PIs require multiple hospital admissions and approximately 95% do not heal within eight weeks from admission (Schryvers et al., 2000).

The mortality rates associated with PIs are also significant. Brown (2003) reported the six-month mortality rate of patients with advanced PIs to be as high as 68.9%. Kuwahara et al. (2005) reported 68.8% mortality amongst elderly patients with advanced PIs due to secondary systemic complications. A report using data collected from 187 countries indicates that deaths associated with PI have increased by 32.7% (from 321,000 patients to 426,000 patients in 1990 and 2010, respectively) (Lozano et al., 2012). In a systematic review of health conditions in hospital patients and nursing home residents (Thomas, Cooney & Fried, 2013), PIs were cited as one of seven conditions associated with short-term mortality. Furthermore, PIs are listed as the direct cause of death in 7-8% of all patients with paraplegia (Kruger et al., 2013).

The length of hospital stay for a patient who develops a PI, on average, has reported to be prolonged by four to six days than for a patient without a PI. Furthermore, upon discharge, 76.9% of patients with chronic PI will require transfer to sub-acute facilities or the need for home-care services, further contributing to the financial burden associated with the management of PIs (Girard et al., 2014). It has also been estimated that approximately 30% of advanced non-healing PIs result in bacterial infection (Khor, Tan & Saedon, 2014). If left untreated, the bacterial infection can rapidly permeate to underlying tissues and bone, resulting in life-threatening complications such as osteomyelitis (Bodavula et al., 2015; Rennert, Golinko & Yang, 2009), necrotizing fasciitis (Braga et al., 2017) and septicaemia (Abutaleb et al., 2021; Al Aboud & Manna, 2021). Redelings, Lee, and Sorvillo (2015) reported septicaemia as an underlying or contributing cause in 39.7% of all PI-associated deaths. Recent data indicate that PI-associated infection results in an estimated death of 60,000 patients globally (Padula & Delarmente, 2019).

Due to the substantial impact of PIs on patient quality of life, the prevention of PIs is prioritised by healthcare providers and policy makers (Gorecki et al., 2009; Spilsbury et al., 2007; Whitty et al., 2017). To date, the prevention of PI is a major healthcare priority area

both in Australia and internationally (Australian Commission on Safety and Quality in Health Care (ACSQHC); World Health Organization (WHO), 2019). Within clinical practice, PI prevalence is considered to be a marker of quality of healthcare, particularly nursing care (NPUAP, 2019). Currently, the National Safety and Quality Health Service (NSQHS) Standards (ACSQHC, 2019) identify *Preventing and Managing Pressure Injuries* as part of the Comprehensive Care Standard. Based upon this standard, evidence-based strategies to prevent PIs are mandatory if screening identifies that a patient is at risk of PI (ACSQHC, 2019).

It becomes evident from the published literature that PIs constitute a significant problem within society, imposing substantial physical, psychological, social and economic burdens. They are debilitating, painful and costly encumbrances to those affected, with the propensity to become life-threatening injuries. Despite considerable efforts to curb their occurrence, PI prevalence rates on a global scale continue to be considerably high.

2.4 Prevalence of Pressure Injuries

There is a general consensus that PIs are largely preventable (EPUAP-NPUAP, 2019). Yet, they remain a frequently-occurring problem across various healthcare settings, including acute care (Graves, Birrell & Whitby, 2005; Gunningberg et al., 2012; Lyder et al., 2012) and the community (Asimus, Li & Kendall, 2011; Baath et al., 2014). The global estimated average PI prevalence in acute care settings ranges from 7.8% (Mehta & Wangmo, 2015) to 54% (Moore et al., 2015), and in the community from 2.5% (Halfens, 2013) to 29.2% (Hahnel et al., 2017). Santamaria et al. (2005) reported PI prevalence rates to be as high as 43% in some Australian nursing homes, while the Australian Government Department of Health and Ageing (2012) reported PI prevalence among Australian nursing homes to oscillate between 13% to 37% in any one year.

The prevalence rates of hospital-acquired pressure injury (HAPI) have ranged from 1.1% to 26.7% (Bredesen et al., 2015; Graves & Zheng, 2014; Moore et al., 2013). Australian data suggest a 4.0% to 9.0% HAPI prevalence rate (Miles, Fulbrook & Nowicki, 2013) of which it is estimated that up to 22.6% of these HAPIs are avoidable (Mulligan et al., 2011). A recent meta-analysis (Li, Lin & Chaboyer, 2020) of almost two million hospitalised patients reported a pooled estimate PI prevalence of 12.8%, and a pooled HAPI rate of 8.4%. PI prevalence is also reported to be considerably higher in the intensive care setting. PI incidence in the adult intensive care population has been reported to be between

10.0% to 25.9% (Coyer et al., 2017), which is higher than the general hospital population (5.4%) (Li, Lin & Chaboyer, 2020).

The accumulated data clearly indicate that PIs and their sequelae are a significant problem worldwide. PIs have been associated with morbidity and mortality and, at present, represent a quality of care indicator and a nursing-sensitive outcome. Despite extensive and continued research regarding PI prevention and management, sustained reductions in PI prevalence rates are challenging.

2.5 Classification of Pressure Injuries

PI classification systems have evolved with greater clinical knowledge. The initial PI classification system was first developed in 1989 by the NPUAP. The panel has continued to update the classification system throughout the years by adding terminology to better identify PIs in individuals with deeply-pigmented skin, and to expand upon the role of deep tissue injury (DTI) as a precursor to PI formation (NPUAP, 2019). Two new definitions have recently been included in the PI staging system: "medical device-related pressure injury" (MDRPI) and "mucous membrane/mucosal pressure injury" (MMPI).

Currently, PIs are classified according to a standardised international staging system on a scale ranging from I to IV. This staging system classifies PIs based on the extent of anatomical tissue damage detected; the higher the stage, the more severe the tissue damage (NPUAP, 2019). These PI stages range on a continuum of 'suspected injury and initial redness' to 'full-thickness tissue loss and necrosis' (Edsberg et al., 2016).

Stage I PIs are the most superficial type of PI marked by the presence of non-blanchable erythema of intact skin (NPUAP, 2019). Clinically, non-blanching erythema localised over a bony prominence is considered to indicate localised acute ischemic damage (Fletcher, 2010; Romanelli et al., 2006; Sussman & Bates-Jensen, 2007; Vanderwee, Grypdonck & Defloor, 2007). The detection of non-blanchable erythema may be difficult to discern in deeply-pigmented skin (Bethall, 2005) due to the masking effect of melanin pigment (Sprigle et al., 2003).

Stage II PIs are characterised by partial-thickness skin loss involving the epidermis extending to, but no deeper than, the dermis; in this stage, the PI may be clinically visible as a blister or an abrasion. Stage III PIs involve full-thickness skin loss and damage of

subcutaneous tissue that may extend down to, but not through, underlying fascia. At this stage, there may also be undermining; significant erosion underneath the outwardly visible wound margins results in more extensive damage beneath the skin surface. Consequently, while the external wound might appear small, large areas of tissue loss will be detected beneath the surface upon measurement of the wound depth with a cotton-top applicator (Grey, Enoch & Harding, 20016). Finally, Stage IV PIs represent extensive destruction and tissue necrosis, extending into the muscle, tendon or bone. Wound undermining (erosion of the tissue underneath the wound edges, resulting in a 'pocket' beneath the skin at the wound edge) may also be associated with this stage of PI progression (NPUAP, 2019).

The current PI staging system also includes two additional sub-categories: "unstageable pressure injury" and "deep-tissue injury". The term unstageable pressure injury is used to classify lesions in which the depth of tissue damage cannot be confirmed as the wound base is obscured by slough or eschar; a type of necrotic devitalised tissue. If slough or eschar is removed, a Stage III or Stage IV PI may be revealed (NPUAP, 2019). Deep tissue injury (DTI) refers to a deep lesion that presents as persistent non-blanchable deep red, purple or maroon localised area of discoloured intact skin or blood-filled blister. DTIs are predominantly caused by damage to the underlying subcutaneous tissue from pressure and/or shearing injury or torsion of the perforating vessels (Black, 2003). DTIs represent a unique form of PI in that they start to form in the underlying tissue and are often not visible at the skin surface until they have significantly advanced (Stekelenburg et al., 2008). DTIs tend to occur in tissue that has been subjected to pressures that exceed the tolerance level of muscle tissue; the muscle cells gradually deform and may become irreversibly injured from the pressure (Aronovitch et al., 2007).

Two new definitions have recently been included in the PI staging system: "medical device-related pressure injury" (MDRPI) and "mucous membrane/mucosal pressure injury" (MMPI) (NPUAP, 2019). MDRPIs result from the use of devices designed and applied for diagnostic or therapeutic purposes, such as cervical collars, pulse oximeters, nasal cannula and oxygen masks. The resultant PI generally conforms to the pattern or shape of the device (Schank, 2016). Often, these devices must be secured tightly to ensure a proper seal which, in turn, creates pressure at the underlying soft tissue (Reger et al., 2007). Additionally, humidity and heat develop between the device and the skin, changing the local microclimate (Black et al., 2010). MDRPIs are iatrogenic and account for more than 30% of all HAPIs (Martel & Orgill, 2020).

The COVID-19 pandemic introduced a large population of patients at risk for MDRPIs due to prolonged intubation and prone positioning. Prone positioning exerts considerable compressive and shear forces from medical devices (such as endotracheal tubes exiting the mouth or nasal cannula exiting the nose) on adjacent facial structures, increasing the risk for ulceration (Gefen & Ousey, 2020). Furthermore, standard PI risk mitigating interventions, such as repositioning and off-loading, are limited and challenging in prone patients given the context of care for these critically unstable patients.

MMPIs arise on the mucous membranes with a history of a medical device in use at the location of the PI (Schank, 2016). Mucosal membranes are defined as the moist lining of body cavities/organs (Guyton & Hall, 2005). The mucous membranes most susceptible to PI are those of the oral mucosa (particularly the anterior surface of the lips and tongue), nasal mucosa, and the inner lining of the urinary tract. These mucous membranes are highly vulnerable to pressure from the application of medical devices such as endotracheal tubes, nasal cannula, nasogastric tubes, and urinary catheters. The NPUAP (2019) does not recommend staging MMPIs because anatomically analogous tissue comparisons or distinctions cannot be made.

2.6 Anatomical Locations of Pressure Injury Occurrence

The most frequently-reported anatomical locations for PI occurrence are those overlying a prominence of the body (Edsberg et al., 2016; Gefen et al., 2009). A bony prominence is described as the largest most prominent segment of bone, where the bone is in close proximity to the overlying sub-dermal tissue, and where less protection is afforded by the tissue layers (Edsberg et al., 2016). As such, bony prominences are particularly vulnerable to ischemic damage from sustained mechanical loading (Mak et al., 2010; Scheel-Sailer et al., 2013) when the patient is in prolonged contact with hard surfaces. The vulnerability of the tissue overlying a bony prominence to PI formation is dependent on the individual's position, as well as the length of time that they remain in that position (Larson et al., 2012). In bedbound patients immobilised for prolonged periods of time, the supine, semi-recumbent and lateral positions are the three standard patient positions (Bhattacharya & Mishra, 2015). In the supine and semi-recumbent positions, examples of regions where pressure tends to be magnified include the sacrum, coccyx, elbows and calcanei. These anatomical regions also tend to be the most weight-bearing regions in patients who are chair-bound. In the lateral position, the most weight-bearing regions include the ischial

tuberosities, trochanters, lateral and medial malleoli, and lateral aspect of the ear (Bhattacharya & Mishra, 2015).

Mulligan et al. (2011) conducted a study of 517 hospitalised patients who developed PIs and found that 83% of these PIs had occurred at the sacral region. A more recent study (Al Aboud & Manna, 2021) similarly reported that an estimated 75% of PIs occur at the sacral and ischial tuberosities. It should be noted that although the sacral and ischial tuberosities have a relatively thick covering of soft tissue and a wide supporting surface, the underlying cutaneous blood vessels at these regions are not well adapted for weight-bearing. As such, even with limited compression, tissue ischemia can develop rapidly during prolonged weight-bearing at the sacral and ischial bony prominences (Bhattacharya & Mishra, 2015; Therattil et al., 2013).

The heel is another common anatomical site for PI development (Wong & Stotts, 2003) due to its calcaneus pointed shape. The muscle overlying the heel is also poorly vascularised and equipped to absorb the compressive forces of pressure or shear that are generated during limb movement or transfers (Wong & Stotts, 2003). Sopher et al. (2011) added that the loading intensity is more concentrated at the heel because the soft tissues between the calcaneus and the support surface are highly compressed. It has been reported that approximately 26% of PIs develop at the lower extremities such as the heel and malleolar regions (Amlung et al., 2001; Pickenbrock et al., 2017).

2.7 Pressure Injury Aetiology and Pathogenesis

There is a long-standing history of the literature that has examined PI aetiology. Several aetiological pathways for PI development have been proposed over the decades and supported by laboratory evidence, including direct tissue deformation (Bouten et al., 2003; Gefen et al., 2021; Oomens et al., 2015), impaired interstitial fluid flow and lymphatic drainage (Miller & Seale, 1981; Reddy et al., 1981), ischemia from capillary closure (Herrman et al., 1999; Gebhardt, 2002; Jan & Brienza, 2006; Jan, Liao & Foreman, 2012; Lustig et al., 2018; van Damme et al., 2020), and reperfusion injury (Peirce et al., 2009; Tsuji et al., 2005; Unal et al., 2001). To date, the precise aetiology of PIs remains a complex and multifactorial phenomenon that is not clearly understood.

Conventionally, localised ischemia was considered the dominant aetiological factor associated with PI development (Bader et al., 1986; Dinsdale, 1974; Kosiak, 1961). The

ischemia theory proposes that tissue ischemia—resulting from the sustained occlusion of capillaries at the compressed weight-bearing region—gradually leads to insufficient local tissue perfusion and, consequently, the formation of a PI. Although the ischemic pathway is well-documented in the research literature as the final common pathway to pressure ulceration, it does not account for the aetiological pathways initiated by cellular and tissue deformation (Gefen et al., 2021).

Sustained deformations of skin and deeper tissues, either due to bodyweight loads or exerted from the environment, must be present in order to cause the initial cell death and tissue damage that, eventually, result in PI formation (Gefen et al., 2021). Specifically, sustained tissue deformation ultimately leads to loss of structural integrity in cells, disrupts the oxygen transport to cells and, eventually, results in cell death. The first cell death events further trigger an inflammatory response, which generates inflammatory oedema (Lustig et al., 2015) that, in turn, raises the interstitial pressure and exacerbates the mechanical loads on cells and tissues in regions confined between the bony prominence and support surface (Gefen et al., 2021; Lustig et al., 2018). After several hours of exposure to sustained bodyweight load and under the influence of elevated interstitial pressure, ischemic damage may begin to build-up, accelerating the overall extent and rate of tissue damage (Lustig et al., 2018). As such, several mechanisms appear to induce cellular and tissue damage or increase the vulnerability of the cells and tissue to damage.

Gefen et al. (2021) postulates a possible damage cascade in PIs, which includes sequential cell and tissue damage associated with direct deformation (first factor), damage associated with the inflammatory response (second factor), and damage induced by ischemia (third factor) (Lustig et al., 2018; Oomens et al., 2015; van Damme et al., 2020). Each of these three factors contributes to the cumulative cell and tissue damage, which has been theorised to develop in an escalated manner as a result of the added contributions of the above factors. In other words, the damage pathway can be described as a vicious cycle of cell and tissue damage resulting from sustained mechanical deformations (the triggering event), which inflicts the primary, direct deformation damage (first damage event), then leading to secondary inflammatory oedema-related damage (second damage event), and finally to tertiary ischemic damage (third damage event).

An important observation is that while it is likely that ischemic damage follows from the inflammatory damage, ischemia alone may also be present prior to the onset of a PI (for

example, due to persistent peripheral vascular disease or prolonged pressure-induced capillary closure) or develop independently from or concurrently with the inflammation (for example, as a result of a thrombotic event) (Black & Cuddigan, 2020). Once the cycle of a PI enters the ischemic phase, biochemical stress develops in the affected tissues due to the lack of adequate blood supply and impaired clearance of metabolic waste products. Specifically, the ischemia resulting from inflammatory oedema (through an increase in the interstitial pressure level that distorts the vasculature) may lead to hypoxia and impaired removal of carbon dioxide and nitrogenous compounds (Gefen et al., 2021). Deprivation of nutrients and decrease in the pH of the interstitial fluid towards a more acidic extracellular environment, due to the accumulation of metabolic waste products, will eventually lead to tissue necrosis (Gawlitta et al., 2007).

2.8 Critical Determinants of Pressure Injury Development

Several PI conceptual frameworks that provide the basis for understanding the critical determinants of PI development have been proposed over the course of the last few decades. Braden and Bergstrom (1987), in their conceptual model, implicated intensity and duration of interface pressure, as well as tissue tolerance, as critical determinants of PI development. The latter related to the ability of the skin and its underlying structures to tolerate pressure without damage. It was proposed that tissue tolerance would be influenced by extrinsic factors (skin surface moisture, friction and shear) and intrinsic factors (metabolic supply and demand, pressure distribution capacity, and threats to skin integrity). Defloor (1999) developed a conceptual scheme that highlighted the importance of pressure and shearing forces, while recognising the importance of tissue tolerance. However, Defloor (1999) viewed tissue tolerance as an 'intermediate variable' and not as a 'causal factor'. Benoit and Mion (2012), who developed their conceptual model of PI development for critically ill patients, also incorporated interface pressure and tissue tolerance as key variables implicated in PI development.

Another conceptual framework was proposed by the EPUAP-NPUAP (2009) and was based on two major domains. The first domain encompasses 'mechanical boundary conditions', which include the type of loading (pressure, shear, friction) and magnitude and duration of the mechanical load applied to the skin-support surface interface (Coleman et al., 2014). The second domain encompasses 'susceptibility and tolerance of the individual', and includes factors such as individual physiology, mechanical properties and geometry (morphology) of the tissues, sensory perception, age, and ethnicity (Chung et al., 2022;

Coleman et al., 2014). There are factors that can also exert an effect on both the mechanical boundary conditions and on the susceptibility and tolerance of the individual; therefore, some overlap exists between the two major domains. For example, diabetes, as a chronic disease, can affect both the mechanical boundary conditions through sensory deficits, and individual tolerance and susceptibility through altered perfusion (Alderden et al., 2017).

As such, it is clear that a complex interplay of contributing and confounding factors has been implicated in PI development. Despite various models and theoretical assumptions, the development of PI is dependent on the internal tissue response to the mechanical loading state of the individual (Gefen et al., 2021). The mechanical loads applied to soft tissues are all the types of forces that can possibly act upon skin and underlying tissues of an individual as a result of contact between the skin and an external surface, object, or medical device (Gefen et al., 2021). These loads include the bodyweight forces typically transferred through bony structures into soft tissues. Sustained mechanical loading gives rise to two forces acting either perpendicular to the skin (interface pressure) or parallel to the skin (shear). These factors tend to act synergistically to influence the tolerance and vulnerability of the skin and soft tissue to the ischemic effects of sustained mechanical loading (Black, Baharestani & Cuddigan, 2007; Kosiak, 1961; Thomas et al., 2013; Pieper, 2012). Furthermore, Lustig et al. (2018) describes that prolonged interface pressure and shear cause focal deformations and mechanical stress concentrations in the underlying tissues at the contact sites. There is a large volume of literature that postulates interface pressure and shear as the primary contributing factors to tissue ischemia associated with PI formation (Bergstrand et al., 2010; Gawlitta et al., 2007; Jiang et al., 2011; Salcido et al., 1994; Shilo & Gefen, 2012). The next sections provide a detailed overview of interface pressure (Section 2.8.1) and shear (Section 2.8.2) and their impact on soft tissue.

2.8.1 Interface Pressure

Interface pressure, measured in millimetres of mercury (mmHg), is the perpendicular force per unit area between a support surface (such as a bed mattress) and the body (Peterson et al., 2013). Interface pressure is exerted when the support surface (in contact with the skin) and the weight of the patient's skeleton compress soft tissue (Kanj et al., 1998). Tissue ischemia will develop if the underlying capillaries are compressed enough to impede perfusion (Gefen, 2011). Specifically, oxygen delivery will be compromised if the pressure applied over a given anatomical region is greater than the capillary-closing pressure. If pressure at the compressed tissue is not relieved within four to six hours of sustained loading

(Edsberg et al., 2014; Gefen at al., 2009), the vessels extending to the dermal papillae collapse, compromising adequate tissue perfusion (Dini et al., 2006). The occlusion of the microvascular capillary network of the skin and deeper tissues at the affected area eventually precipitates early onset PI (Bouten et al., 2003; Rithalia & Gonsalkorale, 2000). While it is universally recognised that both the intensity and duration of pressure are of prime relevance in the development of PIs, it is difficult to discern the relative contribution of these two parameters (Coleman et al., 2014).

Kuffler (2010) and Peterson et al. (2013) reported that the processes implicated in tissue ischemia occur rapidly, with interface pressures as high as 150mmHg generated in as little as one to two hours at compressed bony prominences of the body. Similarly, Okonkwo et al. (2019) claim that in patients with high risk for PI formation, non-blanchable erythema can develop in as little as two hours from injury. A seminal study (Kosiak, 1961) found that when sitting naturally, only a small amount of interface pressure (limited to a period of one to two hours) could lead to superficial pressure-induced ischemia. Bodyweight, when in the sitting position, is redistributed over a smaller area, creating higher interface pressures at the weight-bearing bony prominences. Tissue that is weakened by ischemic damage may be particularly vulnerable to the effects of shear. Within the literature, shear is regarded as critical factor implicated in the processes that give rise to PI formation (Lustig et al., 2020; Schwartz et al., 2018; Shaked & Gefen, 2013; Stekelenburg). The concept of shear is discussed below.

2.8.2 Shear

Within the biomedical engineering literature, shear is defined as a mechanical force that acts internally on the skin tissue in a direction parallel to the body's surface (Hess, 2004). Shear forces can be iatrogenic (for example, occurring when a patient is transferred across the bed surface) or can result from a combination of pressure and movement (for example, occurring when a semi-reclined patient slides down the bed due to gravity) (Pieper, 2012). Thus, anatomical regions most vulnerable to shear are those on which the body is frequently supported when in a position which allows forward slide, such as when a patient is positioned semi-recumbent. These regions typically include the scapulae, olecranon, coccyx, sacrum and heels (Bhattacharya & Mishra, 2015; Collier & Moore, 2008).

Shear force is generated by the mechanical motion of bone and subcutaneous tissue relative to the skin while the skin is fixed (Lachman & Kottner, 2011). In other words, the

skeleton and deep fascia slide downward with gravity, while the skin and upper fascia remain in their original position (Bennett & Bok, 2001). As the skin layers move relative to one another, the subsequent sliding pressure is transmitted to deeper tissues, which can become angulated and occlude blood vessels and capillaries, disrupting the local micro-circulation (Feuchtinger, Halfens & Dassen, 2005). Al Aboud and Manna (2001) explain that the sliding motion causes the bony structures to move upwards, whilst the overlying skin moves downward (i.e., the two forces move in opposite directions). The middle tissue layers (which supply and perfuse the dermis and epidermis) may tear, leading to decreased perfusion. In other words, when tissue sliding occurs, blood vessels approaching the skin surface will perpendicularly bend and occlude blood flow at the connective layers between the tissue. Thus, shear will increase the effect of pressure by impeding perfusion and causing impaired blood flow, ultimately resulting in tissue necrosis (Goosens, et al., 1994; Hoogendoorn et al., 2017; Manorama et al., 2013). However, if shear stress is reduced, tissues can generally tolerate higher pressures without blood flow occlusion (Bennett et al., 1979).

2.8.3 Soft Tissue Responses to Sustained Pressure and Shear

As outlined in Section 2.7 of this thesis, the most widely adhered to theory of PI development is that compressive mechanical loading causes occlusion of capillary blood flow, resulting in localised ischemia (Daniel et al., 1981, Dinsdale; 1964; Kosiak, 1961). The duration of time for which cells and tissues can endure ischemia without irreversible damage differs for various tissue types. For example, skeletal muscle tissue tends to be more susceptible to ischemic damage than skin due to the greater capillary and mitochondrial volume density in muscle, which is associated with their greater metabolic demand (Linder-Ganz & Gefen, 2007; Salcido et al., 1994; Shilo & Gefen, 2012; Stekelenburg et al., 2005). PIs that are initiated in muscle tissue are generally more extensive and develop at a faster rate than superficial PIs (Breuls et al., 2003). In animal experiments, the first signs of ischemic damage in skeletal muscle occurred after two to four hours of sustained deformations (Bader et al., 1986; Gawlitta et al., 2007; Linder-Ganz et al., 2006; Loerakker et al., 2011). In contrast, the skin is considerably stiffer than muscle and, therefore, deforms to a lesser extent which, in turn, makes it less susceptible to ischemic damage (Gefen et al., 2021; Linder-Ganz & Gefen, 2007). Using an in-vitro model, Bronnenberg et al. (2006) reported that prolonged mechanical loading, over a 20 hour period, led to a visibly altered stratum corneum, superficial tissue damage, and a small decrease in tissue viability. Similarly, Rawlings et al. (1995), also using in-vitro model systems, found that prolonged deformation of the stratum

corneum leads to damage of the microstructure of the intercellular bilayers, impacting skin barrier function.

Studies indicate that the formation of superficial PIs, which onset as skin damage, and those that originate and form in the deeper tissue layers (Agam & Gefen, 2007) appear to be dependent on the nature of the surface loading (Black & Berke, 2020; Breuls et al., 2003; Daniel et al., 1981, Dinsdale; 1964; Kayser et al., 2019; Kosiak, 1961; Salcido et al., 1994; Zhang & Roberts, 1993). As previously outlined in Section 2.5 of this thesis, PIs can present as superficial loss of the epidermis (that progresses to deeper tissues if the interface pressure remains unrelieved) or as deep tissue injury (DTI) with necrosis of muscle and subcutaneous tissue before destruction of the superficial layers and the appearance of a DTI (Gefen et al., 2021). Gefen et al. (2021) explain that deeper ulceration predominantly results from sustained tissue compression and high pressures in combination with shear at the surface of the bony prominences, or under a stiff medical device where tissues are continuously distorted between the device and musculoskeletal structures. Conversely, superficial PIs seem to be primarily caused by excessive shear *strain* (a measure of the relative deformation) and *stress* (force transferred per unit area) exposure at the skin surface.

Under normal physiological circumstances, external compression of the blood vessels underlying a bony prominence stimulates protective reflex movements to relieve pressure (Bhattacharyal & Mishra, 2015). This prompt pressure relief is essential to restore the underlying micro-circulation at the previously-compressed tissue (Sterner at al., 2011). Immediately upon pressure relief, the local capillaries begin to dilate, facilitating a rapid and large increase in cutaneous blood flow at the affected tissue. If the lymphatic vessels of the dependent tissue are intact, and excess interstitial fluid resulting from the acute rise in capillary flow is removed, then permanent tissue changes will not progress (Bridel, 1993). The rush of incoming arterial blood that occurs as pressure is released is commonly referred to as reactive hyperaemia (Bliss, 1998; Kosiak, 1961; Shea, 1975). The concept of reactive hyperaemia is detailed below.

2.8.3.1 Reactive Hyperaemia

PIs typically represent a continuum of tissue damage of ischemic aetiology, primarily initiated by prolonged unrelieved pressure with a subsequent reduction in blood perfusion to the local cells (Black et al., 2007; Kosiak, 1961; Thomas et al., 2013; Pieper, 2012). Below a certain damage threshold, however, soft tissue responds to ischemia through several

vasodilatory mechanisms, including reactive hyperaemia (Brienza & Geyer, 2000; Shea, 1975). Hagisawa et al. (2001) describe reactive hyperaemia as a normal compensatory (protective) mechanism that ensures rapid restoration of circulatory activity following a brief episode of interrupted blood flow. Similarly, Bliss (1998) describes reactive hyperaemia as a local vasodilation response which signals the body's attempt to correct a local oxygen debt accumulated during a brief period of tissue ischemia. Kollias and Stamatas (2002) state that reactive hypermania correlates with increased capillary recruitment and diameter in the papillary and reticular dermis and, thus, a transient increase in blood flow and subsequent increase in capillary and superficial plexus haemoglobin concentration. The rate of blood flow increase following load release also depends upon vascular distensibility and the perfusion pressure gradient acting across the papillary micro-vessels (Hagisawa et al., 2001).

Following pressure release, reactive hyperaemia first appears as a bright red transitory flush at the skin surface. This flush is usually termed 'blanchable erythema' as the erythematous area will often blanche (temporarily turn pale) as a haemodynamic response to brief application of pressure (Vanderwee et al., 2007). A blanch response is considered to be a characteristic of a healthy dermal capillary network (Sowa et al., 2002). The hyperaemic response will typically last about one-half to three-quarters as long as the ischemic period (Hagisawa et al., 2001). However, if mechanical loading extends beyond a certain damage threshold, reactive hyperaemia will fail to meet the demand for blood supply and, consequently, extensive tissue damage will occur at the affected area (Bliss, 1998). Hagisawa et al. (2001) reported that an impaired hyperaemic response may delay metabolic tissue recovery and, thus, tissue may become toxic following repeated sequences of ischemia. Failure of the reactive hyperaemic response, therefore, remains one of the most important factors in the PI aetio-pathology pathway.

When the reactive hyperaemia cycle ceases to function adequately, a sustained reduction in cellular perfusion, over time, will lead to ischemic ulceration of soft tissue. That is, if accumulated pressure at a compressed bony prominence is not relieved promptly and exceeds the reported skin damage threshold of two hours (Black et al., 2011; Defloor et al., 2005; Lyder, 2003; Reddy et al., 2006; Vanderwee et al., 2007), persistent erythema will appear at the affected skin surface. Persistent erythema is thought to be an inflammatory response associated with early stage PI (Jiang et al., 2011) and, unlike transient erythema, is recognised by darker hues of red that remain unchanged during brief application and release of pressure. For this reason, persistent erythema is often termed 'non-blanchable erythema',

signalling extensive disruption to the local micro-circulation (Halfens et al., 2001). Histopathological examination of non-blanchable erythema has been associated with red blood cell engorgement of the capillaries and endothelial cell damage (Witkowski & Parish, 1982) as well deep tissue injury (DTI) at the bone-muscle interface (Sato et al., 2006). However, as some PIs develop as abnormalities of the deeper tissue rather than superficial skin (Bouten et al., 2003), not all PIs will be preceded by non-blanchable erythema.

Aetiological studies show that early pressure-induced ischemic damage does not always manifest into a visible PI (Halfens et al., 2001; Oomens et al., 2015; Swisher et al., 2015). Some early damage will progress to a PI and some will reverse back to a healthy state depending on a variety of factors, including the individual's overall health, and whether an intervention has been implemented to alleviate pressure. Researchers investigating early stage PIs have demonstrated the inherent reversible nature of early pressure damage (Halfens et al., 2001; Moore et al., 2017; Oomens et al., 2015; Swisher et al., 2015). The application of timely and appropriate interventions *before* the damage threshold is reached will result in tissue 're-setting' to normal homeostasis, primarily by the restoration of oxygen supply and nutrients to the tissue and removal of waste products (Halfens et al., 2001). Any area with erythematous discolouration will usually resume its normal colour within 24 hours (Romanelli et al., 2006; Shi et al., 2020; Vanderwee et al., 2007), thus, further degradation of the skin and soft tissue will be halted. Oomens et al. (2015) state that because pressure-induced tissue damage is initiated at a cellular level, timely off-loading the affected tissue is able to restore vascular perfusion in the previously-compressed area.

2.9 Factors Contributing to Pressure Injury Development

In patients who are bedbound, the tolerance of skin is constantly challenged by factors affecting it from the outside in (i.e., frictional forces at the skin surface), as well as changes in skin microclimate conditions (i.e., when the skin is moist or wet) (Derler et al., 2012; Gerhardt et al., 2008). The combined effects of friction and moisture can considerably influence the physical and biomechanical interactions between the skin and deeper tissues (Kottner et al., 2011). The next sections provide an overview of friction and skin moisture, and describe their impact on skin integrity.

2.9.1 Friction and Skin Moisture

Friction is described as mechanical force exerted when skin is dragged across a coarse surface (Gerhardt et al., 2008). Repeated dragging (rubbing or sliding) of the skin

against textiles eventually causes altered stress distribution and mechanical destruction of the stratum corneum (Hashmi et al., 2013). Grey, Harding, and Enoch (2006) state that over time, the effects of friction precipitate the formation of epidermal blisters and superficial abrasions, potentiating the risk of deeper tissue injury. Friction-induced skin damage is particularly common in older adults with increased skin fragility (Budri et al., 2021; Jaul et al., 2018). Although two separate phenomena, the mechanical forces of friction and shear, exerted at the skin surface, act synergistically to accelerate soft tissue breakdown (Coleman et al., 2014). However, shear forces typically influence deeper tissue structures via a 'bottom-to-top' mechanism of tissue injury, while friction forces have the greatest impact on superficial layers of skin via a 'top-to-bottom' mechanism of tissue injury (Hoogendoom et al., 2017; Stechmiller et al., 2008).

The effect of frictional forces at the skin surface are particularly deleterious when the skin is moist or wet (Bennett et al., 1979; Derler et al., 2012; Goosens et al., 1997; Rotaru et al., 2013). It has been widely-acknowledged within the literature that skin moisture plays an integral role in the mechanical interactions between the skin and the support surface and, thus, may indirectly contribute to PI formation in susceptible patient populations. Numerous studies (Ayello & Lyder, 2007; Bates-Jensen, 2007; Clark et al., 2011; Elsner et al., 1990; Harrow & Mayrovitz, 2014; Gefen, 2011; Gerhardt et al., 2008; Goretsky & Supp, 1995; Romanelli et al., 2006; Schwartz et al., 2018; Sergio et al., 1981; Shaked & Gefen, 2013; Zeevi et al., 2018) indicate that skin moisture (for example, arising from perspiration, wound drainage, or urinary/faecal incontinence) increases tissue susceptibility to the mechanical effects of pressure, shear, and friction. A possible mechanical connection between skin moisture and pressure effects has been suggested from a study (Lombard, 1912) conducted more than 100 years ago. Lombard (1912) found that the amount of pressure required to compress capillaries was reduced when skin was moist rather than dry (Lombard, 1912). This phenomenon may be attributed to a reduction in the moist skin's stiffness and less elastic resistance to oppose a pressure load (Boyce, Supp & Harriger, 1996; Clark et al., 2010; Faergemann et al., 1983; Palenske & Morhenn, 1999).

If not managed effectively, excessive and prolonged moisture at the skin-support surface interface may precipitate epidermal softening that, in turn, may diminish the mechanical strength of the stratum corneum (Harrow & Mayrovitz, 2014). These processes impact the ability of the skin to withstand frictional forces and are considerably more pronounced in moist/wet skin (Gray et al., 2007). Furthermore, less friction is required to

abrade or blister skin when it is soft/damp (Gray & Weir, 2007; Kemp, 1994). Materials with a higher 'wettability', including skin, generally show a larger increase in the coefficient of friction (COF)[2] when exposed to moist conditions (Klassen, de Vries & Masen, 2017; Schwartz et al., 2018). Accordingly, support surfaces characterised by high COF, or for which the COF increases substantially due to wetness (perspiration, exudates, or other bodily fluids), represent an additional risk for skin integrity. The magnitude of this COF influences the intensity of the frictional forces acting on the body and, ultimately, the skin and subdermal tissue deformations resulting from any frictional sliding movements between the skin and a support surface (Schwartz et al., 2018; Shaked & Gefen, 2013; Zeevi et al., 2018). The resultant effect is erosion of the epidermal tissue which diminishes the skin's efficiency as a protective barrier, heightening its vulnerability to PI.

The barrier function of the stratum corneum is strongly related to the property of intercellular lipids that fill the gaps between the corneocytes (Bouwstra et al., 2000). Studies have found that excessive moisture between the skin and support surface disrupts the intricate molecular arrangement of intercellular lipids in the stratum corneum and the intercellular connections between epidermal cells (corneocytes) (Baranoski & Ayello, 2008; Voegeli, 2013; Yoshimura et al., 2015; Yusuf et al., 2015). These findings are supported by early work of Warner et al. (2003) who demonstrated that extended exposure (four hours) of the skin to moisture (distilled water) led to extensive disruption of the intercellular lipid lamellae in the stratum corneum. The exposure of the stratum corneum to water for four hours resulted in a three-fold expansion of the stratum corneum thickness. Specifically, by four hours of water exposure, there were numerous intercellular pools of water (cisternae) present throughout the stratum corneum. Studies indicate that prolonged over-saturation of the skin precipitates maceration (i.e., skin softening characterised by a whitened appearance), which weakens the stratum corneum and renders the skin vulnerable to the deleterious effects of superficial frictional forces (Clark, Romanelli & Reger, 2011; Gerhardt et al., 2008; Ichikawa-Shigeta et al., 2014; Palenske & Morhenn, 1999).

In clinical practice, patients diagnosed with urinary incontinence are at particular risk of moisture-induced skin damage (Falloon et al., 2018; Gray et al., 2002; Ichikawa-

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[2] Coefficient of friction (COF) describes the interface properties and the potential for sliding of two surfaces with respect to each other (Gefen et al., 2021). COF is expressed at the ratio of the frictional force resisting the motion of two surfaces in contact to the normal force pressing the two surfaces together (Brown & Brengelmann, 1965).

Shigeta et al., 2014). In a recent systematic review and meta-analysis, Beeckman et al. (2014) confirmed that individuals with urinary and/or faecal incontinence are 4.99 times more likely (95% CI, 4.07-4.23) to develop PIs than those who are continent. Moisture in the form of urine may become 'trapped' between the skin and support surface (such as a continence aid or textiles), extending into the perineum, skin folds between the buttocks, and down the inner thighs. In the absence of nursing interventions targeted at adequate moisture-control at the affected regions, the resultant outcome is cutaneous irritation and excoriation with possible epidermal erosion. While epidermal erosion may initially remain superficial, it can quickly progress to affect larger areas of the skin (Gray et al., 2002; Gray & Weir, 2007).

The aforementioned cutaneous changes associated with the exposure of the skin to prolonged moisture are encapsulated by the notion of a disrupted 'skin microclimate' whose role in the aetiology and evolution of PI is becoming increasingly recognised (Bader et al., 2018; Gefen, 2011; Kottner et al., 2018; Yusuf et al., 2015). For the purpose of this thesis, the role of skin microclimate, as a theoretical contributor to PI development, is discussed in Chapter 3 of this thesis.

As such, it can be inferred from the literature that PIs represent a breach of skin integrity and continuum of tissue damage of ischemic aetiology (Jiang et al., 2011; Kosiak, 1961; Shilo & Gefen, 2012), typically occurring at the skin and soft tissue overlying the weight-bearing bony prominences of the body. The processes implicated in tissue ischemia underlying the compressed bony prominences can occur rapidly, having been reported to occur in as little as two hours (Defloor et al., 2005; Lyder, 2003; Reddy et al., 2006). The detrimental effects of interface pressure may be further enhanced by mechanical boundary conditions of shear and friction. Shear stress can lead to tissue deformation and distortion of the deeper layers, thus, comprising blood supply. Furthermore, dynamic conditions such as repositioning or transferring of a patient across textiles can create frictional forces at the skin surface that can act to disturb the barrier function of the stratum corneum. Finally, excessive skin surface moisture (as a microclimate variable) may exacerbate the effects of pressure, shear and friction, thereby, accelerating the skin damage pathway.

2.10 Conclusion

A review of the literature has reinforced that PIs are a serious and prevalent problem in healthcare settings globally, imposing a tremendous burden to both individuals and healthcare systems. The impact of PIs on the quality of life of those affected is significant,

with the occurrence of a PI documented to have detrimental effects on the individual's physical, emotional, social and financial health dimensions. The impact of PIs from both a quality of life and a financial perspective is influenced by their severity. Furthermore, many chronic and non-healing PIs have been reported to contribute to early mortality in some individuals.

In accordance with the NPUAP International Pressure Injury Classification System (NPUAP, 2019), PIs are staged according to the extent of the associated tissue disruption. A PI can present itself at a bony prominence of intact skin, or as an open wound with underlying soft tissue damage. As such, PIs represent a continuum from an erythematous area of intact skin (denoting superficial tissue disruption) to an open wound extending deep into the tissue or injury with an unknown depth. Anatomical regions where bone is in close proximity to the overlying skin are regions considered most susceptible to PI formation, due to their limited ability to withstand the effects interface pressure, shear and friction.

PIs typically develop as a result of a combination of physiological events and external conditions. The primary cause of PIs is sustained mechanical loading in the form of pressure, or pressure in combination with shear, applied to soft tissues. Mechanical loading of soft tissue gives rise to two forces acting either perpendicular to the skin (interface pressure) or parallel to the skin (shear). Shear has also been implicated as a causal factor, as it can cause stretching and tearing of blood vessels, which reduces blood flow and creates cellular damage. In addition, prolonged periods of repeated friction at the skin surface can accelerate the progression of damage of the superficial skin layers. Ultimately, the tolerance of the skin and soft tissue to the effects of interface pressure, shear and friction determines its vulnerability to PI formation.

Several theories have been proposed pertaining to the development of PIs. The most widely adhered to theory is that compressive mechanical loading causes occlusion of capillary blood flow, resulting in localised ischemia (Daniel et al., 1981, Dinsdale; 1964; Kosiak, 1961). It has been postulated within the literature that below a certain damage threshold, soft tissue responds to ischemia through several vasodilatory mechanisms, including reactive hyperaemia. This rapid increase of vascular perfusion in the upper dermis is viewed as a normal physiological response to correct a local oxygen debt accumulated during ischemia. A cardinal symptom of reactive hyperaemia is erythema which, provided that the affected region is promptly off-loaded, will typically resolve on its own. However, if

the mechanical loading extends beyond a certain damage threshold, reactive hyperaemia will fail to meet the demand for blood supply, causing the surrounding tissue to be starved of oxygen and nutrients; ischemia will continue to progress and produce more severe and extensive damage manifesting as early onset PI.

Recent evidence (Bader et al., 2018; Gefen, 2011; Kottner et al., 2018; Yusuf et al., 2015) has implicated the role of the microclimate parameter of skin moisture—in the vicinity of the skin-support surface interface—as a theoretical contributor to PI development. Thus, capturing elevations in skin moisture is a relevant consideration in contemporary approaches to PI risk assessment.

2.11 Chapter Summary

This chapter has provided a comprehensive background on PIs and contextual information pertinent to this thesis. The significance of PIs has been considered, and their prevalence rates have been reported. The classification of PIs has been detailed, and the anatomical regions prone to PI development have been outlined. The aetiological processes proposed in PI development have been explained, and the conceptual frameworks that provide a theoretical model of biomechanical and physiological conditions that influence PI development have been detailed. The chapter concludes that the PIs are initiated by a series of physiological events, including blood vessel occlusion as a result of interface pressure, disruption to the local micro-circulation due to shearing forces, and friction-induced erosion of superficial skin layers. Recent evidence suggests that the effects of pressure, shear, and friction may be compounded by changes in the local skin microclimate between the skin and support surface. The next chapter takes a deeper look at the relevant concepts that relate to the skin microclimate and its theoretical contribution to PI formation.

CHAPTER 3

Skin Microclimate in Pressure Injury Development

3.1 Chapter Overview

This chapter provides a comprehensive review of the literature pertaining to the concept of the skin microclimate and its relevance to PI development. It begins by providing a broad overview of skin microclimate and defines each parameter involved in the current understanding of skin microclimate. An overview of the skin microclimate variables of skin moisture and skin temperature are subsequently provided, followed by an overview the stratum corneum hydration as a relevant dimension in the concept of skin microclimate. Finally, the chapter draws upon previous and the contemporary literature investigating the interplay between the skin microclimate parameters of skin moisture and skin temperature and their association to PI development.

3.2 Introduction to the Concept of Skin Microclimate

As demonstrated by a long-standing body of evidence, it is widely-acknowledged that sustained mechanical loading, in combination with the synergistic effects of shear and friction, are viewed as the most important factors implicated in PI development. More recent evidence presented from key studies (Clark et al., 2010; Denzinger et al., 2020; Falloon et al., 2018; Gefen, 2011; Gefen et al., 2021; Kottner et al., 2018; Lachenbruch, 2005; Reger, Ranganathan & Sahgal, 2007; Schwartz et al., 2018; Schwartz & Gefen, 2020; Yusuf et al., 2015; Zeevi et al., 2018) implicates the microclimate of the skin as a contributing factor to PI development. Based on the available in-vivo and animal-based literature that has been published, it has been suggested that the microclimate at the skin surface can impact the integrity of the epidermal barrier. Thus, establishing an optimal skin microclimate may appear to be a critical factor in deterring the formation of superficial PIs.

The microclimate of the skin is typically considered to reflect the 'external environment' in the vicinity of the skin-support surface interface (Yusuf et al., 2015). In the literature, skin microclimate has traditionally been used to describe the interaction between skin surface moisture and/or humidity, skin temperature, and air movement adjacent to the skin (Clark & Black, 2011). The theory of the 'interaction' between the skin microclimate parameters has emerged over the recent years as a new area for exploration with regard to PI aetiology and pathophysiology (Denzinger et al., 2020; Forriez et al., 2017; Gefen, 2009;

Kottner et al., 2015; Kottner et al., 2018; Posada-Moreno et al., 2011; Roaf, 2006; Sae-Sia et al., 2005; Yoshimura et al., 2015). To date, there is evidence to suggest that alterations in skin microclimate conditions, specifically at the skin-support surface interface where the environment tends to be occlusive, can influence susceptibility and tolerance of the skin barrier to the mechanical effects of pressure, shear and friction. However, there is much that remains unknown in terms of the interaction between microclimate variables and superficial skin changes. Understanding these unknowns may contribute to enhanced identification of skin changes precipitating PI formation.

3.3 Skin Moisture/Humidity as Skin Microclimate Parameters

In a skin microclimate context, moisture generally refers to 'dampness' or 'wetness' immediately above the skin surface (Clark & Black, 2011). The presence of skin surface moisture can be caused by internal mechanisms such as pyrexia, or arise from external factors such as a humid environment. The term 'humidity' is often used interchangeably with 'moisture', however, their meaning is distinct. In broad terms, humidity refers to heat accumulation between the skin and the support surface (Clark, 1996). Specifically, humidity refers to the amount of water vapour in the air, i.e., absolute humidity (expressed as the weight of water in grams per cubic metre of air (g/m3)) and relative humidity (a ratio expressed as a percentage that relates the amount of water vapour in the air at a specific air temperature to the maximum amount of water vapour that body of air would hold at that temperature) (Clark, 1996). The relative humidity of the general surroundings is known as ambient humidity. Absolute humidity is affected by air temperature; warmer air is able to hold more water vapour than cooler air. Therefore, for the same absolute humidity, warmer air will have a lower relative humidity than cooler air (Schäfer et al., 2002).

3.3.1 Skin Moisture and Superficial Skin Changes

Disruptions to the skin microclimate variable of skin moisture can contribute to the development of superficial PI though two mechanisms of action. First, disruptions in skin moisture can affect the mechanical boundary conditions. As outlined in Chapter 2 of this thesis, mechanical boundary conditions include the type of loading (pressure, shear, friction) and magnitude and duration of the mechanical load applied to the skin at the interface with the supporting surface (NPUAP, 2019). Studies suggest that moisture at the skin surface can influence the degree to which the skin and tissue can tolerate the magnitude and duration of the mechanical load, the time duration of the applied mechanical load, and type of loading (pressure, shear, friction) applied at the skin-support surface interface. Second, disruptions

in skin moisture can influence the susceptibility and tolerance of the mechanical properties of the skin (specifically the fibrous protein, keratin, of the stratum corneum) to pressure, shear and friction (Coleman et al., 2014; Wildnauer et al., 1971; Zhong et al., 2008).

The sustained contact of the stratum corneum with moisture can precipitate skin softening and maceration (Derler & Gerhardt, 2012; Gray et al., 2011; Schnelle et al., 1997). Softened skin has significantly diminished mechanical strength and tolerance to pressure, shear and friction, making superficial damage through abrasion more likely. Gerhardt et al. (2008) and Gefen (2011) reported an association between elevated skin moisture levels and an increased coefficient of friction (COF)[3] between the skin and support surface. Sustained weight-bearing at the bony prominences—in combination with moisture at the skin-support surface interface—affects the microtopography (roughness) features of the skin which, in turn, affects the skin's COF with the contacting support surface (Dobos et al., 2015; Schwartz et al., 2018; Shaked & Gefen, 2013; Sopher et al., 2011). Any alterations in the COF will increase the skin's adherence to an outside support surface. This can be observed when a patient is transferred across a stationary/course surface such as bedlinen, which creates a 'drag effect'. Over time, these events accelerate the mechanical irritation and destruction of the stratum corneum, impacting the integrity of deeper cutaneous layers and precipitating superficial skin damage (Schwartz et al., 2018).

Flam (1995) postulated that the risk of friction-induced skin damage at pressure-prone areas in a loaded state increases five-fold in the presence of skin wetness. Rotaru et al. (2013) measured the COF between the forearm of three subjects and hospital sheets in wet and dry conditions. The COF values[4] for the 'wet condition' ranged from 0.5 to 0.95, while the COF in the 'dry condition' ranged from 0.2 to 0.4. Xu et al. (2020) further explained that the COF between the skin and textiles strongly depends on the volume of moisture accumulated over the skin. It should be noted that the moisture accumulation on the skin surface should not exceed 35 g/m^2 (Xu et al., 2020); moisture below this threshold will be absorbed by the materials in direct contact with the skin.

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[3] The term friction is used as an abbreviation of the coefficient of friction (COF) to describe interface properties and the potential for sliding of two surfaces with respect to each other (Gefen et al., 2021).

[4] COF values are usually between 0 and 1; a value of 0 indicates zero friction between two surfaces or objects (Bronzino et al., 2005).

The combination of friction and moisture at the skin-textile interface frequently contributes to epidermal ulceration in patients with poorly-managed incontinence (Clark, 1996; Goossens et al., 1997; Park & Choi, 2016). In the absence of nursing interventions targeted at adequate moisture-control at the affected regions, epidermal erosion may occur. While epidermal erosion may initially remain superficial, it may quickly progress to affect larger areas of the surrounding skin (Gray et al., 2002; Gray & Weir, 2007). Thus, regular monitoring of the skin and responding to raised skin surface moisture constitutes a major component of PI risk assessment and management (Braden & Bergstrom, 1987).

Although the skin microclimate parameter of moisture generally refers to the moisture contiguous to the skin surface, skin moisture can also be a measure of the moisture content within the internal layers of the stratum corneum (Elias, Feingold & Kenneth, 2005). The moisture content within the stratum corneum is a biophysical skin property and, as such, can only be evaluated by capacitance or conductance-measuring devices (Clarys & Barel, 1995). In regard to the literature pertaining to the skin microclimate, the term 'stratum corneum moisture' is often interchangeable with 'stratum corneum hydration'.

3.4 Stratum Corneum Hydration as a Relevant Dimension to Skin Microclimate

The stratum corneum is the outermost layer of the epidermis (Figure 3.1) and is the layer of the skin exposed to the outside environment (Elias, Feingold & Kenneth, 2005). The stratum corneum, with a thickness of 10 to 30 micrometres (µm) (Lademann et al., 2008), is comprised of a two-compartment system; corneocytes that are flattened dead cell bodies of keratinocytes covered in a tough, highly cross-linked protein cornified envelope, and an extracellular matrix filled with lipid lamellae. The latter system consists of roughly equimolar concentrations of ceramides, long-chain free fatty acids, and cholesterol released from lamellar bodies of the uppermost keratinocytes in the stratum granulosum (Elias & Menon, 1991). The stratum corneum's layers of non-viable keratinised cells form a tough impermeable barrier to the environment (Boyle et al., 2019) and, thus, help prevent the penetration of microbes and dehydration of underlying tissues. In addition, these keratinised cells provide mechanical protection against abrasion of the more delicate underlying layers (Elias & Menon, 1991). Cells in the stratum corneum are shed periodically and are replaced by cells pushed upwards from the stratum granulosum (or stratum lucidum in the case of the palms and soles of the feet). This entire layer is replaced during a period of about four weeks (Elias & Menon, 1991). The inner layers of the stratum corneum form the viable epidermis, where keratinocytes proliferate and differentiate to replenish the stratum corneum.

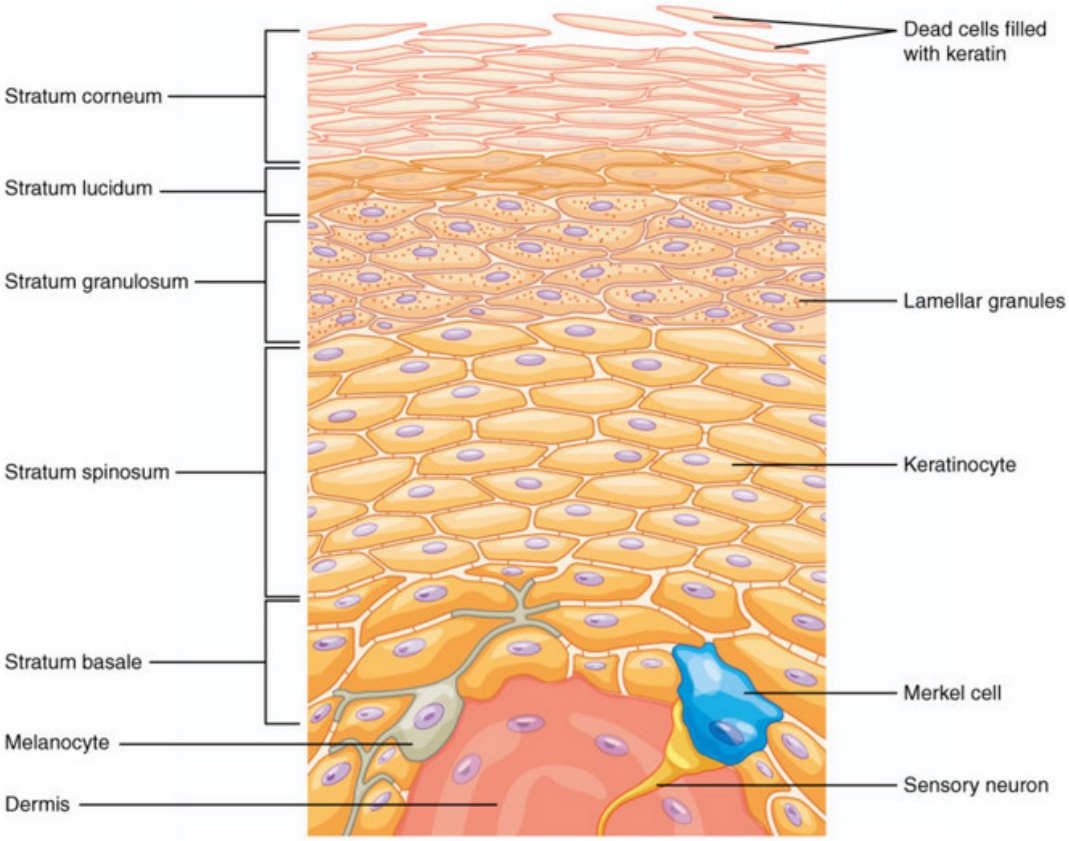

Figure 3.1 The five layers of the epidermis: stratum basale, stratum spinosum, stratum granulosum, stratum lucidum and stratum corneum. Reprinted with permission from "Module 24: The Integumentary System," by Lumen Biology Majors, 2019, Lumen (https://courses.lumenlearning.com/wm-biology2/chapter/epidermis/). Copyright 2019 by Lumen.

The stratum corneum stores about 30% of fluid, varying from around 40% in the inner layers to around 10 to 15% in the outermost layers (Agache & Black, 2004). Hygroscopic glycerol (a component of the stratum corneum derived from sebum) binds water, whereas the lamellar intracellular lipids prevent easy passage of water through the stratum corneum (Fluhr et al., 2003; Horii et al., 1989; Imokawa, Kuno & Kawai, 1991; Rawlings et al., 1994). As such, the stratum corneum performs an important function; it keeps the skin smooth and flexible by holding water even in an external dry environment (Rawlings et al., 1994; Rawlings, Canestrari & Dobkowski, 2004). Although vitally important for stratum corneum homeostasis and flexibility, water also has other critical roles in the stratum corneum. Water acts as a plasticiser on corneocyte proteins, giving elastic properties to the cells (Rawlings et al., 1994). The corneocytes also contain hygroscopic

compounds called natural moisturising factor (NMF)[5] which are essential for maintaining tissue flexibility and, together with the extracellular lipids, prevent tissue dehydration (Praessler & Fluhr, 2005). Since atmospheric conditions vary largely, the corneocytes are hydrated from bodily water lost through the barrier. This imperfect barrier and inbuilt water loss is relevant for tissue functioning and flexibility, as well as for certain metabolic processes. There is also a water gradient from the lowermost cell layer to the top one of the stratum corneum (Tagami et al., 1980). The deeper-most portion of the stratum corneum that faces the fully hydrated viable epidermal tissue is well hydrated, whereas the uppermost portion is much less hydrated because it is exposed to the atmosphere.

The extracellular lipid matrix in the skin's outermost layer, the stratum corneum, is crucial for skin barrier function. The extracellular lipid matrix that creates the brick and mortar organisation of the stratum corneum regulates permeability, initiates corneocyte desquamation, has antimicrobial peptide activity, and allows for selective chemical absorption (Bosko, 2019; Kasting et al., 2003; Tagami et al., 2008). When brought into contact with an aqueous solution such as water, the keratin-filled corneocytes within the stratum corneum immediately absorb the water. The water first seeps into the spaces between the flakes of the dead stratum corneum. Subsequently, the dead corneocytes become rehydrated, swell and expand, thereby increasing their surface area (Gerhardt et al., 2008). As the stratum corneum is firmly attached to the underlying epidermal and dermal skin layers, the stratum corneum must wrinkle (for example, as seen in 'prune fingers') in order to compensate for the greater surface area. The stratum corneum on the fingers, palms and soles is relatively thick (0.5–1 mm) (Agache, 2004) and, as such, holds more keratin and is able to absorb more water, which makes the wrinkling more evident (Gerhardt et al., 2008).

In the stratum corneum, water can be found in three types or chemical bonding states: primary water, which is tightly bound to keratin in corneocytes; secondary water, which is hydrogen-bonded around the protein-bound water, and; free water (Leveque, 2005; Rawlings & Harding, 2004). Under normal physiological conditions, the stratum corneum water mainly exists in a bound state (Leveque, 2005), and the high osmotic strength within

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[5] NMF refers to a complex mixture of low-molecular-weight, water-soluble compounds first formed within the corneocytes by degradation of the histidine-rich protein known as filaggrin (Elias & Menon, 1991). NMF concentration and moisture content naturally decline towards the surface of the skin (Rawlings & Harding, 2004). Highly-structured lipid lamellae, as well as restricted water movement through the stratum corneum effectively prevent the water-soluble NMF compounds from leaching out of the corneocytes in the surface layers of the skin (Rawlings & Harding, 2004).

corneocytes allows them to 'soak' in water, which prevents the accumulation of water between corneocytes (Wertz & Michniak, 2005). However, when the stratum corneum is completely hydrated, extracellular 'water pools' and 'voids' occur that can disrupt the stratum corneum structure and shift lamellar bilayers (Warner et al., 2003).

3.4.1 Stratum Corneum Hydration and Superficial Skin Changes

Morphological changes of the stratum corneum due to water exposure and/or water uptake are well documented in the literature (Bouwstra et al., 2003; Ho et al., 2000; Mojumdar et al., 2017; Querleux et al., 1994; Rawlings et al., 1995; Sato et al., 2000; van Hal et al., 1996; Warner et al., 1999; Warner et al., 2003). For example, a two-fold reduction in the skin surface roughness (Sato et al., 2000), as well as stratum corneum swelling of approximately 8% in the area dimension, and 26% in the thickness dimension, has been observed under 'water treatment' (Norlean et al., 1997). There is a large volume of the literature which indicates that over-hydration (or over-saturation) of the stratum corneum can influence the skin's vulnerability to mechanical injury (Alanen et al., 2004; Baranoski & Ayello, 2008; Clark et al., 2011; Gerhardt & Strässle, 2008; Palenske & Morhenn, 1999). The prolonged over-hydration of the stratum corneum has been reported to weaken the bonds between epithelial cells, contributing to epidermal softening (Bouwstra et al., 2003; van Hal et al., 1996) and possible maceration (Gerhardt & Strässle, 2008; Schnelle et al., 1997).

Evidence demonstrates that a softened skin barrier renders the skin vulnerable to the effects of frictional forces and, thus, to superficial PI (Ichikawa-Shigeta et al., 2014; Yoshimura et al., 2015). Shaked and Gefen (2013) and Sussman and Bates-Jensen (2007) report that prolonged exposure of the skin to moisture can overwhelm and disrupt the normal moisture regulation mechanisms of the epidermis. The resultant effect is extensive disruption of the intercellular lipid lamellae and corneocytes. As the structured lipids of the stratum corneum constitute the primary permeability barrier of the skin (Elias & Feingold, 2005), it follows that prolonged water exposure may enhance permeation by disrupting lipid organisation. Using transmission electron microscopy, Warner et al. (1999) showed that prolonged exposure of porcine skin to warm (46°C) water, in-vitro, caused extensive disruption of the stratum corneum lipid structure. Alterations of the intercellular lipid structure occurred after two hours of water exposure, while extensive delamination (corneocyte separation) of the intercellular lipid lamellae occurred after six hours, and nearly complete delamination was observed after 24 hours of water exposure.

Warner et al. (2003) extended their investigation to in-vivo studies of over-hydrated human stratum corneum and the lipid organisation of the intercellular space. The researchers employed a 24 hour data collection timeframe. It was confirmed that extended water exposure resulted in extensive disruption of stratum corneum intercellular lipid lamellae. Over-hydration of the stratum corneum was found to induce large pools of water in the intercellular space; at times, exceeding the size of water-swollen corneocytes. By four hours of water exposure, there were numerous small and large intercellular pools of water (cisternae) present throughout the stratum corneum and, at 24 hours, these cisternae substantially increased in size. Within cisternae, the lipid structure was disrupted by lamellar delamination. Warner et al. (2003) concluded that prolonged exposure of the skin to water leads to thickening of the stratum corneum due to corneocyte swelling (three-fold after four hours, and four-fold after 24 hours) and the storage of water-soluble substances in intercellular cisternae. Overall, this study illustrates the disruptive effect of over-hydration on the stratum corneum intercellular space.

Bates-Jensen et al. (2007) examined the relationship between epidermal hydration and direct visual skin assessment of erythema/Stage I PI in nursing home residents (n=35). The researchers sought to determine if epidermal hydration measures could be used to predict the presence of or changes in erythema/Stage I PI. Concurrent to visual assessment of skin moisture, epidermal hydration was measured weekly at the sacrum and buttocks using a dermal phase meter. It was found that higher epidermal hydration scores predicted skin damage (defined as erythema/Stage I PI) visible one week later at the sacrum and buttocks. Bates-Jensen et al. (2007) noted that epidermal hydration values, as registered using the dermal phase meter, were higher when there was no visible skin damage at the time of assessment, but skin damage developed and was apparent on the skin the following week. Overall, the epidermal hydration measures predicted 26% of the erythema/Stage I PIs that had emerged on the skin surface of participants.

A drier stratum corneum been shown to also be undesirable, and may indirectly accelerate the progression of pressure-induced skin damage. Dry skin was reported to be a risk factor for PI development more than 30 years ago (Guralnik et al., 1988; Allman et al., 1995). A more recent study by Baumgarten et al. (2006) showed that PI incidence was associated with dry skin in older hospital patients (p=.035) in a multivariable model. Various studies have reported that skin dryness diminishes the skin's tensile strength (Elias & Menon, 1991; Lechner et al., 2017; Rawlings et al., 1994), making it more prone to the development

of cracking or fissures that permits invasion (via permeation) of micro-organisms at the skin surface (Elias & Menon, 1991). Similarly, Proksch and Lachapelle (2005) indicated that chronic dry skin results in impaired skin barrier function and leads to a reduction of elastic properties. Grey et al. (2006) stated that a reduced amount of skin elastin predisposes the skin to the adverse effects of shear which, in vulnerable skin, can contribute to epidermal blisters and initiate and/or accelerate pressure ulceration.

3.5 Skin Temperature as a Skin Microclimate Parameter

Skin temperature refers to the temperature of the outermost surface of the body (Elias, Feingold & Kenneth, 2005). The surface temperature of the human body is determined by the thermal balance of heat (energy) supplied from the core and subcutaneous tissue and heat lost to the environment via radiation, conduction, convection and evaporation (Pompei, 1996). In order to remain comfortable, the human body must maintain a skin temperature of ~33°C and be in thermal equilibrium with the ambient environment (Houdas & Ring, 1982). However, skin temperature is sensitive to the ambient (external) environment and can vary by several degrees in a short period of time to establish thermal equilibrium (Brienza & Geyer, 2005). Under normal circumstances, heat released by the skin is dispersed rapidly into the environment and is the primary means that that body uses to cool itself. Additionally, heat dissipation ensures that the local skin microclimate is not appreciably different from the surrounding (ambient) environment (Lachenbruch, 2005).

At present, there is no formal reference range for skin surface temperatures of the human body (Armstrong & Lavery, 1997). Skin temperatures can vary widely between person to person and by anatomical region; however, mean skin temperature values are often reported in thermoregulation studies with human subjects. Assuming neutral (21–25°C) ambient temperatures and a core temperature of ~37.0°C, skin surface temperature at the trunk of the human body varies between 33.5–36.9°C, and between 23.5–35.6°C at the extremities (Millington, 1983; Montalto et al., 2013). Furthermore, the skin temperature on any site of the human body is said to be comparable to a symmetrical site under normal physiological circumstances (Jones, 1998). Thus, differences in skin temperature between a target site and a symmetrical contralateral reference point, such as the opposite limb, can be used to provide diagnostic insight into skin temperature changes (Armstrong et al., 1997; Pompei, 1996).

3.5.1 Skin Temperature and Superficial Skin Changes

An example of an extrinsic confounding factor that can impact tissue tolerance to pressure damage is the microclimate parameter of skin temperature (Kottner et al., 2018; Lachenbruch et al., 2013; Zeevi et al., 2018). Skin temperature was first proposed in the conceptual schema of factors in the aetiology of PI development in 1987 by Braden and Bergstrom (1987). To date, there continues to be some evidence apparent in the literature to suggest that sustained elevations in skin temperature affect the mechanical properties of loaded skin. The results of pre-clinical studies (DuBois, 1936; Fisher et al., 1978; Kokate et al., 1995; Stewart, Palmieri & Cochran, 1980) as well as more recent studies (Armstrong et al., 2007; Iazzio, 2004; Sprigle et al., 2001; Zeevi et al., 2018) have shown that warming the skin increases its metabolic demand. Dubois (1936) and Brown et al. (1965) noted that an elevation as small as 1°C in skin temperature accelerates tissue metabolism by approximately 10% which, in turn, increases oxygen consumption. As a result, tissue blood perfusion increases to minimise accumulation of tissue waste, reducing tissue tolerance to ischemia and making the skin more vulnerable to injury at lower pressure levels. As such, any condition that causes skin temperature to rise, in conjunction with interface pressure, would increase tissue susceptibility to ischemic injury.

Gefen (2011) modelled the hypothetical interactions between microclimate properties and the development of superficial skin damage associated with a PI. A mathematical model was used to predict the tolerance of the skin to the combined effects of skin temperature and interface pressure. This mathematical model quantitatively determined that increases in skin temperature, in combination with interface pressure, raised the risk of PI. Gefen's mathematical model predictions in regard to the effects of skin temperature and interacting parameters on the tolerance of the skin to superficial PI are consistent with the literature (Clark, Romanelli & Refer, 2010; Reger & Ranganathan, 2009; Sae-Sia et al., 2005). For example, Sae-Sia et al. (2005) measured sacral skin temperatures in 17 neurologically impaired patients who were positioned supine and laterally on standard foam mattresses. The incidence rate of PI in this patient group was 47% within two weeks of admission. The authors reported that the sacral skin temperature scores in patients who had developed a PI during the study follow-up period were significantly higher, by approximately 1.2°C, compared to those who did not develop a PI. Sae-Sia et al. (2005) noted that regardless of the resting position, the mean sacral skin temperature in patients who developed a PI was ~37.2°C, compared to ~36.0°C in those patients who did not develop a PI.

The interactions between skin temperature and PI development have been widely investigated in animal studies. Patel et al. (1999) investigated the effects of varying local skin temperature on surface pressure-induced changes in skin perfusion and deformation in hairless rats. Skin surface pressure was applied by a computer-controlled plunger with corresponding skin deformation. In the first protocol, skin surface perfusion was measured without heating (control; 28°C), with heating (36°C), and at different skin surface pressures; 3.7mmHg (control), 18mmHg and 73mmHg. Patel et al. (1999) noted that heating caused perfusion to increase at both the control pressure and at 18mmHg, however, not at 73mmHg. In the second protocol, skin perfusion was measured with and without heating as in the first protocol, however, this time, skin surface pressure was increased from 3.7 to 62mmHg in increments of 3.7mmHg. For unheated skin, perfusion increased as skin surface pressure increased from 3.7 to 18mmHg. Further increases in surface pressure had caused a decrease in perfusion, until zero perfusion was reached for pressures above 55mmHg. It was noted that heating had increased skin perfusion for surface pressures from 3.7 to 18mmHg, but not for pressures greater than 18mmHg. After the release of surface pressure, the reactive hyperaemia peak of perfusion increased with heating. In the third protocol, where skin deformation was measured during the application of 3.7 and 18mmHg, heating caused the tissue to become stiffer. It was found that for surface pressures below 18mmHg, increasing skin temperature significantly increased skin perfusion and tissue stiffness. Overall, Patel et al.'s (1999) findings indicate that higher skin temperature lead to increased cutaneous stiffness under loading and, thus, higher susceptibility to deformation-inflicted injury and cutaneous irritation.

Iaizzo et al. (2004) developed a porcine model[6] to facilitate investigations of deformation-inflicted injury. The model was specifically used to study the relationship between applied temperature, applied pressure, and the duration of time in the formation of cutaneous and deep tissue injuries. An apparatus and procedure were created to simultaneously apply twelve metal discs (each with a diameter of 51mm) on the dorsal aspect of the swine, each at an equal pressure of 100mmHg, for a five hour period, while servo-controlling the disc temperatures at either 25, 35, 40 or 45°C. The severity of the resultant tissue injuries correlated with an increase in the applied temperature. No damage was

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[6] The most accurate model for human skin is porcine skin from the perspectives of anatomy and physiology, immunogenicity, cellular composition, and morphology (Lavker, 1991; Vardaxis, 1997). Porcine skin is composed of an epidermis and dermis with characteristics like those of human skin and, thus, is assumed to have similar mechanical properties to human skin (Meyer al., 1978).

observed in the superficial or deep tissues underlying the sites of the 25°C pressure applicators. However, cutaneous tissue damage resulted from the application of a 35°C temperature. The application of higher temperatures caused both cutaneous and sub-dermal tissue damage (the extent of damage being greater at the 45°C sites). There was a high degree of reproducibility of these results among the sites per temperature (n=64) and number of swine investigated (n=16). The results of Iaizzo et al. (2004) concluded that this animal model of temperature-modulated PI has the potential for clinical use in the field of PI prevention in humans.

An inextricable interplay between elevations in skin temperature and skin moisture has been suggested in-vitro by key studies (Egawa et al., 2002; Falloon et al., 2018; Lachenbruch, 2005; Wu, van Osdol & Daukardt, 2016). The results of these studies suggest that the interaction between the skin microclimate parameters can directly precipitate soft tissue damage through weakening of the epidermis. In other words, soft issue can become susceptible to ischemic damage when exposed to concurrent heat and moisture accumulation between the skin and support surface. To illustrate, skin warming increases vasodilation which, in turn, influences trans-epidermal water loss[7] (Akdeniz et al., 2018; Ngo & Maibach, 2010) and increases perspiration by virtue of vasodilation-induced permeability (Di Pasquale et al., 2003). As previously outlined, perspiration constitutes a source of moisture and, if present for prolonged periods of time at the skin surface, can exacerbate the processes which give rise to soft tissue breakdown (Gefen, 2011; Kottner et al., 2018). Over time, sustained exposure of the skin to moisture can lead to the over-saturation and possible maceration of the epidermis (Gray et al., 2011). As stated by Schnelle et al. (1997), maceration begins with the softening and disruption of the mechanical properties of the stratum corneum, ultimately de-establishing epidermal cells and weakening linkages between dermal collagen fibres. Such degenerative processes reduce the skin's integrity, resistance and resilience to pressure, shear and friction (Hopkins, 2004; McCreath et al., 2006; Schultz, 2005), heightening the skin's risk of external injury. Studies have reported the presence of skin maceration to be linked to the formation of epidermal wounds, including Stage I PIs (Cameron, 2004; Cutting, 1999; Gray et al., 2011; Palenske & Morhenn, 1999).

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[7] Transepidermal water loss (TEWL) is the amount of water that passively evaporates through skin to the external environment due to water vapor pressure gradient on both sides of the skin barrier, and is used to characterise skin barrier function (Ngo & Maibach, 2010). The average rate of TEWL in humans is about 300–400 mL/day; however, it can be affected by environmental and intrinsic factors. In high humidity, the amount of water loss will decrease due to the drop in the water vapor pressure gradient.

As such, there is an extensive body of literature to suggest that an altered skin microclimate may mediate soft tissue damage in animal subjects and in in-vitro models. However, direct clinical evidence from human studies supporting this association is sparse. The extent of the contribution of the microclimate as a factor in PI development and its interaction with sustained tissue loading deformations, in-vivo, has yet to be quantified (Dinsdale, 1974; Clark, Romanelli & Reger, 2010).

3.6 Skin Microclimate Management in Nursing Care

Alterations in skin microclimate conditions can be particularly damaging in the context of 'trapped' moisture and heat accumulated at the skin-support surface interface (Dealey et al., 2015; Denzinger, 2020; Lachenbruch, 2005), often as a result of poor or limited airflow. Sustained elevations in moisture and heat between the skin and support surface are of particular significance and frequently problematic in incontinent patients with limited ability to self-reposition (Mayrovitz & Sims, 2001). If not managed effectively, over-hydration of the stratum corneum may occur, precipitating epidermal softening and maceration. Ultimately, these events result in gradual loss of mechanical strength of the skin barrier, considerably raising its susceptibility to ischemic injury (Agrawal & Chauhan, 2012; Brienza et al., 2015; Harrow & Mayrovitz, 2014; Wu, van Osdol & Dauskardt, 2016).

As such, an early detection of clinically concerning elevations in skin moisture and skin temperature may be used to direct strategies focused on local microclimate management. In patients with a predisposition to PI development, the NPUAP (2019) recommends that clinicians employ measures to keep the skin cool and dry, allowing recovery periods between phases of skin occlusion. Specifically, the *Prevention and Treatment of Pressure Ulcers Clinical Practice Guidelines* (EPUAP-NPUAP-PPPIA, 2019) for nursing care advocate to "protect the skin from excessive moisture with a barrier product in order to reduce the risk of pressure damage" (p. 17). Studies indicate that 'skin cooling' at the bony prominences appears to be a protective factor against PI development (Kottner et al., 2015; Lachenbruch, 2005; Lee et al., 2014; Patel et al., 1999; Wong, 2014). One way to enable skin cooling is to implement regular patient turning schedules in an attempt to expose the skin to the ambient environment, maximining airflow and promoting heat convection at the skin surface (Webb et al., 2015; Werner et al., 1992). Patel et al. (1999) tested and established a protective effect of cooling the skin from 36°C to 28°C, which was found to be equivalent to reducing interface pressure from 56 to 40mmHg (i.e., approximately 2mmHg pressure reduction per 1°C reduction).

Overall, there is evidence to suggest that disruptions in the skin microclimate may be linked to PI development in vulnerable skin. However, further research is required to establish the clinical efficacy of using microclimate variables of skin moisture and skin temperature as clinical indictors of impending PI. At present, the thresholds for skin moisture and skin temperature over the bony prominences, that would create conditions conducive to skin damage, are unknown (Kottner et al., 2018). As such, there is a need for skin microclimate damage thresholds to be explicitly defined within the literature. Transferring this knowledge into the clinical setting could guide informed clinical decision-making and enable the instigation of actions intended to prevent undesirable microclimate effects at the skin-support surface interface. To date, there remains a paucity of the in-vivo literature exploring the quantitative evaluation of the skin microclimate from which damage thresholds could be established.

3.7 Conclusion

The skin microclimate refers to the interaction of the variables of skin moisture and/or humidity, and skin temperature adjacent to the skin. Stratum corneum hydration has also been identified as a relevant dimension in the concept of the skin microclimate. Many studies based on evidence from animal subjects and in-vitro models, as well as limited human-based studies, have postulated the skin microclimate as a theoretical indirect contributor to PI development. According to the literature reviewed in this chapter, there is some evidence to suggest that sub-optimal microclimate conditions can affect the mechanical boundary conditions of the skin (pressure, shear and friction) and accelerate the progression of deformation-inflicted skin damage. As such, it can be argued that the maintenance of a favourable skin microclimate may be a key modifier of the skin's ability to withstand prolonged stresses arising from pressure, shear and friction.

The characteristics of an optimal microclimate are still a matter of debate and ongoing research. However, there is evidence to suggest that with an elevation in skin temperature and skin moisture at the skin-support surface interface, the skin becomes more vulnerable to mechanical loading. It has been documented within the literature that prolonged elevations in skin temperature have been postulated to influence the functional and mechanical properties of the epidermal barrier. Similarly, sustained elevations in skin moisture can lead to softening of the epidermal barrier and, thus, diminished mechanical and tensile strength of the stratum corneum. In PI risk assessment context, the clinician's assessment of the skin microclimate tends to be largely arbitrary and subjective.

3.8 Chapter Summary

This chapter has introduced the concept of the skin microclimate and has reflected on the lack of in-vivo evidence exploring the proposed association between skin microclimate and PI development. The chapter has detailed the parameters of skin moisture/stratum corneum hydration and skin temperature involved in the current understanding of the skin microclimate, and has provided a discussion on the skin microclimate and its theorised contribution to the processes which give rise to PI development. The chapter concluded by reviewing the role of the skin microclimate in the maintenance of skin integrity. To date, there continues to be limited in-vivo research exploring skin changes under different microclimate conditions. The next chapter provides a review of contemporary clinical methods employed in the appraisal of the skin microclimate, and considers innovative methods that may enhance current approaches to skin microclimate appraisal.

CHAPTER 4

Skin Appraisal in Pressure Injury Risk Assessment

4.1 Chapter Overview

Pivotal to the prevention of PI is the clinician's timely identification of risk factors that increase an individual's predisposition to PI development. This early awareness is critical to enabling the instigation of risk-mitigating actions, and begins with a thorough appraisal of the skin overlying the bony prominences of the body. To date, an appraisal of the skin remains a focal point of PI risk assessment. This chapter presents an overview of skin appraisal within a PI risk assessment context, and considers technology that may enhance contemporary appraisal methods. The chapter begins by outlining the general purpose of skin appraisal as part of PI risk screening, and specifically considers assessable parameters of skin colour (melanin and erythema), skin moisture and skin temperature. The chapter then presents a review of the literature pertaining to skin sensing technology as a quantitative method of measuring the parameters of skin colour, skin moisture and skin temperature. The chapter concludes with an articulation of the research gaps, and presents the aims, questions and objectives of this research.

4.2 Skin Appraisal in Pressure Injury Risk Assessment

Several risk factors known to be associated with PI development can be reflected by cutaneous manifestations at the skin surface, typically overlying the bony prominences of the body. Thus, a comprehensive appraisal of the skin considered the first and fundamental step to PI prevention (EPUAP-NPUAP-PPPIA, 2019). The *Prevention and Treatment of Pressure Ulcers Clinical Practice Guidelines* (EPUAP-NPUAP-PPPIA, 2019) together with the *National Institute for Health and Care Excellence (NICE) Guidelines* (2020) recommend that nurses regularly inspect the skin surface of pressure-prone areas of the body of individuals with an existing predisposition to PI. In immobile or bedbound patients, this assessment routinely takes place during each off-loading interval, with the aim of identifying cutaneous signs of existing or imminent superficial skin damage.

To date, visual skin assessment (VSA) comprises most PI prevention initiatives, and is considered a key nursing responsibility (Dealey et al., 2015; Falloon et al., 2018; EPUAP-NPUAP-PPPIA, 2019). VSA is specifically directed at the skin overlying the weight-bearing regions of the body where interface pressure is exerted with the most magnitude (Edsberg et

al., 2014; Keller et al., 2002). These regions are determined based on patient's position. In bedbound patients, the supine, semi-recumbent and lateral positions are the three basic patient positions (Bhattacharya & Mishra, 2015). In the supine and semi-recumbent positions, examples of weight-bearing regions include the sacrum, coccyx, elbows and calcanei. In the lateral position, the most weight-bearing regions include the ischial tuberosities, trochanters, lateral and medial malleoli, and lateral aspect of the ear (Mulligan et al., 2011; NPUAP, 2019).

Traditionally, clinicians have been taught to appraise the skin overlying the bony prominences for changes in colour, moisture and skin temperature (Balzer et al., 2014; Coleman et al., 2013; Falloon et al., 2018; Keller et al., 2002; Rycroft-Malone & McInes, 2000). The appearance of erythematous skin discolouration at a given bony prominence has been long-regarded as a cardinal indicator of tissue distress associated with the initial stages of possible pressure-induced skin damage. As such, capturing early changes in skin colour is critical in initating strategies aimed at preventing the evolution of more serious stages of skin injury (NPUAP, 2019). Also of pivotal importance is the detection of skin moisture and changes in the temperature of the skin overlying the bony prominences (NPUAP, 2019). Sustained elevations in skin moisture and skin temperature—particularly if occurring at the skin-support surface interface where the environment tends to be occlusive—have been reported to affect the integrity of the epidermal barrier, rendering the skin susceptible to the mechanical forces of pressure, shear and friction (Dealey et al., 2020; McElhinny & Hooper, 2008; Stechmiller et al., 2008). This chapter will now detail the parameters of skin colour (Section 4.2.1), skin moisture (Section 4.2.2) and skin temperature (Section 4.2.3), together with the clinical methods of their appraisal.

4.2.1 Visual Appraisal of Skin Colour

The early identification of skin colour changes at pressure-prone areas of the body is considered the cornerstone to PI risk prevention (NPUAP, 2019; Vanderwee et al., 2007). Skin colour is determined, in part, by levels of haemoglobin; an oxygen-transporter contained within red blood cells (Diffey & Farr, 1991) and melanin pigment (Cichorek & Wachulska, 2013). Melanin is a broad term used to describe a group of naturally-occurring pigments found in most organisms. In humans, melanin exists as three forms: eumelanin, pheomelanin, and neuromelanin (D'Alba & Shawkey, 2019). Eumelanin and pheomelanin are produced in various amounts in the basal layer of the epidermis within melanocytes (Figure 4.1). The melanocytes, after cell division, separate and migrate along the basement

membrane where they extend their dendrites and establish multiple contacts with keratinocytes (Haass & Herlyn, 2019).

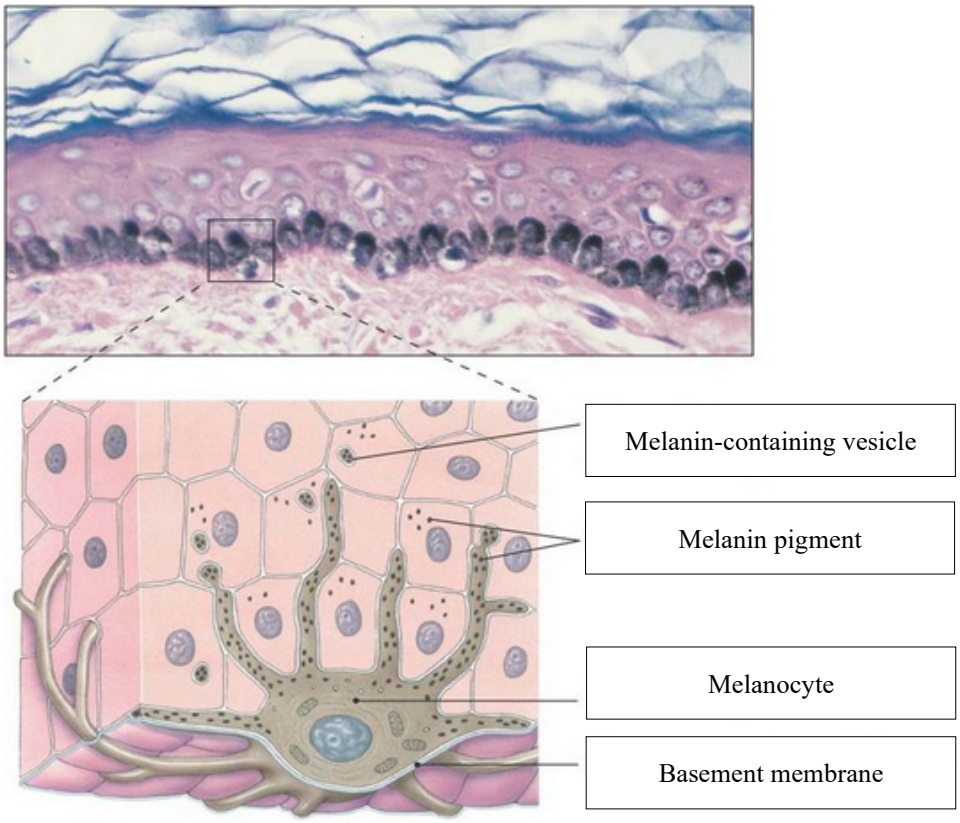

Figure 4.1 Melanin pigments scattered throughout the stratum basale of the epidermis. Reprinted from "The Integumentary System," by Austin Community College, 2019, Austin Community College (https://www.austincc.edu/apreview/PhysText/Integument.html). Copyright 2019 by Austin Community College.

Although humans generally possess a similar concentration of melanocytes in their skin, the melanocytes in some individuals and ethnic groups produce variable amounts of melanin; higher concentrations of melanin are found in individuals with darker skin tones (Cichorek & Wachulska, 2013; Diffey & Far, 1991). Thus, the overall melanin density correlates with the darkness of skin. These differences in pigmentation result from both the relative ratio of eumelanin (brown to black) to pheomelanin (yellow to red), and the number of melanosomes within melanocytes. Lightly-pigmented skin tends to contain melanocytes with clusters of two to three melanosomes, whereas deeply-pigmented skin tends to contain individual melanosomes which can melanise neighbouring keratinocytes more readily (Haass & Herlyn, 2019). Melanin—although not routinely used a prognostic factor for PI—has been shown to contribute to the maintenance of skin barrier function (Baumgarten et al., 2004;

Maranduca et al., 2019; Reed, Ghadially & Elias, 1995). There is some clinical evidence to suggest that the epidermal barrier is stronger and more rapidly repaired following trauma in deeply-pigmented skin (Maranduca et al., 2019; Reed, Ghadially & Elias, 1995).

When caring for individuals at risk of PI development, clinicians have traditionally been taught to observe the colour of the skin overlying the bony prominences for a reddened surface discolouration. This reddened skin surface discolouration is clinically termed 'erythema', and may represent either a normal reaction to pressure (reactive hyperaemia) or an abnormal one (non-blanching hyperaemia) (Chacon et al., 2010; Shi et al., 2020; Vanderwee et al., 2007). The parameter of skin erythema and its methods of appraisal in the clinical setting are detailed below.

4.2.1.1 Erythema Appraisal

Within the medical and nursing literature, there is a large body of evidence (Arnold, 2003; Chacon, 2010; Kosiak, 1961; Liao, Burns & Yan, 2013; Lyder et al., 2012; Sowa et al., 2002; Vanderwee et al., 2007) which denotes erythematous skin discolouration as the most important cutaneous sign of localised inflammation associated with possible ischemic events. The occurrence of erythema is regarded as a manifestation of hyperaemia-induced vasodilation of cutaneous capillaries. As described previously in Chapter 2, reactive hyperaemia is described as the initial physiological response of the skin to pressure-induced ischemia (arterial occlusion) of a short duration. Reactive hyperaemia is characterised by a temporary increase in blood flow to the upper dermis immediately on release of pressure (Liao, Burns & Yan, 2013). The duration of reactive hyperaemia has been reported to vary, and it may be visible at the skin surface only for a few minutes or up to 45 minutes after pressure release (Herrman et al., 1999). Studies have reported that the degree and duration of reactive hyperaemia is proportional to the period of arterial occlusion; that is, the longer the period of arterial occlusion, the greater the vasodilatation (Liao, Burns & Yan, 2013).

Reactive hyperaemia can be distinguished from Stage I PIs in two ways. First, reactive hyperaemia resolves itself within one-half to three-quarters of the time pressure was applied. Second, the erythematous skin area will blanch when pressure is applied (Jan et al., 2013; Yapp et al., 2017). Thus, in clinical practice, Stage I PIs are routinely confirmed with a blanch response test. This is a simple procedure which involves applying brief pressure to the erythematous area sufficient to cause cutaneous capillary closure (Sowa et al., 2002). Capillary closure is achieved at around 30mmHg in humans (Landis, 1930), which is the

equivalent of a light fingertip touch. At this pressure, blood is forced out of the capillary bed. In healthy tissue, this decrease in local blood volume will result in 'blanching' (pallor) of the skin underneath the area where fingertip pressure was applied. Upon pressure release, blood quickly refills the capillary bed, followed by a prompt reappearance of the erythema (Sprigle et al., 2003). In other words, an erythematous site is defined as blanching if it visibly transitions from skin redness to pallor to skin redness during brief application and release of pressure. This return to normal colour should take no longer than twice the length of time that the fingertip pressure was applied (NPUAP, 2019).

A blanch response is considered to be a characteristic of a healthy dermal capillary network and underlying micro-circulation (Holloway & Watkins, 1977; Sowa et al., 2002), and is usually detected by the clinician upon patient repositioning. In patients with deeply-pigmented skin, blanching may not always be visualised when compared to the surrounding skin due to the overwhelming contribution of melanin to the reflectance of the skin (Sowa et al., 2002). However, the affected area may exhibit non-visual characteristics (independent of skin colour) such as tenderness, firmness, warmth or coolness on palpation as compared to adjacent tissue (NPUAP, 2019). Provided that the erythematous area of the body is off-loaded promptly upon detection, the skin will usually resume its normal colour within 24 hours (Romanelli et al., 2006; Shi et al., 2020; Vanderwee et al., 2007).

In the absence of timely pressure relief, a more significant change in local vasculature termed 'non-blanchable' erythema results (Bates-Jensen et al., 2007; McCreath et al., 2006). Non-blanchable erythema is characterised by a lack of pallor at a localised area after the compressive force is released (Vanderwee et al., 2007), and is conventionally regarded as an early sign of pressure ulceration (NPUAP, 2019). As previously outlined, a lack of visible blanch upon fingertip pressure is a convenient diagnostic test for Stage I PI (Bates-Jensen et al., 2007; Boyko et al., 2018; Chacon et al., 2010; NPUAP, 2019). The occurrence of non-blanching erythema has been correlated with post-ischemic vascular engorgement and plasma leakage from blood vessels into the interstitial tissues (Witkowski & Parish, 1982). In high risk patients, non-blanchable erythema can develop in as little as two hours of sustained pressure-loading (Kosiak, 1961), and is prone to progress into more severe injury if the pressure at the affected region is not relieved within four to six hours (Bliss, 1998). Thus, if managed early, non-blanchable erythema is generally reversible (Edsberg et al., 2014; Gefen at al., 2009). In clinical practice, the detection of erythema overlying a pressure-prone area warrants immediate off-loading of the affected region

(NPUAP, 2019) to promote tissue recovery, and is critical in preventing further tissue deterioration (Cuddigan et al., 2009; Duncan, 2007; NICE, 2020; Yapp et al., 2017).

The descriptor of 'skin discolouration' is featured as a sub-scale component of a universally-recognised PI risk assessment scale, the Waterlow Scale (Waterlow, 1985). The Waterlow Scale specifically includes the category 'Skin Type/Visual Skin Areas' in which descriptors of 'Healthy' and 'Discoloured/Stage I PI' are used to depict the colour of intact skin at pressure-prone areas of the body (Waterlow, 1985). In general, it is recommended that a PI risk assessment scale is completed within eight hours of a patient's admission to a healthcare facility (NPUAP, 2019). The re-assessment frequency is then based on the patient's acuity or when there is a significant deterioration in the patient's medical condition (NICE, 2020; NPUAP, 2019).

As such, the early visual identification of localised erythematous discolouration at the skin surface overlying the bony prominences is a pivotal component of PI risk assessment. The earlier the erythema is identified, the earlier a blanch test can be performed to detect changes in blood re-flow behaviour during the hyperaemic period. While blanching erythema is considered to be a normal physiological response to brief pressure in healthy tissue, non-blanchable erythema is regarded as a clinical manifestation of Stage I PI. In conjunction with an assessment of erythema, it is imperative that clinicians inspect for signs of excessive moisture at the skin-support surface interface of bedbound individuals (Agrawal & Chauhan, 2012; Clark et al., 2011; Romanelli et al., 2006). The parameter of skin moisture and its methods of appraisal within nursing practice are outlined below.

4.2.2 Visual Appraisal of Skin Moisture

As overviewed previously in Chapter 3 of this thesis, skin moisture represents one of the fundamental parameters of skin microclimate (Clark et al., 2011). It is well-established within the medical and nursing literature that the long-term presence of moisture at the skin-support surface interface can contribute to cutaneous breakdown (Ayello & Lyder, 2007; Bates-Jensen, 2007; Clark et al., 2011; Harrow & Mayrovitz, 2014; Romanelli et al., 2006). The continual exposure of the skin to moisture, in any form, gradually induces over-saturation of the stratum corneum and can affect the lower levels of the epidermis. The resultant effect is skin softening and possible maceration, reducing the skin's protective barrier function to frictional forces (Agrawal & Chauhan, 2012; Boyce, Supp & Harriger, 1996; Clark et al., 2010; Palenske & Morhenn, 1999). Thus, in practice, the clinician's

detection of elevated moisture at the skin-support surface interface of bedbound patients is routinely followed by interventions designed to optimise the local skin microclimate (NPAUP, 2019). An example of one such intervention includes the application of a topical barrier product specifically formulated to control excess wetness at the target site (Elias, 2005; Gray et al., 2002).

A number of PI risk assessment scales have been developed to guide the clinician's assessment of skin surface moisture, and have been advocated for use in the clinical setting by the *Prevention and Treatment of Pressure Ulcers Clinical Practice Guidelines* (EPUAP-NPUAP-PPPIA, 2019). Although there are various standardised risk-assessment scales, three common scales include the Braden Scale (Bergstrom & Braden, 1987), Waterlow Scale (Waterlow, 1985) and Norton Scale (Norton, 1987). There tends to be some heterogeneity with regard to the manner in which these scales 'measure' skin moisture. The Braden Scale, for example, considers the 'degree' of moisture (dampness and/or wetness) that the patient's skin is exposed to, with an assessment of moisture based on the frequency of bedlinen changes. The Braden Scale further classifies moisture into four sub-categories: 'Constantly Moist' (i.e., dampness at the skin surface is detected every time the patient is repositioned), 'Very Moist' (i.e., the skin is often but not always moist; linen must be changed at least once a shift), 'Occasionally Moist' (i.e., the skin is occasionally moist, requiring an extra linen change approximately once a day), and 'Rarely Moist' (i.e., the skin is usually dry; linen only requires changing at routine intervals) (Bergstrom & Braden, 1987).

The Waterlow Scale (Waterlow, 1985) includes the category 'Skin Type/Visual Skin Areas', and includes the descriptors 'Dry' and 'Clammy/Diaphoretic' to guide the assessment of skin moisture. Additionally, the Waterlow Scale features a 'Continence' category that comprises four items: 'Complete/Catheterised', 'Urine Incontinence', 'Faecal Incontinence' and 'Doubly', with each item assigned a numerical weighting. In this respect, the Waterlow Scale is similar to the Norton Scale (Norton, 1987), which includes a 'Continence' category comprising four items: 'None', 'Occasional', 'Usually Urinary' and 'Urinary and Faecal', with each item scored based on a numerical scale. The presence of incontinence generally translates to a higher risk of moisture-associated skin damage in susceptible individuals.

4.2.3 Tactile Appraisal of Skin Temperature

As highlighted previously in Chapter 3, skin temperature is regarded as a key local skin microclimate parameter (Clark et al., 2011). The empirical evidence indicates an

association between elevations in skin temperature and a reduction in the tolerance threshold of skin and soft tissue during sustained loading (Baldwin, 2001; Knox, 1999; Gefen, 2011; Kottner et al., 2018; Lechner et al., 2021), with increased skin temperature described a as an important indirect risk factor for the development of PIs in susceptible individuals (Agrawal & Pardasani, 2016; Cox et al., 2020; Gefen, 2011; Kottner et al., 2015; Lachenbruch, 2005; Yusuf et al., 2015). There is evidence to suggest that elevations in skin warmth may act as an important clinical indicator of underlying tissue inflammation preceding imminent PI development (Kokate et al., 1995; Soares et al., 2019) that has been reported to occur from three to ten days before visual signs of skin breakdown. It can, therefore, be argued that skin temperature data has the capacity to provide an early warning sign of underlying pathologic processes before skin damage becomes clinically visible. The NPUAP (2019) recommends that nurses use 'touch' to detect for temperature changes at the skin surface of the bony prominences to determine possible underlying local inflammation.

Overall, PI risk assessment forms the basis of planning, implementing and evaluating PI prevention measures. Pivotal to PI risk assessment is the clinician's appraisal of the patient's skin condition overlying the bony prominences. Three critical parameters that comprise skin appraisal include colour (erythema), skin moisture, and skin temperature. While the presence of erythema has been traditionally regarded as a clinical manifestation of pressure-induced skin damage, elevations in skin moisture and skin temperature have emerged as indirect risk factors for compromised epidermal barrier function and reduced tissue tolerance to mechanical loading. To date, the clinician's timely recognition of these skin parameters is used to inform PI risk-mitigating interventions, with the aim of restoring or preserving skin integrity at the affected area. However, such an assessment is heavily subjective in nature, requiring a large degree of clinical judgement of the clinician. It can, therefore, be argued that the subjectivity associated with skin appraisal is a major limitation of current PI risk assessment processes.

4.3 Limitations of Current Methods of Skin Appraisal

The early detection of initial pressure-induced tissue damage through regular visual skin assessment (VSA) is a strategy to prevent possible progression of ulceration (Okonkwo et al., 2020). To date, VSA continues to be an integral measure used to direct decision-making regarding appropriate PI preventative actions (NPUAP, 2019). Underpinning these actions, however, is a strong reliance on the assessor's diagnostic accuracy of visual appraisal itself, which is deeply subjective and may be limited. Consequently, there is

potential for discrepancy among assessors due to varied interpretations of the skin descriptors used to depict the condition of the skin which, in turn, may yield inconsistent and even erroneous assessment findings (Kottner & Balzer, 2010). Some studies indicate that VSA tends to have poor reliability (Anthony et al., 2010; Pancorbo-Hidalgo et al., 2006). Furthermore, Beeckman et al. (2007) noted that because of the skill dependency of VSA, the correct identification of erythema/Stage I PI was observed to be as low as 60% in a diverse group of 1452 nurses. Thus, the initiation of PI prevention measures may be hindered by the fact that VSA is not always reliable. The next sections consider the limitations associated with the subjective appraisal of the PI-associated parameters of erythema, skin moisture and skin temperature.

4.3.1 Erythema

A key source of VSA inconsistency emerges from differences in how humans perceive cutaneous characteristics such as colour. Although the human eye is sensitive to and capable of distinguishing between a vast range of similar colours (Lahti, Kopola & Harila, 1993), linguistic description is unable to satisfactorily convey distinct colour tones and cannot designate an absolute value for the detected colour difference (Banu, Toacse & Danciu, 2014; Fadzil et al., 2009). In particular, VSA may be poor in precisely delineating the margins between distinct levels of colour which—when considered in a PI risk assessment context—can preclude the accurate classification of red hues as evidenced in erythematous skin. The assessment of red hues can be further compounded by the varying degrees of colour blindness which may exist undeclared among assessors (Cockburn, 2004).

Halfens et al. (2001) identified that transient hues of erythema may be difficult to classify for clinicians who lack the appropriate training and experience. The various classification systems of erythema that exist compound the challenge. For example, erythema can be classified as ‘minimal’, ‘moderate’ or ‘severe’. Furthermore, ‘minimal erythema’ can be further described as ‘pinkness’ or ‘slight redness’ of the skin, while ‘moderate erythema’ can be described as ‘bright red’ in lightly-pigmented skin and ‘purple’ in deeply-pigmented skin. ‘Severe erythema’ can present as ‘dark red to purple’ in lightly-pigmented skin and ‘black to blue-grey’ in deeply-pigmented skin (Halfens et al., 2001). Furthermore, different hues of localised erythema are considered to reflect distinct physiological processes underlying the skin. For example, regions with light to moderate hues of erythema have been reported to indicate red blood cell engorgement of the capillaries, while regions with dark

hues of erythema are indicative of red blood cell leakage following blood vessel disruption (Aster et al., 2009).

It is particularly challenging for clinicians to visually discern non-blanching erythema in deeply-pigmented skin (Bergstrom & Braden, 2002; Kottner & Balzer, 2010; Scanlon, 2004; Vanderwee et al., 2007) which, consequently, can inhibit the early identification of Stage I PIs (Baumgarten et al., 2004; Berardesca & Maibach, 2003; Sprigle et al., 2003). The blanch response may not always be visually apparent in individuals with deeply-pigmented skin due to a high concentration of melanin pigment (Sowa et al., 2002). The difficulties inherent in assessing erythema in deeply-pigmented skin and, thus, the consequent lack of recognition of early pressure-induced skin damage is reflected in the literature. PI incidence rates have been reported to be higher in individuals with deeply-pigmented skin (Amlung, Miller & Bosley 1999; Barczak et al., 1997; Bates-Jensen, 2007; Baumgarten et al., 2004; Fuhrer et al., 1993; Lyder et al., 1999; Rosen et al., 2006). For example, Amlung, Miller, and Bosley (1999), in their prevalence study of hospitalised patients (n=42,817) in 356 acute care facilities, reported that Stage I PIs represented 37% of all PIs; of these, Stage I PIs accounted for 48% of all PIs in individuals with lightly-pigmented skin, and only 20% in individuals with deeply-pigmented skin. This discrepancy corroborates findings of Lyder et al. (1999) and Bennett (1995) who identified challenges of identifying erythema in deeply-pigmented skin. Rosen et al. (2006) examined incidence rates of Stage I-IV PIs in nursing home residents with darker skin tones when compared to nursing home residents with lighter skin tones. Stage I PIs were not detected in any of the residents with darker skin tones, whereas Stage I PIs were detected in 7 of 22 (31.8%) residents with lighter skins tones. One interpretation of these findings is that patients with darker skin tones might not receive adequate PI assessment measures.

Overall, the literature postulates erythema as a subjective indicator of skin status and, as such, is routinely appraised by the clinician as part of routine PI risk assessment. The detection of non-blanching erythema has been traditionally regarded as an indicator of possible early onset ischemic damage. However, the classification of erythema is subjective and arbitrary in nature, which poses a critical challenge to its accurate appraisal.

4.3.2 Skin Moisture

The assessment of skin moisture at the skin-support surface interface is a routine part of PI risk screening. However, the manner in which skin moisture is currently assessed by

the clinician may present a limitation to this assessment. As previously identified, skin moisture assessment is embedded as a standard sub-scale component of many standardised PI risk assessment scales. Yet, there may be a theoretical limitation in regard to the manner in which skin moisture is represented by these scales. The scales tend to rely on a 'broader' representation of skin moisture; that is, the scales guide the clinician to visually inspect for the presence of 'dampness' or 'wetness' at the outer surface of the skin. However, the scales do not consider a 'direct' measure of moisture, as for example, derived from the moisture content within the stratum corneum. Studies report that stratum corneum moisture may be associated with maintaining the mechanical properties of the skin barrier (Berardesca, 1997; Clarys et al., 2021; Fluhr et al., 1999; Warner et al., 1999) and, therefore, may be a critical factor implicated in PI development. However, given that stratum corneum moisture is a biophysical skin property (Clarys et al., 2012), it can only be evaluated by capacitance or conductance-measuring devices (Clarys & Barel, 1995). Consequently, visual methods of skin moisture appraisal, informed by PI risk assessment scales, may often fail to recognise what is happening 'below' the skin surface.

4.3.3 Skin Temperature

Skin temperature has been postulated as relevant dimension of PI development (Gefen et al., 2021). As previously outlined in this chapter, the NPUAP (2019) recommends that clinicians use touch and palpation to detect differences in skin temperature when assessing for PI risk at pressure-prone areas of the body. Although localised areas of elevated skin warmth may be felt merely by the touch of the hand, this technique is highly interpretive and only as sensitive as the hand of the assessor (Pompei, 1996). In other words, skin temperature is relative to the assessor; a cold hand will indicate a warm skin surface and a warm hand will indicate a cooler skin surface. It is, therefore, challenging to accurately measure and interpret variations in relation to skin temperature using touch alone (Mifsud et al., 2021; Murff et al., 1998).

Overall, there is a considerable amount of subjectivity involved in the current processes pertaining to the appraisal of skin condition as part of contemporary PI risk assessment. Thus, the reliability of VSA and tactile assessment may be lacking which, in turn, creates limitations for risk assessment. In view of these limitations, there is an opportunity to augment appraisal accuracy with more robust, objective techniques. There are biophysical skin-analysis instruments with the capacity to generate quantitative measurements of erythema, skin moisture and skin temperature. These instruments could

have the potential to overcome the challenges associated with subjective appraisal methods and, in turn, enable a more swift and enhanced identification of skin condition.

4.4 Biophysical Methods of Skin Appraisal

Portable hand-held biophysical instruments are commercially available to enable specialised, non-invasive measurements of skin properties. These instruments have a range of practical applications in cosmetic and dermatological fields. They are routinely used for product development, monitoring of skin therapies, and efficacy-testing of topical products related to the aging process, solar exposure and dermatological conditions (Enviroderm, 2019). Many of these instruments rely on the metrics of the Mexameter—which enables quantitative measurements of melanin and erythema—and the Corneometer—which measures the bio-capacitance of soft tissues and, thus enables measures of stratum corneum hydration, with readings converted into arbitrary units (AUs) (Courage+Khazaka Electronic GmbH, Köln Germany). Other examples of biophysical instruments include infrared dermal thermometers, which have the capacity to quantitatively appraise skin surface temperature. The next sections overview the Mexameter (Section 4.4.1), the Corneometer (Section 4.4.2), and infrared dermal thermometers (Section 4.4.3) with consideration to their principles of measurement and applications within research and clinical areas of practice.

4.4.1 The Mexameter

The Mexameter (referred to in full as the Mexameter MX 18®) (Courage+Khazaka Electronic GmbH, Köln Germany) relies on spectrometer measurement technique (Enviroderm, 2019) to enable the measurement of erythema (haemoglobin concentration) as well as the density of melanin pigment (Clarys et al., 2000). The Mexameter consists of a photodetector and 16 light-emitting diodes that emits light at three defined wavelengths: 568nm (green), 660nm (red) and 880nm (infrared) (Clarys et al., 2000; Shin et al., 2011). Haemoglobin exhibits high absorption of green light and minimal absorption of red light (Diffey & Farr, 1991), therefore, an erythema index can be calculated by measuring the amount of 568nm and 660nm light that is absorbed and reflected (Clarys et al., 2000). In erythematous skin, vasodilation increases the blood content of the sub-papillary plexus, thus, more green light is absorbed and less reflected (Diffey & Farr, 1991). A melanin index, on the other hand, is established through measurement of the amount of absorbed and reflected red and infrared light – a greater absorption of light at these wavelengths is indicative of increased melanin pigment (Clarys et al., 2000). The intensity of the light absorbed is measured and converted into arbitrary units (AU) ranging from 0 to 999. Although there is

currently no 'gold standard' for human skin colour measurement, studies by van der Wal (2013) indicate that the Mexameter melanin measures are correlated with the Fitzpatrick Skin Phototype (Fitzpatrick, 1988); a tool used for the evaluation of skin type based on visual assessment of skin colour and other parameters. Melanin—although not routinely used a prognostic factor for PI—has been shown to contribute to the maintenance of skin barrier function (Baumgarten et al., 2004; Maranduca et al., 2019; Reed, Ghadially & Elias, 1995).

The Mexameter is commonly employed in cosmetic and dermatological practice in facilitating the diagnoses of hypo- and hyper-pigmentary disorders and vascular disorders such as rosacea, as well as the measurement of melanoma and scar tissue properties (Baquié, & Kasraee, 2014; Shin et al., 2011). The sensitivity, repeatability and reproducibility of the Mexameter have been investigated in both in-vivo and in-vitro studies. The reported inter-individual day-to-day repeatability with the Mexameter indicates reliable measurements with a very low variability (1%) and a precision of ±5% (Clarys et al., 2000). In-vitro study results using standardised colour charts demonstrate that the Mexameter is a discriminative and sensitive instrument with a very low coefficient of variance, ranging from 0.3% to 1.9% (Pierard, 1998). Similarly, in-vivo tests reflect that inter- and intra-repeatability is good, with variability of melanin and erythema indices reported to be 1 to 2% (Diffey & Farr, 1991). An additional study assessed the reliability of the Mexameter by measuring the intraclass correlation coefficient (ICC) with 95% confidence intervals (van Der Wal et al., 2013). In normal skin, the ICC was 0.92 and 0.94 for erythema and melanin, respectively. Given that an ICC of 0.7 is considered a minimum requirement for reliable test results (Pallant, 2011), these figures indicate that the Mexameter is a highly reliable measure of erythema and melanin in normal skin.

Overall, the Mexameter is a commercially-available device with capacity to provide quantitative skin colour data. Studies have investigated the clinimetric properties and clinical feasibility of the Mexameter and have reported it to be both a valid and reliable instrument. Additionally, strong correlations between the Fitzpatrick Skin Phototype Classification (Fitzpatrick, 1988) and the Mexameter melanin measures suggest that the Mexameter may reliably assist in the assessment of human skin colour.

4.4.2 The Corneometer

Since it was first commercially manufactured in 1982, the Corneometer (referred to in full as the Corneometer CM 825®) (Courage+Khazaka Electronic GmbH, Köln Germany)

is recognised as the gold standard for measuring the water content in the stratum corneum (Kajs & Gartstein, 1991). The Corneometer is commonly used in cosmetic and dermatological practice to evaluate and monitor the effects of topical products and therapies in both healthy skin and in pathological skin conditions such as psoriasis (Berardesca, 1997; Lee et al., 2012; Serup, 2001) and atopic dermatitis (Sator, Schmidt & Honigsmann, 2003). Corneometer measurements rely on the skin acting as a dielectric, i.e., material that resists the flow of electrical current (Edwards & Marks, 2005). Dielectrics electrically isolate two metal plates to form a capacitor; a device that stores electric charge. The impedance of the skin to electrical forces is used to calculate surface electrical capacitance, which directly reflects the moisture in the epidermal tissues (Goretsky et al., 1995). Surface electrical capacitance has been used to quantify wound healing in burn patients (Ho, Bello & Grove, 2000) and to examine relationships between skin hydration and chronic wound healing in several small case series studies (Fore-Pfliger, 2004; Goretsky et al., 1995; Harrow & Mayrovitz, 2014; Palenske & Morhenn, 1999).

The Corneometer consists of a measuring probe which, when depressed onto the skin surface and supplied with a current, functions as a capacitor (Conti, Schiavi & Seidanari, 1995). When depressed onto the skin surface, the probe exerts constant pressure using a spring system and measures depths from 10 to 20µm. This measurement depth is very small to exclude the influence of the deeper skin layers. The amount of electrical charge stored by the capacitor is a reflection of stratum corneum hydration. The unit of measurement is expressed by an arbitrary unit (AU) displayed on the device screen. The higher the measured value, the higher the moisture content of the stratum corneum. In other words, a higher level of stratum corneum hydration reflects higher dielectric constants.

The reproducibility of the Corneometer measurements has been shown to be high, with reported values for coefficient of variation ranging from 11 to 14% (Clarys, Barel & Gabard, 1995), suggesting a high degree of agreement between independent assessors (Polit & Beck, 2006). Intra-person repeatability has also been demonstrated to be very good, with a mean coefficient of variation of 7% (Clarys, Barel & Gabard, 1995), indicating low variation in Corneometer measures when taken at the same area of the skin and under the same conditions.

The Corneometer has been compared to similar instruments such as the Skicon 200EX and Nova DPM which measure the parameters conductance and impedance,

respectively, as an indication of skin hydration (Clarys, Barel & Gabard, 1995). Despite differences in measuring principles, the Nova NPM, Skicon 200EX, and Corneometer, are highly correlated; r=0.97 between the Nova NPM and Corneometer, and r=0.89 between the Skicon 200EX and Corneometer (Clarys, Barel & Gabard, 1995). According to these study findings, all three instruments provided valid and coherent information relative to skin hydration levels. In a more recent study by Clarys et al. (2012), in-vivo measurements were conducted on a range of very dry skin (sole of foot and palm) to well-hydrated (forehead, cheek and forearm) skin areas among 20 participants aged 20 to 71 years. Capacitance and conductance measures of skin hydration were performed using the Corneometer and Skicon 200EX. Statistically significant correlations ($p < .01$) emerged between the instruments, with both providing good estimates of skin hydration. Significant strong correlations ($p < .01$) have also been reported between the Corneometer and Nova DPM by Fluhr et al. (1999), in which measurements were performed across the forearm, hand, cheek and upper back among twelve participants aged 25 to 49 years. It was found that both instruments produced results that showed comparable changes in skin hydration over time, and were able to adequately differentiate between areas of highly- and under-hydrated areas of the skin.

As such, it can be argued that there are non-invasive biophysical skin-analysis instruments—the Mexameter and Corneometer—with the capacity to provide valid and reliable skin property data; skin colour and stratum corneum hydration, respectively. Aside from the Mexameter and Corneometer, there are well-established instruments with the capacity to quantitatively appraise skin surface temperature. These instruments are commonly referred to as infrared dermal thermometers, and are detailed in the next section.

4.4.3 Infrared Dermal Thermometers

Specialised infrared technology, also referred to as infrared dermal thermometry, has the ability to provide non-invasive digital measurements of the temperature of the tissue to about a centimetre below the skin surface. Infrared dermal thermometers have applications in both clinical settings, as well as non-clinical settings where they are used to study thermal properties of the tissue (Dallimore et al., 2020; Foto et al., 2007). They are widely applied in physiatry to assist in the diagnosis of conditions such as complex regional pain syndrome, diabetic neuropathy, vascular insufficiency, and vasospastic disorders (Armstrong et al., 2007; Birklein et al., 2000; Harada, 2002; Kelechi et al., 2003; Lavery et al., 2004). Infrared dermal thermometers are also commonly used for monitoring the skin temperature of healing wounds (Sprigle et al., 2001) and diabetic foot ulcers (Lavery et al., 2004), and for

identifying sub-epidermal inflammation associated with post-operative wound infection (Meijer, 1994).

Lavery et al. (2004) sought to evaluate the effectiveness of at-home infrared temperature monitoring as a preventative tool in individuals diagnosed with diabetes mellitus at high risk of lower-extremity ulceration. Eighty-five patients with diabetic neuropathy, foot deformity and/or previous history of ulceration were randomised into a standard therapy group (n=41) or an enhanced therapy group (n=44). Standard therapy consisted of therapeutic footwear, diabetic foot education, and regular foot evaluation by a podiatrist. Enhanced therapy included the addition of a handheld infrared dermal thermometer (TempTouch®; Xilas Medical, San Antonio, TX) to measure the temperature on the sole of the foot in the morning and evening. Study participants were followed over six months. Lavery et al. (2004) reported the elevated foot temperatures (>2°C compared with the opposite foot) were considered to be 'at risk' of ulceration at the site of measurement.

The results of the above study by Lavery et al. (2004) indicated that the enhanced therapy group had significantly fewer diabetic foot complications; enhanced therapy group 2% versus standard therapy group 20% [$p < .01$, 95% CI (1.2–85.3)]. The standard therapy group was 10.3 times as likely to develop a foot complication compared to participants using the infrared dermal thermometer to monitor their foot temperature. Overall, these results suggest that at-home infrared dermal thermometers, when employed as a self-monitoring aid for measuring foot temperatures, may be an effective adjunctive tool to prevent foot complications in individuals at high risk for lower-extremity ulceration.

Overall, the effectiveness of novel infrared dermal thermometers in quantifying skin temperature has been investigated in several studies. The results of these studies indicate that infrared dermal thermometers are able to generate valid and reliable skin temperature data, and could enable detection of sub-epidermal inflammatory processes associated with PI *before* visual signs of damage manifest at the skin surface. Thus, these devices could have potential applications within the clinical setting as a viable method of objectifying skin appraisal in a PI risk assessment context.

As outlined in this chapter, contemporary methods of skin colour and skin microclimate appraisal in PI risk assessment are largely based on the clinician's clinical judgement and interpretation. This has the potential to preclude an accurate skin appraisal

and, consequently, can limit the ability to focus PI prevention strategies appropriately. In view of the widespread use of subjective methods to appraise PI skin parameters, there is a possibility that these parameters may be misinterpreted, underestimated, or even overlooked completely. However, there are commercially available biophysical skin-analysis instruments that have been previously validated (Borzdynski, McGuiness & Miller, 2016; Huimin et al., 2017) in their ability to yield non-invasive quantitative measurements of skin colour (erythema and melanin) and skin microclimate parameters (stratum corneum hydration and skin temperature). These instruments—if applied relative to current PI risk assessment approaches—could overcome the challenges and potential deficiencies attributed to subjective skin appraisal. As such, there is an opportunity to explore biophysical skin-analysis instruments in the quantification of skin parameters associated with PI development.

4.5 Research Gaps

Chapter 2 of this thesis identified the contemporary research related to PI aetiological schema. It was inferred that tissue ischemia, induced by sustained pressure-loading and shear at the weight-bearing bony prominences, is acknowledged as the key factor implicated in PI development. Additionally, repeated frictional forces at the skin surface can considerably accelerate the damage pathway. Chapter 3 presented a review of the literature that investigated the concept of the skin microclimate. It was concluded that there is evidence to suggest that disruption to the local skin microclimate conditions may indirectly contribute to PI risk by impacting skin barrier function.

Chapter 4 presented an overview of skin appraisal in PI risk assessment, with consideration to assessable parameters of skin colour (melanin and erythema), skin moisture and skin temperature. It was identified that erythematous hues over pressure-prone areas represent a cardinal indicator of localised underlying inflammation associated with possible ischemic events. Furthermore, skin moisture/stratum corneum hydration and skin temperature emerged as factors that have been theorised to indirectly influence the mechanical properties of the stratum corneum. Chapter 4 additionally described some biophysical technologies with the capacity to numerically quantify measures of skin colour (erythema and melanin), skin moisture/stratum corneum hydration and skin temperature in intact healthy skin. The incorporation of biophysical technologies within the clinical setting—alongside clinical judgement—could aid in skin appraisal and potentially strengthen current methods of PI risk identification.

This review identified six research (knowledge) gaps and one methodological issue in the literature that are explicitly addressed in the forthcoming chapters of this thesis. In this thesis, research gaps I, II and III and the one methodological issue have been addressed in Studies I and II, while research gaps IV, V and VI have been addressed in Study III. The gaps in the literature are:

I. A lack of descriptive data to profile measurements of erythema, melanin, stratum corneum hydration and skin temperature when assessed at the elbows, heels and sacrum;
II. A lack of research which has tested and reported differences in measurements of erythema, melanin, stratum corneum hydration and skin temperature when assessed at the elbows, heels and sacrum over time;
III. A lack of research which has tested and reported differences in measurements of erythema, melanin, stratum corneum hydration and skin temperature between supine and semi-recumbent positions;
IV. A lack of research which has examined the effect of skin warming on measurements of erythema, melanin, stratum corneum hydration and skin temperature when assessed at the elbows, heels and sacrum; and
V. A lack of research which has examined the effect of skin wetness on measurements of erythema, melanin, stratum corneum hydration and skin temperature when assessed at the elbows, heels and sacrum.

In order to address the above six research gaps, the research team first needed to address the following methodological issue, which became another research question considered by the current research program:

I. Does monetarily shifting pressure to enable skin parameter measurements of erythema, melanin, stratum corneum hydration and skin temperature alter skin parameter scores at pressure-prone areas of the body?

This methodological issue presented a necessary area of investigation which, in turn, would allow the research team to proceed with confidence regarding the use of the original protocol in Study III. Specifically, it was necessary to identify if pressure (being an extrinsic factor) impacted skin parameters in a way that would not make it possible to represent skin changes, over time, accurately.

4.6 Thesis Aims, Questions and Objectives

This thesis is based on three studies involving healthy human participants. Studies I and II (the preliminary descriptive phase of this research) piloted key methodological and logistical aspects to allow the progression of and to accurately address the aims of Study III (the final experimental phase of this research). In other words, Studies I and II provided a foundation for a larger-scale investigation concerning evaluation of skin manifestations in response to local pressure and skin microclimate in healthy adults.

4.6.1 Studies I and II

The aim of Studies I and II was to assess for skin parameter changes following brief consecutive off-loading—over one hour—at pressure-prone areas of the body (the sacrum, elbows and heels) amongst healthy adults lying supine and then semi-recumbent. More broadly, Studies I and II sought to inform the feasibility protocol and procedural aspects of the proceeding experimental study (Study III).

Studies I and II sought to answer the following research questions:

1. How do erythema, melanin, stratum corneum hydration and skin temperature measures—when assessed at the skin overlying bony prominences of the body—change over time in response to local pressure?[8]
2. Is there agreement between uninterrupted and interrupted methods when assessing erythema, melanin, stratum corneum hydration and skin temperature over time? That is, are the results from skin parameter measurement altered or relatively unchanged during brief off-loading of bony prominences?

In order to achieve the aim of Studies I and II, the following objectives were adopted:

1. To establish a descriptive library of skin parameter scores—as measured over time and across pressure-prone areas—during intermittent pressure off-loading;
2. To compare differences in skin parameter scores between timepoints (baseline to final) as measured at pressure-prone areas amongst participants;
3. To compare differences in skin parameter scores as measured at pressure-prone areas in the supine and semi-recumbent position; and
4. To examine the agreement between two methods (uninterrupted and interrupted) when attending to skin parameter measurements at pressure-prone areas, over time, amongst participants.

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[8] Pressure generated from internal bodyweight forces.

Pending confirmation of whether minimal variation would emerge between skin parameter scores gathered in the two methods (uninterrupted and interrupted) of Studies I and II, the feasibility and protocol for Study III could be considered and informed.

As such, Studies I and II piloted methodological aspects to allow the progression of and to accurately address the aims and objectives of a larger-scale investigation (Study III). Data gathered in Studies I and II indicated that minor shifts in positioning did not alter skin parameter scores at pressure-prone areas of the body. Thus, these results confirmed capacity to progress with Study III with some minor variations to the original study approach.

4.6.2 Study III

The aim of Study III was to examine the skin microclimate effects on clinical manifestations often attributed to PI development in healthy adults. Specifically, Study III sought to assess for differences in erythema, stratum corneum hydration and skin temperature on exposure to heat and moisture at pressure-prone areas in healthy adults lying in the semi-recumbent position, over time (one hour), with periodic off-loading to record the data.

Study III sought to answer the following research question:

1. How do erythema, stratum corneum hydration, and skin temperature measures—when assessed quantitatively at the skin overlying bony prominences of the body—change over time in response to local pressure and the controlled manipulation of skin microclimate at the skin-support surface interface?

In order to achieve the aim of Study III, the following objectives were adopted:

1. Establish a descriptive library of skin parameter scores—as measured over time and across pressure-prone areas—during intermittent pressure off-loading in the control, heat intervention, and moisture intervention;
2. Assess for differences in skin parameter scores, over time, and across pressure-prone areas between the control and heat intervention; and
3. Assess for differences in skin parameter scores, over time, and across pressure-prone areas between the control and moisture intervention.

A series of research hypotheses were further generated pertaining to Study III of this research and are presented in Section 6.5 of Chapter 6.

4.7 Conclusion

A comprehensive visual appraisal of intact skin at pressure-prone areas is a focal point of PI prevention. When caring for individuals who are at risk of PI development, it is essential that skin appraisal is carried out regularly to identify any change in skin condition which could allude to pressure area damage. One such indicator is non-blanching erythema, which may indicate both a normal reaction to pressure (reactive hyperaemia) or an abnormal one (non-blanching hyperaemia). In conjunction with erythema assessment, the clinician's evaluation of skin moisture and changes in skin temperature over pressure-prone areas is pivotal to estimate the vulnerability of the skin and soft tissue to pressure-induced skin damage. The parameters of skin moisture and skin temperature are encapsulated by the notion of skin microclimate, and may have an influential role in PI formation.

To date, the clinician's visual skin appraisal (VSA) of skin colour and the skin microclimate at pressure-prone areas of the body forms an integral component of PI risk assessment and is used to inform risk-mitigating interventions. Although VSA is rapid and convenient, it lacks precision and may underestimate, overexaggerate, or even overlook indicators of skin and tissue injury. Yet, there are commercially available biophysical skin-analysis instruments capable of quantitative description of skin condition. Many of these instruments are able to non-invasively quantify skin colour (erythema and melanin) and microclimate parameters (moisture/stratum corneum hydration and skin temperature) in human skin. However, clinically meaningful ranges for biophysical measures of skin colour and skin microclimate properties over intact healthy skin at pressure-prone areas of the body remain unknown.

Although there are few published studies that report clinical thresholds for erythema, melanin, stratum corneum hydration and skin temperature, these studies are largely dominated by cosmetic research and, as such, may not be directly transferrable to a PI risk assessment context. Furthermore, there is a lack of the literature that has sought to explore the manner in which these skin parameters change in response to the compounding effects of pressure at the bony prominences under different skin microclimate conditions. This paucity in the literature creates a knowledge gap and provides the impetus for this research. The findings generated from this research are anticipated to contribute to the existing body of knowledge concerning the theoretical contribution of the skin microclimate to superficial skin changes. Ultimately this knowledge could help to inform current PI guidelines and frameworks pertaining to optimisation of the local skin microclimate.

4.8 Chapter Summary

This chapter has presented a review of the current methods employed in the clinical appraisal of the skin as part of PI risk assessment. The evidence described has reinforced that the contemporary approaches used to appraise PI-associated skin parameters are largely subjective and may be unreliable. The chapter also identified that non-invasive technology is available that can generate biophysical measures of skin colour, stratum corneum hydration, and skin temperature. The collection of these data could supplement current PI risk assessment processes and, as such, inform targeted action regarding PI prevention efforts. Yet, to date, there is a paucity of the literature that has investigated changes in biophysical skin properties in response to pressure-loading.

The chapter has concluded with an articulation of the research gaps to be addressed by the current research. The chapter has also re-stated the aims, questions and objectives specific to Studies I and II (the preliminary phase of this research) and Study III (the final research phase). The next chapter reports the method and results of Studies I and II. In this thesis, Studies I and II are presented as a publication, as published by the *Journal of Tissue Viability* and titled "Brief intermittent pressure off-loading on skin microclimate in healthy adults – A descriptive-correlational pilot study" (Borzdynski, Miller, Vicendese & McGuiness, 2021).

CHAPTER 5

The Effect of Brief Intermittent Pressure Off-loading on Skin Microclimate In Healthy Adults

5.1 Chapter Introduction

This chapter reports the background, method and results of two studies (in this research, referred to as Studies I and II) examining skin changes in response to intermittent pressure-loading over a one hour period of time. A pilot study was undertaken to compare a series of skin parameter measurements gathered at pressure-prone areas of 41 healthy active adults lying supine and then semi-recumbent. The objective of this pilot study was to assess for skin parameter differences between two methods (interrupted and uninterrupted) with respect to timepoints (baseline, interim, and final). This, in turn, would enable consideration if momentarily shifting pressure to enable measurements (a method which, at that time, was being considered for other research initiatives) altered skin parameter readings.

In Study I, repeated measures were taken whilst participants lay supine, while in Study II, repeated measures were taken whilst participants lay semi-recumbent. In the interrupted method, repeated measures were taken at baseline and immediately after pressure off-loading every 10 minutes, with brief loading repeated six times of the course of hour. In the uninterrupted method, repeated measures were taken at baseline and immediately after pressure off-loading following one hour of sustained loading. It should be noted that in Studies I and II, the main concern was not change over time (as reflected by intermittent timepoints of data collection), rather—by virtue of the definition for the uninterrupted method—only the first and final measurements needed to be compared.

These two pilot studies are presented as a publication in this chapter, as published by the *Journal of Tissue Viability* and titled "Brief intermittent pressure off-loading on skin microclimate in healthy adults – A descriptive-correlational pilot study" (Borzdynski, Miller, Vicendese & McGuiness, 2021). This publication represented an important milestone in the progression of the understanding of cutaneous changes measured immediately after pressure off-loading every 10 minutes, over the course of one hour in supine and semi-recumbent healthy adult participants. The results and conclusions emerging from this preliminary

research phase were subsequently used to guide the experimental research design of the related larger-scale study (Study III).

To supplement the readers' knowledge of the research process and results further to what could be included in the published manuscript, the following appendices relevant to Studies I and II are provided:

Appendix A	Ethics Approval (Studies I and II)
Appendix C	Participant Information Statement and Consent Form (Studies I and II)
Appendix E	Telephone Screening Checklist (Studies I and II)
Appendix G	Norton Risk Assessment Scale (NRAS)
Appendix H	Fitzpatrick Phototype Classification
Appendix I	Participant Demographic and Health Data Form (Studies I and II)
Appendix K	Participant Data Collection Form for Skin Parameter Results (Study I)
Appendix L	Participant Data Collection Form for Skin Parameter Results (Study II)

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Brief intermittent pressure off-loading on skin microclimate in healthy adults – A descriptive-correlational pilot study

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ABSTRACT

Aim: This study examined microclimate changes to the skin as a result of pressure over a 1 h period. The results were compared to skin parameter results following brief consecutive off-loading of pressure-prone areas.
Design: A descriptive-correlational pilot study was undertaken.
Method: A convenience sample of 41 healthy adults aged 18–60 years was recruited. Participants engaged in four 1 h data collection sessions. The sessions were conducted in both semi-recumbent and supine positions. Measures of erythema, melanin, stratum corneum hydration, and skin temperature were taken at pressure-prone areas at baseline and after 1 h in an uninterrupted method (continuous pressure-loading) and every 10 min in an interrupted method (brief off-loading). The Corneometer and Mexameter (Courage + Khazaka Electronics GMbH, 2013) and Exergen DermaTemp DT-1001 RS Infrared Thermographic Scanner (Exergen Corporation, 2008) provided a digital appraisal of skin parameters. Intraclass correlation coefficients (ICC) were calculated to indicate test-retest reliability and absolute agreement of results between the two methods.
Results: Strong agreement between the interrupted and uninterrupted method was observed with ICCs ranging from 0.72 to 0.99 (supine) and 0.62–0.99 (semi-recumbent). Endpoint measures tended to be higher compared to baseline measures for all skin parameters. Differences in skin parameters results by anatomical location were evident particularly for erythema and stratum corneum hydration; the elbows and heels yielded lower scores compared to the sacrum. Erythema had the most variation across methods. The supine and semi-recumbent positions had negligible effect on measured skin parameters.
Conclusions: Minimal variation between skin parameter results indicates that brief off-loading in the interrupted method did not significantly change the outcomes; minor shifts in positioning do not alter changes to the skin from pressure. Skin parameters varied by anatomical location and changed over a 1 h period of pressure-loading.
Relevance to clinical practice: Biophysical techniques may be able to assist accurate assessment of skin microclimate and skin colour. As brief off-loading (interruptions) to enable skin parameter measurement does not alter skin readings, researchers can proceed with some confidence regarding the use of this protocol in future studies assessing skin parameters. This study data provides a library of cutaneous changes at pressure-prone areas of healthy adults and is expected to inform innovative approaches to pressure injury risk assessment.

1. Introduction

Pressure injuries (PIs) (also known as bedsores, pressure sores, or pressure ulcers) are injuries to the skin and the underlying soft tissue [1] caused by sustained mechanical loading and deformation of soft tissues between internal bony prominences and the external support surface [2–5]. If the intensity and duration of deformation exceeds the capacity and resistance of the affected tissues, tissue ischemia will occur and necrotic regions may develop [6,7]. PIs are one of the most frequently-occurring yet potentially preventable adverse events in hospital [8]. On average, PI prevalence rates in hospitals range from 0.3% to 46% globally [9]. They are costly and challenging to heal, and result in poor patient outcomes [10]. In Australia, PIs are one of the top five most expensive adverse events [11], costing $1.8 billion per annum [12]. Epidemiological data indicate that PIs occur in all settings and age groups [6,13], however, they are a particularly common affliction in the

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bed-bound geriatric population [14–16]. In supine and semi-recumbent lying positions, the sacrum, elbows and heels are most susceptible to PI development [17,18] and are well known as PI predilection areas [2].

1.1. Factors implicated in pressure injury aetiology

1.1.1. Skin microclimate

There are key risk factors that have been proposed in the conceptual schema of PI aetiology [19]. While interface pressure is generally considered to be the primary direct risk factor for PI development [20–23], skin microclimate can be considered an indirect contributor [24–27]. The concept of skin microclimate typically reflects the combination of temperature, moisture and/or humidity, and air movement at the skin support-surface interface [24] A review of the literature [28–31] indicates that a suboptimal microclimate frequently precipitates early forms of PI onset. According to Gefen's mathematical model [32], altered microclimate conditions decrease cutaneous resistance which, in turn, leads to superficial skin changes at the affected area. Skin temperature, for instance, has been correlated with tissue injury in an animal study [33] and in a human study, it has been shown to increase by 1.2 °C in 24–96 h before PIs develop [34]. Specifically, a temperature increase in the microclimate causes a temperature increase of the stratum corneum and underlying cutaneous layers [35]. The water permeability of the stratum corneum is then raised [36], leading to an increase of transepidermal water loss [37,38] which aggravates possible moisture accumulation on the skin surface and in the stratum corneum. At the same time, the cohesive strength of the stratum corneum is reduced [39]. Studies [26,40–44] indicate that increased stratum corneum hydration results in changes in the mechanical properties of skin, influencing the resilience of the epidermal layer and thereby rendering the skin susceptible to damage due to shear and friction. The presence of prolonged moisture—in the form of perspiration, urine or wound exudate, for example—at the skin support-surface interface further weakens and softens the stratum corneum [26,43], making superficial damage through abrasion more likely (less friction is required to abrade the skin when it is damp) [43]. The establishment of an optimal microclimate of the skin is, therefore, a critical factor in deterring PI formation. Although suggestions about an optimal microclimate have been made [45,46], the exact range of an acceptable microclimate—including possible upper and lower thresholds— remains unknown.

1.1.2. Skin colour

Skin colour is determined, in part, by levels of haemoglobin; an oxygen-transporter contained within red blood cells [47], and melanin; a skin pigment produced by melanocytes [48]. The assessment of skin colour at pressure-prone areas can provide a direct reflection of several underlying physiological processes reflective of pressure-induced damage [49]. Specifically, erythema/skin redness signals imminent tissue damage. The presence of erythema is correlated with increased capillary and blood vessel dilation in the papillary and reticular dermis, and the subsequent increase in superficial plexus haemoglobin concentration. These protective mechanisms are designed to increase oxygenated blood delivery to the affected tissues [49,50]. If the pressure at the local tissue is not relieved, a domino effect is triggered, and extensive disruption to the local micro-circulation begins to occur. In this state, plasma has leaked from blood vessels into the interstitial tissues, and release of the iron pigment from haemoglobin leads to a persistent red staining of the skin [49].

1.2. Appraisal of skin microclimate and skin colour at pressure-prone areas

In clinical practice, prevention of PI requires accurate assessment of skin status and monitoring of skin changes associated with PI development [2,51]. Skin moisture/wetness [52,53] and skin colour [49] are both critical and informative descriptors of skin status and, as such, are included as subscales in many standardised PI risk assessment scales [53–58]. The assessment of these skin parameters, however, is heavily guided by the assessor's visual assessment and interpretation of skin status and—from a measurement perspective—may be unreliable. In general, empirical evidence supporting the validity of PI risk assessment scale scores is weak, with obtained scores containing varying amounts of measurement error [59–61]. For instance, the acquisition and communication of colour information visually is limited by descriptive language [62,63]. Although the human eye is capable of distinguishing between a vast range of similar colours [64], linguistic description is unable to satisfactorily convey distinct colour tones. Even though the clinician eye is capable of detecting the difference between skin colours, it cannot designate an absolute value for the detected colour difference. To illustrate, visual assessment is poor in precisely delineating the margins between distinct erythematous levels. Another point of critique regarding visual skin assessment is varying degrees of colour blindness which may exist undeclared among clinicians [65]. These limitations act as motivation for the application of biophysical techniques to assist accurate assessment of skin status. There are a range of non-invasive biophysical instruments—presently used within clinical dermatology for diagnostic and therapeutic purposes—which can numerically quantify stratum corneum hydration and skin colour [66]. The application of biophysical instruments in capturing these parameters in a PI context has the potential to enhance assessment accuracy by eliminating measurement subjectivity and, thus, addressing the aforementioned disadvantages of visual assessment.

A pilot study was undertaken to compare a series of skin parameter measurements taken during a period of sustained pressure-loading and intermittent pressure-relief of pressure-prone areas in healthy active adults lying supine and then semi-recumbent. To allow a comparison, repeated measures were taken across an uninterrupted method (immobilisation time 1 h) and an interrupted method (1 h of 10-min loading intervals, with a 20 s recovery in between). The objective of this study was to assess for skin parameter differences between methods (interrupted and uninterrupted) and timepoints (baseline and final) to capture their associations with skin parameters. This, in turn, would enable consideration if momentarily shifting pressure to enable measurements (a method being considered for other research initiatives) altered skin parameter readings.

Specifically, the following questions were raised:

1. How do erythema, melanin, stratum corneum hydration, and skin temperature measures when assessed using biophysical techniques at the skin overlying bony prominences change over time?
2. Is there agreement between uninterrupted and interrupted methods when assessing stratum corneum hydration, skin colour, and skin temperature over time? That is, are the results from skin parameter measurement altered or relatively unchanged during brief off-loading of bony prominences?

2. Materials and method

2.1. Ethics

This research was conducted in full concordance with the ethical principles of the 1975 Declaration of Helsinki, and within Australian laws and regulations for Higher Research (National Statement on Ethical Conduct in Human Research, 2018). Institutional review and approval for this study (Ref. #HEC20300) was received from the Human Research Ethics Committee of the affiliated university.

2.2. Design

A descriptive-correlational design pilot study was undertaken between February and July 2019.

2.3. Sample and recruitment

Convenience sampling was used to recruit eligible people from the student population of the affiliated university. A planned sample size target of 30 was sought based on the number of participants needed to evaluate feasibility goals of the study method. To be eligible, the person needed to be a healthy adult with the capacity to provide written informed consent. People were not eligible for the study if they;

- had a diagnosis of cognitive impairment and/or disability that precluded their ability to provide informed consent;
- had tattoos, wounds, or current dermatological condition/s at one or more of the anatomical testing sites; and
- reported current symptoms related to musculoskeletal injury, macrovascular disease or any biomechanical abnormalities that could affect the ability to lay stable for up to 1 h at a time.

Prospective participants contacted the researcher and were provided with the study's information statement. A telephone screening checklist confirmed eligibility and an appointment was booked.

2.4. Setting

This research was conducted within a private nursing laboratory teaching room at a major metropolitan university in Melbourne. The study site remained unchanged throughout data collection. Prior to each appointment, an analogue thermometer and hygrometer were used to gauge the ambient temperature and relative humidity within the laboratory room. Average room temperature was recorded at 23.7 °C (range: 21.0–25.2 °C) while relative humidity was recorded at 56% (range: 52–66%).

2.5. Primary and secondary outcome measures

The primary outcome measures in this study included a series of cutaneous parameters measured repeatedly, over time, at pressure-prone areas of the body. These measures—stratum corneum hydration, erythema, melanin, and skin temperature—and the data collection procedures are described further below. Secondary outcome measures included participants' demographic profile and health data.

2.6. Data collection instruments

2.6.1. SD202 skin diagnostic corneometer

Stratum corneum hydration was measured with a capacitance-based measurement device: the SD202 Corneometer (Courage + Khazaka, Köln, Germany) [66]. The Corneometer uses the high dielectric constant of water for analysing the water-related changes in the electric capacitance of the skin in the outermost skin layers (10–20 μm of the epidermis). Measurement results are given in system-specific arbitrary units and were found to be highly reproducible and precise (±3%) [67–71]. A hydration score of 0–23 indicates very dry skin, 24–45 indicates dry skin, 46–64 indicates an optimal balanced zone, 65–82 indicates a higher level of hydration, and 83–99 implies over-hydration/saturation of the skin [72] (see Box 1).

Box 1
Skin Diagnostic Reading Interpretation Guide

SD202 measure	Range	Reading interpretation
Epidermal Hydration	0–23	very dry
	24–45	dry
	46–64	optimal balanced zone
	65–82	higher level of hydration
	83–98	over-hydrated
Erythema	1–18	no erythema
	19–36	minimal erythema
	37–54	diffused redness
	55–80	high erythema
	80–99	extreme erythema
Melanin	1–8	very fair/pale
	9–13	pale/fair
	14–21	medium/cream
	22–30	moderate brown/olive
	31–37	dark brown
	38–99	deeply-pigmented/dark to black

2.6.2. SD202 skin diagnostic mexameter

Erythema and melanin were measured using the SD202 Mexameter (Courage + Khazaka, Köln, Germany) [66]. The erythema index is calculated, in arbitrary units, of a circular 5 mm^2 area from the quantity of the reflected light when emitting light at wavelengths of 568 and 660 nm [73]. An erythema score of 1–18 indicates an absence of erythema, 19–36 indicates minimal erythema, 37–54 indicates diffused redness, 55–80 indicates high erythema, and 80–99 indicates that the skin is extremely erythematous [72]. A melanin index is established through measurement of the amount of absorbed and reflected red and infrared light; a greater absorption of light at these wavelengths is indicative of increased melanin [66]. Readings correlate with the darkness of the skin as per Fitzpatrick Skin Type Classification [74]; a melanin score of 1–8 is correlated with Pale White Skin (Type I), 9–13 with White Skin (Type II), 14–21 with Light Brown Skin (Type III), 22–30 with Moderate Brown Skin (Type IV), 31–37 with Dark Brown Skin (Type V), and 38–99 with Dark Brown to Black Skin (Type VI). The reported inter-individual day-to-day repeatability with the Mexameter indicates reliable measurements with a very low variability (1%) and a precision of ±5% [74–76].

2.6.3. Exergen DermaTemp

The Exergen DermaTemp DT-1001 RS Infrared Thermographic Scanner (Exergen Corporation, 2008) [77] was used to measure participants' skin surface temperature in degrees Celsius (°C). The device measures the heat naturally emitting from the body when held ~5 mm from the skin surface, and is capable of detecting subtle skin temperature variations as, for example, caused by underlying perfusion variations [77]. While there is no formal interpretation guide for skin temperature values as measured using the Exergen Derma Temp, most thermoregulation studies provide mean skin temperature values. mean skin temperature values are often reported in thermoregulation studies [78–80]. Assuming neutral (21–25 °C) ambient temperatures and a core temperature of ~37.0 °C, skin temperature at the trunk of the human body varies between 33.5 and 36.9 °C, and between 23.5 and 35.6 °C at the extremities.

2.7. Data collection procedures

2.7.1. Baseline and sequential skin parameter appraisal at anatomical testing sites

Participants attended two study visits. In effect, each participant served as their own matched control. In visit 1, written informed consent was obtained and participants' demographic and health data were recorded. Participants were provided with a hospital-type back-tie exam gown to change into, and were subsequently allowed to acclimatise to the temperature of the room for at least 15 min to allow full adaptation of their skin to the environmental conditions. As part of initial telephone screening, it was explicitly requested that participants do not apply topical products/preparations to the heels, elbows and/or lower back for at least 24 h prior to each visit. Participants were then asked to lie in a

supine position (0° incline of the head of the bed) on a hospital-grade bed/mattress composed of high-density polyurethane foam (30 kg/m^3 and 120 mm in thickness) fitted with a cotton-polyester bottom-sheet. A top-sheet and blanket were used to cover the participant and were folded at the participant's upper chest. The arms were left uncovered and extended along the midline of the body in the horizontal plane. Baseline skin parameter measurement was attended to consecutively at five anatomical testing sites: at the elbows and calcanei—with measurements taken bilaterally—and at the sacral region 2 cm caudal of the spinous process of L5. To enable measurement at the sacral skin, the participant repositioned laterally and the top-sheet was readjusted to briefly expose the lower back. On each measurement occasion, a single measure of skin temperature, stratum corneum hydration, erythema, and melanin were taken—in a 1 cm radius area—at each anatomical testing site. Skin temperature preceded all other parameters with consideration to heat dissipation [2,81] on exposure of the skin to the ambient environment. Once baseline measurements were recorded, an electronic timer was set to time intervals specific to either the uninterrupted or interrupted method (with the order in which the methods were attended to determined through randomisation; see Section 3.6.2). In the uninterrupted method, participants lay immobile continuously for 60 min with skin parameter measurements recorded immediately after the skin was unloaded at two timepoints; baseline (*T*0) and end (*T*60). In the interrupted method, measurements were recorded at 10 min intervals (repeated 7 times) over 60 min. Participants completed both methods in a single visit. A 15 min washout period was used as a pragmatic interval to separate the two methods, which was determined to be of sufficient duration for the results to return to baseline. The participant was instructed to stand up from the bed whilst skin parameters were re-assessed at 5 min intervals as per the washout period protocol. Endpoint measures of the washout period were attended to with the participant lying back down in the supine position, and corresponded to baseline results of the second method.

Visit 2 took place on a separate occasion with the same participant. All procedural aspects were duplicated as per visit 1, however, participants rested in a semi-recumbent position (45° incline of the head of the bed) instead of supine.

2.7.2. Allocation sequence generation and concealment

A computer-generated method allocation schedule was implemented by using allocation concealment via sequentially-numbered sealed opaque envelopes. The sequence of methods was generated using a random list via a Microsoft Excel spreadsheet (Microsoft Excel 2018, Microsoft Corp, Redmond, Washington, USA).

2.7.3. Justification for intermittent off-loading frequency

The tissue off-loading frequency in the current study was justified with consideration to research by Loerakker et al. (2010) [82] who investigated the effects of load-exposure time and intermittent load-relief on the development of deformation-induced tissue damage in animal models. A 2 h continuous loading regime was compared with 2 h of intermittent 10 min loading (with a 2 min recovery in between). A controlled mechanical system via an indenter was used to apply compression to the tibialis anterior of brown rats (n = 6). Magnetic resonance images were used to reconstruct the spatial locations where tissue changes appeared after a certain period and magnitude of mechanical loading. In this way, local tissue damage could be directly related to the areas with high strains within tissue. Results implied that intermittent off-loading during the 2 h loading period had minimal effect on tissue damage.

Table 1
Participant demographic profile for the current study (n = 41).

	Total n = 41
Age (years) [M (SD)]	27.9 (10.6)
[Med (IQR)]	25.0 (11.0)
Min.-Max.	18.0–60.0
Sex (% female)	68.3
Ethnicity (%)	
Caucasian	63.2
Asian	23.7
Hispanic	8.4
Middle Eastern	4.7
Body Mass Index (kg/m^2) [M (SD)]	24.8 (2.8)
[Med (IQR)]	24.5 (3.1)
Min.-Max.	19.0–30.5
% Within normal range (18.5–24.9)	70.7
% Underweight (<18.5)	–
% Overweight (25–29.9)	29.3
% Obese (>30 kg)	–

IQR, interquartile range.
M, mean; SD, standard deviation.
Med, median; IQR; interquartile range.
Min, minimum; Max, maximum.

2.8. Statistical analyses

Demographic data were explored using descriptive statistics and frequencies. Multi-level models were used to capture the differences between methods and timepoints within participants. The overall differences between methods and timepoints (baseline and final) were modelled as fixed effects within multi-level models in order to quantify their associations with skin parameters. Intraclass correlation coefficients (ICC) were calculated to indicate test-retest reliability and absolute agreement of results between methods [83]. These were termed as ICC^1 (test-retest reliability) and ICC^2 (absolute agreement). See equations 1 and 2 below for their definitions.

Equation 1: ICC$_1$ (test-retest reliability) which indicates agreement between measurements on the same participant using the same method, defined as:

$$\frac{\text{variability (individual differences) between participants} + \text{variability of the methods within a participant}}{\text{variability (individual differences) between participants} + \text{variability of the methods within a participant} + \text{random error}}$$

Equation 2: ICC$_2$ (absolute agreement) which indicates absolute agreement between the two methods, defined as:2

$$\frac{\text{variability (individual differences) between participants}}{\text{variability (individual differences) between participants} + \text{variability of the methods within a participant} + \text{random error}}$$

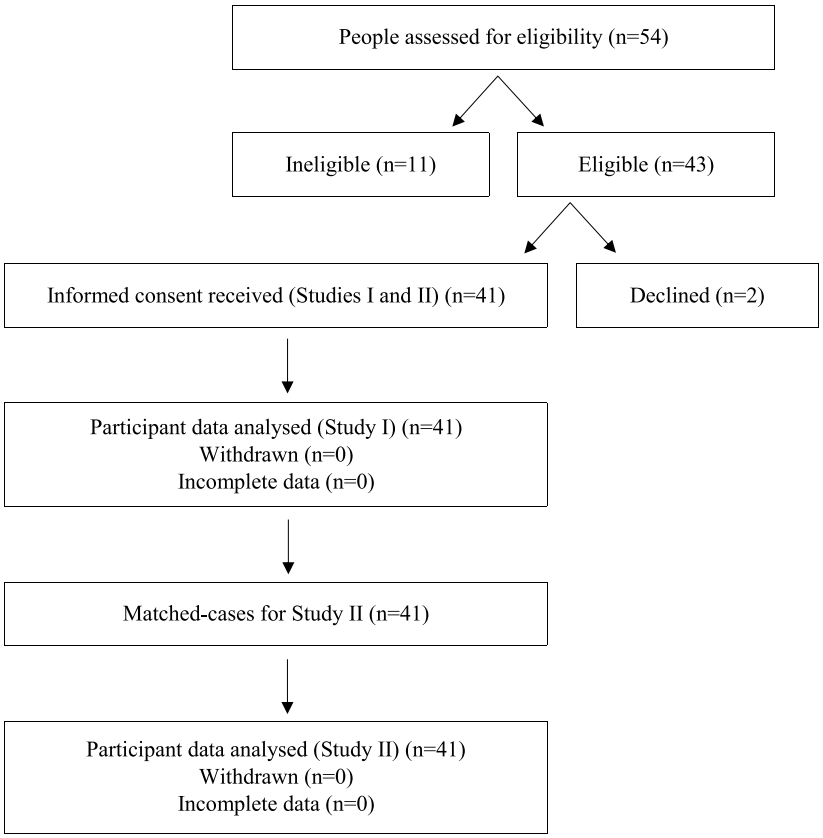

Fig. 1. Study participant flow diagram illustrating recruited participants' involvement at baseline screenings, with indication of withdrawals and inclusion in analyses.

Table 2
Uninterrupted and interrupted erythema measurements by anatomical location - Supine (n = 41).

			Time (minutes)						
			0	10	20	30	40	50	60
Right Elbow	Uninterrupted	M (SD)	21.5 (10.1)						25.6 (12.7)
		Med (IQR)	21.0 (16.5)						22.0 (20.0)
		Min.-Max.	6.0–42.0						6.0–59.0
	Interrupted	M (SD)	21.3 (11.1)	21.4 (11.2)	20.7 (10.5)	21.4 (11.1)	20.8 (10.6)	22.2 (12.9)	22.8 (12.6)
		Med (IQR)	19.0 (15.0)	20.0 (15.0)	21.0 (15.0)	21.0 (17.0)	19.0 (13.0)	19.0 (17.5)	20.0 (17.0)
		Min.-Max.	4.0–50.0	5.0–48.0	4.0–42.0	2.0–49.0	5.0–48.0	3.0–50.0	6.0–55.0
Left Elbow	Uninterrupted	M (SD)	21.5 (11.9)						24.7 (12.7)
		Med (IQR)	20.0 (17.5)						22.0 (18.5)
		Min.-Max.	5.0–51.0						7.0–53.0
	Interrupted	M (SD)	21.6 (11.9)	20.0 (11.0)	21.5 (11.9)	21.5 (12.6)	20.9 (12.3)	22.0 (11.8)	21.7 (11.4)
		Med (IQR)	20.0 (16.0)	19.0 (17.0)	25.2 (16.0)	18.0 (19.0)	17.0 (19.5)	19.0 (14.5)	20.0 (16.5)
		Min.-Max.	3.0–49.0	2.0–49.0	2.0–52.0	5.0–51.0	2.0–48.0	6.0–51.0	6.0–49.0
Right Heel	Uninterrupted	M (SD)	22.9 (10.6)						26.5 (12.7)
		Med (IQR)	22.0 (15.5)						29.0 (21.5)
		Min.-Max.	4.0–49.0						8.0–58.0
	Interrupted	M (SD)	23.0 (10.8)	23.4 (11.0)	24.6 (12.3)	23.4 (10.9)	23.3 (11.3)	24.2 (11.5)	24.4 (11.8)
		Med (IQR)	21.0 (18.0)	22.0 (17.5)	23.0 (19.0)	21.0 (15.5)	21.0 (17.5)	22.0 (18.0)	22.0 (16.0)
		Min.-Max.	4.0–42.0	4.0–51.0	4.0–52.0	5.0–51.0	5.0–52.0	4.0–49.0	5.0–55.0
Left Heel	Uninterrupted	M (SD)	25.4 (11.5)						27.5 (12.4)
		Med (IQR)	29.0 (17.0)						30.0 (22.0)
		Min.-Max.	4.0–50.0						8.0–60.0
	Interrupted	M (SD)	23.9 (11.5)	24.5 (11.3)	24.0 (11.5)	25.2 (13.0)	25.2 (12.4)	24.4 (11.5)	25.3 (11.3)
		Med (IQR)	22.0 (17.0)	29.0 (16.5)	22.0 (17.0)	24.0 (20.5)	26.0 (19.5)	22.0 (18.0)	26.0 (16.5)
		Min.-Max.	5.0–55.0	4.0–53.0	3.0–52.0	5.0–54.0	5.0–61.0	4.0–44.0	4.0–49.0
Sacrum	Uninterrupted	M (SD)	27.2 (12.4)						33.5 (13.0)
		Med (IQR)	28.0 (20.0)						32.0 (21.5)
		Min.-Max.	7.0–55.0						8.0–55.0
	Interrupted	M (SD)	26.9 (12.5)	26.7 (12.3)	27.9 (12.6)	28.3 (13.0)	28.1 (12.7)	30.1 (12.7)	30.3 (13.0)
		Med (IQR)	29.0 (21.0)	27.0 (19.5)	29.0 (19.0)	29.0 (21.5)	29.0 (22.5)	30.0 (21.0)	31.0 (21.5)
		Min.-Max.	9.0–55.0	8.0–55.0	8.0–56.0	7.0–56.0	7.0–59.0	7.0–61.0	8.0–59.0

Table 3
Uninterrupted and interrupted melanin measurements by anatomical location - Supine (n = 41).

			Timepoint (minutes)						
			0	10	20	30	40	50	60
Right Elbow	Uninterrupted	M (SD)	22.1 (30.0)						23.0 (29.8)
		Med (IQR)	6.0 (29.0)						7.0 (30.5)
		Min.-Max.	1.0–99.0						1.0–98.0
	Interrupted	M (SD)	22.4 (29.2)	22.3 (28.6)	22.1 (28.5)	21.8 (27.7)	22.0 (28.7)	22.9 (29.3)	22.8 (29.1)
		Med (IQR)	8.0 (31.0)	8.0 (29.0)	6.0 (29.0)	8.0 (30.5)	7.0 (30.0)	9.0 (26.5)	9.0 (28.5)
		Min.-Max.	1.0–92.0	1.0–90.0	1.0–90.0	1.0–88.0	1.0–91.0	1.0–91.0	1.0–91.0
Left Elbow	Uninterrupted	M (SD)	22.0 (29.6)						22.3 (28.9)
		Med (IQR)	8.0 (28.5)						8.0 (27.5)
		Min.-Max.	1.0–89.0						1.0–89.0
	Interrupted	M (SD)	22.1 (28.8)	21.9 (29.3)	21.8 (28.8)	22.3 (28.7)	22.0 (28.2)	21.8 (28.6)	22.5 (28.4)
		Med (IQR)	8.0 (31.0)	8.0 (29.0)	8.0 (30.5)	9.0 (29.5)	8.0 (29.5)	9.0 (24.0)	8.0 (29.0)
		Min.-Max.	1.0–98.0	1.0–92.0	1.0–92.0	1.0–90.0	1.0–89.0	1.0–89.0	1.0–91.0
Right Heel	Uninterrupted	M (SD)	20.8 (27.1)						21.0 (27.9)
		Med (IQR)	8.0 (22.5)						8.0 (23.5)
		Min.-Max.	1.0–89.0						1.0–91.0
	Interrupted	M (SD)	21.3 (27.5)	21.3 (28.7)	22.0 (29.4)	21.3 (28.7)	20.9 (28.1)	21.8 (28.4)	21.7 (28.3)
		Med (IQR)	9.0 (24.0)	9.0 (24.0)	8.0 (27.5)	8.0 (27.5)	7.0 (22.5)	9.0 (21.5)	8.0 (27.0)
		Min.-Max.	1.0–91.0	1.0–99.0	1.0–99.0	1.0–98.0	1.0–90.0	1.0–89.0	1.0–89.0
Left Heel	Uninterrupted	M (SD)	21.4 (28.2)						21.4 (28.3)
		Med (IQR)	8.0 (21.0)						8.0 (20.5)
		Min.-Max.	1.0–92.0						1.0–98.0
	Interrupted	M (SD)	20.7 (26.8)	20.1 (27.0)	20.9 (28.4)	21.1 (27.7)	21.2 (27.3)	21.2 (27.7)	21.2 (28.3)
		Med (IQR)	8.0 (23.5)	8.0 (21.5)	8.0 (21.5)	9.0 (22.0)	8.0 (20.5)	8.0 (25.0)	8.0 (20.5)
		Min.-Max.	1.0–88.0	1.0–89.0	1.0–99.0	1.0–89.0	1.0–89.0	1.0–91.0	1.0–98.0
Sacrum	Uninterrupted	M (SD)	27.0 (31.2)						27.2 (31.3)
		Med (IQR)	12.0 (31.0)						11.0 (32.5)
		Min.-Max.	1.0–99.0						1.0–99.0
	Interrupted	M (SD)	26.3 (31.9)	25.9 (31.9)	26.3 (31.8)	26.6 (31.1)	26.6 (31.3)	26.8 (32.2)	27.4 (31.8
		Med (IQR)	11.0 (32.0)	12.0 (32.0)	12.0 (31.5)	12.0 (27.0)	12.0 (29.0)	12.0 (32.0)	11.0 (30.0)
		Min.-Max.	2.0–99.0	2.0–99.0	1.0–99.0	1.0–99.0	1.0–99.0	1.0–99.0	2.0–99.0

Table 4
Uninterrupted and interrupted epidermal hydration measurements by anatomical location - Supine (n = 41).

			Timepoint (minutes)						
			0	10	20	30	40	50	60
Right Elbow	Uninterrupted	M (SD)	7.3 (6.9)						7.9 (7.3)
		Med (IQR)	6.0 (9.0)						9.0 (10.5)
		Min.-Max.	0.0–30.0						0.0–34.0
	Interrupted	M (SD)	6.2 (6.6)	6.2 (7.2)	6.5 (7.5)	6.5 (6.8)	6.2 (6.9)	6.9 (7.3)	7.0 (7.4)
		Med (IQR)	5.0 (8.5)	5.0 (9.5)	5.0 (10.0)	6.0 (9.5)	5.0 (8.0)	5.0 (9.0)	6.0 (9.5)
		Min.-Max.	0.0–33.0	0.0–39.0	0.0–41.0	0.0–31.0	0.0–32.0	0.0–34.0	0.0–36.0
Left Elbow	Uninterrupted	M (SD)	7.2 (7.7)						7.7 (7.5)
		Med (IQR)	6.0 (10.0)						8.0 (11.0)
		Min.-Max.	0.0–39.0						0.0–32.0
	Interrupted	M (SD)	6.5 (6.7)	6.9 (6.6)	6.6 (7.4)	6.8 (7.5)	6.4 (7.0)	6.5 (7.0)	6.7 (7.7)
		Med (IQR)	5.0 (9.0)	8.0 (9.9)	6.0 (8.5)	6.0 (10.0)	5.0 (10.0)	5.0 (9.5)	5.0 (9.5)
		Min.-Max.	0.0–31.0	0.0–33.0	0.0–39.0	0.0–41.0	0.0–33.0	0.0–34.0	0.0–39.0
Right Heel	Uninterrupted	M (SD)	7.2 (14.0)						7.5 (14.5)
		Med (IQR)	0.0 (10.0)						0.0 (9.5)
		Min.-Max.	0.0–60.0						0.0–62.0
	Interrupted	M (SD)	7.0 (14.3)	7.0 (13.4)	7.4 (14.2)	7.2 (13.3)	7.3 (13.8)	7.4 (13.3)	7.6 (13.4)
		Med (IQR)	0.0 (11.0)	0.0 (8.5)	0.0 (10.5)	0.0 (10.0)	0.0 (9.5)	0.0 (9.5)	0.0 (10.5)
		Min.-Max.	0.0–61.0	0.0–61.0	0.0–63.0	0.0–59.0	0.0–56.0	0.0–52.0	0.0–66.0
Left Heel	Uninterrupted	M (SD)	7.0 (13.0)						7.8 (14.3)
		Med (IQR)	0.0 (10.5)						0.0 (10.5)
		Min.-Max.	0.0–49.0						0.0–58.0
	Interrupted	M (SD)	7.0 (13.4)	7.3 (13.2)	7.2 (13.2)	7.3 (14.1)	7.3 (13.4)	7.4 (13.6)	7.6 (13.8)
		Med (IQR)	0.0 (9.5)	0.0 (10.0)	0.0 (10.0)	0.0 (8.5)	0.0 (10.0)	0.0 (10.0)	0.0 (11.0)
		Min.-Max.	0.0–55.0	0.0–52.0	0.0–50.0	0.0–61.0	0.0–57.0	0.0–55.0	0.0–53.0
Sacrum	Uninterrupted	M (SD)	19.8 (8.6)						25.0 (11.0)
		Med (IQR)	18.0 (8.0)						22.0 (11.0)
		Min.-Max.	6.0–49.0						7.0–57.0
	Interrupted	M (SD)	16.9 (8.0)	17.7 (8.5)	18.5 (8.9)	20.3 (9.3)	20.7 (8.9)	21.4 (7.7)	22.5 (9.1)
		Med (IQR)	16.0 (9.0)	16.0 (9.0)	16.0 (7.5)	18.0 (8.0)	19.0 (8.0)	20.0 (8.5)	20.0 (10.5)
		Min.-Max.	6.0–41.0	6.0–52.0	3.0–49.0	6.0–52.0	6.0–49.0	6.0–41.0	5.0–49.0

Table 5
Uninterrupted and interrupted temperature measurements by anatomical location –Supine (n = 41).

			Timepoint (minutes)						
			0	10	20	30	40	50	60
Right Elbow	Uninterrupted	M (SD)	29.1 (1.1)						30.7 (1.1)
		Med (IQR)	29.5 (1.6)						30.6 (1.6)
		Min.-Max.	26.5–30.9						28.5–33.0
	Interrupted	M (SD)	28.9 (1.1)	29.2 (1.1)	29.4 (1.1)	29.6 (1.1)	29.9 (1.1)	30.1 (1.1)	30.4 (1.1)
		Med (IQR)	29.1 (1.8)	29.3 (1.7)	30.0 (1.5)	30.0 (1.6)	30.0 (1.7)	30.1 (1.6)	30.2 (1.5)
		Min.-Max.	26.0–30.6	26.6–31.0	26.9–31.0	27.1–31.5	27.5–31.9	28.0–32.1	28.2–32.9
Left Elbow	Uninterrupted	M (SD)	29.1 (1.0)						30.7 (1.1)
		Med (IQR)	29.2 (1.2)						30.9 (1.7)
		Min.-Max.	26.4–30.9						28.4–32.9
	Interrupted	M (SD)	28.9 (1.1)	29.1 (1.1)	29.4 (1.1)	29.6 (1.1)	29.9 (1.1)	30.2 (1.1)	30.4 (1.1)
		Med (IQR)	29.2 (1.3)	29.2 (1.3)	30.0 (1.4)	29.7 (1.6)	29.9 (1.7)	30.2 (1.6)	30.3 (1.6)
		Min.-Max.	25.9–30.9	26.4–30.9	26.8–31.0	27.2–31.6	27.2–31.9	28.0–32.2	28.2–32.8
Right Heel	Uninterrupted	M (SD)	27.9 (1.8)						29.6 (1.9)
		Med (IQR)	28.0 (3.0)						29.6 (2.9)
		Min.-Max.	24.5–31.0						25.9–33.4
	Interrupted	M (SD)	27.6 (1.9)	27.8 (1.8)	28.0 (1.8)	28.3 (1.8)	28.6 (1.8)	29.0 (1.8)	29.2 (1.8)
		Med (IQR)	27.6 (3.2)	27.7 (3.1)	28.0 (2.7)	28.2 (2.9	28.3 (2.6)	28.6 (2.9)	29.0 (2.7)
		Min.-Max.	24.1–30.8	24.3–30.9	24.5–31.2	24.8–31.9	24.8–32.1	25.2–32.5	25.7–33.1
Left Heel	Uninterrupted	M (SD)	27.8 (1.8)						29.6 (1.9)
		Med (IQR)	28.0 (3.2)						29.8 (2.9)
		Min.-Max.	24.0–30.9						25.6–33.5
	Interrupted	M (SD)	27.6 (1.9)	27.8 (1.8)	28.1 (1.8)	28.4 (1.8)	28.7 (1.8)	29.0 (1.8)	29.2 (1.9)
		Med (IQR)	28.0 (3.0)	28.0 (2.8)	28.2 (2.8)	28.2 (3.0)	28.5 (2.8)	28.6 (2.9)	28.6 (2.3)
		Min.-Max.	23.8–30.6	24.1–30.8	24.6–31.6	24.7–32.0	24.9–32.2	25.2–32.9	25.4–33.2
Sacrum	Uninterrupted	M (SD)	31.3 (0.8)						33.8 (0.9)
		Med (IQR)	31.2 (1.1)						33.9 (1.4)
		Min.-Max.	29.5–32.6						31.8–35.1
	Interrupted	M (SD)	30.8 (0.8)	31.2 (0.9)	31.7 (1.0)	32.1 (1.0)	32.6 (1.1)	33.4 (2.2)	33.4 (1.0)
		Med (IQR)	30.9 (1.2)	31.1 (1.6)	31.6 (1.5)	32.2 (1.4)	32.9 (1.7)	32.9 (1.5)	33.5 (1.3)
		Min.-Max.	29.0–32.5	29.2–32.9	29.5–33.8	29.9–33.9	30.5–34.9	31.0–34.9	31.2–35.5

Table 6
Uninterrupted and interrupted erythema measurements by anatomical location - Semi-recumbent (n = 41).

			Time (minutes)						
			0	10	20	30	40	50	60
Right Elbow	Uninterrupted	M (SD)	24.0 (11.5)						30.3 (12.5)
		Med (IQR)	21.0 (14.5)						31.0 (19.0)
		Min.-Max.	5.0–58.0						6.0–60.0
	Interrupted	M (SD)	23.1 (11.4)	23.3 (10.9)	22.9 (10.5)	23.7 (12.3)	24.6 (10.8)	25.9 (11.5)	26.7 (12.8)
		Med (IQR)	21.0 (15.5)	21.0 (16.5)	21.0 (16.0)	22.0 (17.5)	24.0 (16.5)	25.0 (19.0)	22.0 (18.0)
		Min.-Max.	4.0–55.0	5.0–49.0	2.0–42.0	4.0–58.0	3.0–49.0	5.0–52.0	9.0–59.0
Left Elbow	Uninterrupted	M (SD)	24.0 (10.9)						31.0 (11.5)
		Med (IQR)	22.0 (14.5)						31.0 (18.5)
		Min.-Max.	5.0–51.0						13.0–61.0
	Interrupted	M (SD)	23.1 (11.6)	23.7 (12.4)	24.7 (12.1)	23.9 (12.7)	25.0 (12.0)	25.8 (12.2)	27.2 (12.1)
		Med (IQR)	21.0 (18.0)	22.0 (18.0)	25.0 (15.5)	21.0 (16.0)	22.0 (18.5)	24.0 (17.0)	25.0 (19.0)
		Min.-Max.	3.0–49.0	4.0–55.0	5.0–59.0	3.0–61.0	5.0–58.0	7.0–59.0	7.0–56.0
Right Heel	Uninterrupted	M (SD)	25.2 (11.1)						32.3 (12.0)
		Med (IQR)	26.0 (17.5)						33.0 (20.0)
		Min.-Max.	7.0–52.0						12.0–59.0
	Interrupted	M (SD)	24.3 (11.4)	23.6 (10.2)	24.5 (10.7)	26.3 (11.3)	26.5 (11.5)	26.8 (11.1)	27.4 (11.4)
		Med (IQR)	23.0 (18.5)	22.0 (17.0)	21.0 (17.0)	26.0 (17.5)	27.0 (19.0)	28.0 (18.0)	26.0 (19.0)
		Min.-Max.	6.0–52.0	6.0–42.0	5.0–49.0	5.0–53.0	7.0–51.0	9.0–51.0	6.0–52.0
Left Heel	Uninterrupted	M (SD)	26.3 (11.4)						33.4 (12.0)
		Med (IQR)	26.0 (19.5)						34.0 (19.0)
		Min.-Max.	9.0–51.0						12.0–56.0
	Interrupted	M (SD)	25.2 (11.3)	25.3 (11.8)	25.1 (11.4)	25.6 (12.2)	27.0 (12.5)	27.3 (12.2)	27.8 (11.9)
		Med (IQR)	26.0 (15.0)	26.0 (16.5)	26.0 (18.5)	26.0 (18.0)	27.0 (19.0)	29.0 (19.0)	26.0 (20.0)
		Min.-Max.	5.0–55.0	4.0–59.0	4.0–51.0	4.0–52.0	8.0–55.0	5.0–58.0	6.0–57.0
Sacrum	Uninterrupted	M (SD)	30.2 (13.0)						41.8 (15.6)
		Med (IQR)	29.0 (19.0)						41.0 (22.0)
		Min.-Max.	10.0–58.0						11.0–71.0
	Interrupted	M (SD)	30.1 (12.5)	29.5 (11.9)	30.5 (13.0)	30.5 (11.5)	32.1 (12.9)	34.1 (13.3)	35.0 (13.3)
		Med (IQR)	31.0 (20.0)	30.0 (17.0)	31.0 (17.0)	30.0 (13.0)	32.0 (15.5)	36.0 (18.5)	36.0 (16.0)
		Min.-Max.	9.0–55.0	10.0–54.0	10.0–58.0	11.0–56.0	8.0–56.0	10.0–61.0	11.0–62.0

Table 7
Uninterrupted and interrupted melanin measurements by anatomical location - Semi-recumbent (n = 41).

			Timepoint (minutes)						
			0	10	20	30	40	50	60
Right Elbow	Uninterrupted	M (SD)	21.1 (28.6)						21.9 (29.2)
		Med (IQR)	7.0 (19.5)						8.0 (20.5)
		Min.-Max.	1.0–97.0						1.0–98.0
	Interrupted	M (SD)	22.0 (29.3)	21.5 (29.1)	21.3 (29.4)	20.2 (27.7)	21.3 (28.4)	21.4 (28.2)	21.5 (28.9)
		Med (IQR)	8.0 (18.5)	8.0 (17.5)	6.0 (20.0)	6.0 (17.5)	8.0 (22.0)	6.0 (20.5)	8.0 (21.5)
		Min.-Max.	1.0–98.0	1.0–99.0	1.0–88.0	1.0–85.0	1.0–89.0	1.0–91.0	1.0–98.0
Left Elbow	Uninterrupted	M (SD)	20.9 (27.5)						22.2 (28.3)
		Med (IQR)	8.0 (30.5)						8.0 (19.5)
		Min.-Max.	1.0–88.0						1.0–89.0
	Interrupted	M (SD)	21.5 (29.0)	20.6 (28.4)	21.2 (28.9)	21.2 (28.2)	21.5 (28.4)	21.5 (28.1)	22.5 (28.5)
		Med (IQR)	7.0 (19.0)	6.0 (17.5)	7.0 (17.5)	9.0 (19.0)	8.0 (16.0)	8.0 (20.0)	7.0 (17.5)
		Min.-Max.	1.0–92.0	1.0–93.0	1.0–91.0	1.0–86.0	1.0–89.0	1.0–91.0	1.0–91.0
Right Heel	Uninterrupted	M (SD)	19.9 (26.3)						19.9 (26.6)
		Med (IQR)	8.0 (14.0)						8.0 (17.5)
		Min.-Max.	1.0–84.0						1.0–91.0
	Interrupted	M (SD)	20.3 (26.3)	20.8 (27.1)	19.7 (27.1)	20.5 (27.5)	20.7 (27.4)	20.0 (26.5)	20.0 (27.1)
		Med (IQR)	8.0 (15.5)	8.0 (19.5)	6.0 (14.5)	8.0 (16.5)	8.0 (16.5)	9.0 (17.0)	8.0 (17.5)
		Min.-Max.	2.0–91.0	2.0–92.0	1.0–88.0	1.0–99.0	1.0–88.0	1.0–92.0	1.0–92.0
Left Heel	Uninterrupted	M (SD)	19.9 (25.8)						21.0 (27.8)
		Med (IQR)	8.0 (16.0)						8.0 (16.5)
		Min.-Max.	1.0–89.0						1.0–91.0
	Interrupted	M (SD)	19.8 (25.9)	20.0 (25.7)	20.1 (26.2)	20.5 (26.7)	20.3 (26.6)	19.6 (26.6)	20.2 (26.7)
		Med (IQR)	8.0 (18.0)	8.0 (16.0)	8.0 (19.0)	8.0 (17.0)	7.0 (18.0)	7.0 (17.5)	8.0 (17.0)
		Min.-Max.	2.0–89.0	2.0–88.0	1.0–84.0	1.0–86.0	1.0–85.0	1.0–86.0	1.0–91.0
Sacrum	Uninterrupted	M (SD)	25.9 (31.3)						26.7 (31.8)
		Med (IQR)	12.0 (22.5)						11.0 (23.5)
		Min.-Max.	1.0–99.0						2.0–99.0
	Interrupted	M (SD)	25.5 (31.6)	25.6 (31.8)	26.2 (31.7)	26.4 (30.7)	26.6 (31.4)	25.6 (30.8)	26.0 (31.3)
		Med (IQR)	11.0 (22.5)	12.0 (25.0)	12.0 (28.5)	12.0 (28.5)	12.0 (32.0)	12.0 (26.5)	12.0 (29.5)
		Min.-Max.	2.0–99.0	2.0–99.0	2.0–99.0	2.0–99.0	1.0–99.0	2.0–99.0	2.0–99.0

Table 8
Uninterrupted and interrupted epidermal hydration measurements by anatomical location – Semi-recumben) (n = 41).

			Timepoint (minutes)						
			0	10	20	30	40	50	60
Right Elbow	Uninterrupted	M (SD)	7.1 (6.7)						9.7 (8.7)
		Med (IQR)	6.0 (5.9)						8.0 (13.0)
		Min.-Max.	0.0–26.0						0.0–35.0
	Interrupted	M (SD)	7.3 (6.9)	6.8 (7.2)	7.1 (6.9)	6.7 (6.3)	6.7 (6.6)	7.4 (6.5)	7.5 (7.4)
		Med (IQR)	6.0 (8.5)	6.0 (8.0)	6.0 (8.0)	6.0 (8.0)	6.0 (8.0)	7.0 (8.5)	7.0 (7.5)
		Min.-Max.	0.0–33.0	0.0–35.0	0.0–34.0	0.0–29.0	0.0–30.0	0.0–29.0	0.0–37.0
Left Elbow	Uninterrupted	M (SD)	7.7 (7.7)						9.7 (8.3)
		Med (IQR)	6.0 (9.0)						9.0 (9.5)
		Min.-Max.	0.0–30.0						0.0–39.0
	Interrupted	M (SD)	6.9 (6.4)	7.3 (6.8)	6.6 (6.5)	7.3 (6.9)	6.8 (7.1)	7.3 (7.1)	7.7 (7.8)
		Med (IQR)	6.0 (8.0)	6.0 (8.0)	5.0 (7.5)	6.0 (9.0)	5.0 (8.0)	5.0 (8.5)	6.0 (9.0)
		Min.-Max.	0.0–31.0	0.0–34.0	0.0–35.0	0.0–33.0	0.0–31.0	0.0–31.0	0.0–35.0
Right Heel	Uninterrupted	M (SD)	5.6 (13.5)						6.2 (15.0)
		Med (IQR)	0.0 (6.5)						0.0 (5.0)
		Min.-Max.	0.0–71.0						0.0–79.0
	Interrupted	M (SD)	5.7 (13.7)	6.0 (14.0)	6.2 (14.2)	6.4 (13.3)	6.3 (14.6)	6.5 (14.7)	6.5 (14.4)
		Med (IQR)	0.0 (8.0)	0.0 (9.0)	0.0 (9.5)	0.0 (10.0)	0.0 (7.5)	0.0 (8.5)	0.0 (10.0)
		Min.-Max.	0.0–71.0	0.0–72.0	0.0–72.0	0.0–59.0	0.0–77.0	0.0–73.0	0.0–71.0
Left Heel	Uninterrupted	M (SD)	5.7 (13.9)						6.1 (14.3)
		Med (IQR)	0.0 (5.0)						0.0 (3.5)
		Min.-Max.	0.0–70.0						0.0–72.0
	Interrupted	M (SD)	6.3 (14.1)	6.3 (14.1)	6.2 (13.1)	6.2 (13.7)	6.3 (14.0)	6.5 (14.2)	6.6 (14.3)
		Med (IQR)	0.0 (8.0)	0.0 (6.0)	0.0 (8.0)	0.0 (5.0)	0.0 (7.5)	0.0 (7.0)	0.0 (8.5)
		Min.-Max.	0.0–70.0	0.0–66.0	0.0–61.0	0.0–67.0	0.0–69.0	0.0–71.0	0.0–70.0
Sacrum	Uninterrupted	M (SD)	20.7 (9.9)						27.9 (13.9)
		Med (IQR)	18.0 (12.5)						26.0 (19.0)
		Min.-Max.	8.0–50.0						8.0–71.0
	Interrupted	M (SD)	19.7 (10.1)	20.9 (10.7)	21.0 (11.7)	22.4 (10.8)	23.2 (12.5)	24.4 (12.8)	25.5 (12.7)
		Med (IQR)	18.0 (11.5)	20.0 (11.0)	18.0 (12.5)	20.0 (12.0)	20.0 (13.5)	21.0 (17.0)	22.0 (20.5)
		Min.-Max.	7.0–41.0	7.0–52.0	6.0–56.0	5.0–56.0	7.0–59.0	7.0–61.0	5.0–62.0

Table 9
Uninterrupted and interrupted temperature measurements by anatomical location – Semi-recumbent (n = 41.

			Timepoint						
			0	10	20	30	40	50	60
Right Elbow	Uninterrupted	M (SD)	29.0 (1.1)						31.1 (1.0)
		Med (IQR)	29.2 (1.8)						31.1 (1.6)
		Min.-Max.	26.2–30.8						29.0–32.8
	Interrupted	M (SD)	28.9 (1.1)	29.1 (1.1)	29.4 (1.1)	29.7 (1.0)	29.9 (1.1)	30.5 (1.0)	30.8 (1.0)
		Med (IQR)	29.2 (1.8)	29.4 (1.9)	30.0 (2.0)	30.1 (1.4)	30.0 (1.7)	30.4 (1.3)	30.8 (1.5)
		Min.-Max.	26.3–30.5	26.9–31.2	27.1–31.2	27.4–31.6	27.5–31.9	28.4–32.4	28.9–32.5
Left Elbow	Uninterrupted	M (SD)	30.1 (1.2)						31.2 (1.0)
		Med (IQR)	29.1 (1.7)						31.3 (1.1)
		Min.-Max.	26.4–32.3						29.2–32.9
	Interrupted	M (SD)	28.9 (1.1)	29.1 (1.1)	29.4 (1.1)	29.8 (1.0)	30.1 (1.0)	30.5 (1.0)	30.8 (1.0)
		Med (IQR)	29.2 (2.0)	30.0 (2.0)	29.8 (2.2)	30.0 (1.6)	30.2 (1.2)	30.5 (1.4)	30.8 (1.4)
		Min.-Max.	26.3–30.5	26.7–30.8	27.2–31.0	27.6–31.5	28.3–32.0	28.8–32.3	29.0–32.7
Right Heel	Uninterrupted	M (SD)	27.9 (1.7)						30.0 (1.6)
		Med (IQR)	28.1 (2.7)						30.3 (2.9)
		Min.-Max.	24.4–30.1						26.8–32.5
	Interrupted	M (SD)	27.7 (1.8)	28.0 (1.7)	28.4 (1.8)	28.6 (1.7)	29.0 (1.7)	29.3 (1.6)	30.1 (1.6)
		Med (IQR)	28.0 (3.3)	28.3 (3.1)	28.6 (3.3)	28.9 (3.3)	29.0 (3.1)	29.7 (2.8)	29.9 (2.9)
		Min.-Max.	24.6–30.5	24.7–30.7	25.1–30.9	25.6–31.2	25.7–31.4	26.1–31.6	26.3–32.1
Left Heel	Uninterrupted	M (SD)	27.9 (1.8)						30.0 (1.6)
		Med (IQR)	28.2 (3.1)						30.5 (3.0)
		Min.-Max.	24.4–30.4						26.9–32.5
	Interrupted	M (SD)	27.7 (1.8)	28.1 (1.8)	28.4 (1.7)	28.7 (1.7)	29.0 (1.7)	29.4 (1.6)	29.6 (1.6)
		Med (IQR)	28.2 (3.1)	28.3 (3.5)	28.5 (3.0)	28.9 (3.0)	29.4 (3.0)	29.4 (3.1)	29.6 (3.1)
		Min.-Max.	24.3–30.3	24.6–30.9	25.2–31.0	25.3–31.2	25.9–31.6	26.3–31.8	26.8–32.3
Sacrum	Uninterrupted	M (SD)	31.4 (0.8)						34.3 (0.9)
		Med (IQR)	31.3 (1.1)						34.4 (1.0)
		Min.-Max.	30.0–33.1						31.9–35.9
	Interrupted	M (SD)	30.9 (0.9)	31.3 (1.0)	31.6 (1.1)	32.1 (1.0)	32.5 (1.0)	33.0 (1.0)	33.6 (1.0)
		Med (IQR)	30.6 (1.2)	31.0 (1.3)	31.3 (1.2)	32.0 (1.7)	32.5 (1.2)	32.9 (1.5)	33.5 (1.1)
		Min.-Max.	29.7–33.1	29.8–34.3	30.1–34.9	30.5–34.9	30.7–35.1	31.0–35.1	31.3–35.3

All statistical analyses were performed using the IBM Statistical Package for the Social Sciences (SPSS) Statistics, Release Version 23.0.0 (IBM Corporation, NY, USA, Release 2015) [84] and STATA Statistical Software: Release 13 (StataCorp, 2013) [85]. Statistical significance was set at $p < .05$.

2.9. Safety measures

As per the study's safety protocol, at each assessment interval, the researcher inspected the anatomical testing sites for the presence of skin irritation/damage by conducting a brief skin blanching test [15]. If the skin did not respond as desired, the study was stopped.

3. Results

The sample (n = 41) comprised 28 females and 13 males with a mean age of 27.9 (SD = 10.6) years. Table 1 provides an overview of participant demographic data and clinical characteristics. A participant flow diagram illustrating participants' involvement at baseline screening and inclusion in analyses is presented in Fig. 1

3.1. Descriptive results for skin parameters

Tables 2–5 and Tables 6–9 provide the descriptive results for skin parameters for the supine and semi-recumbent conditions, respectively.

3.1.1. Descriptive results for erythema

There was minimal variation in mean scores of erythema across timepoints at all anatomical testing sites and amongst individual study participants. The highest mean erythema score was registered at the sacrum at $T60$ (uninterrupted) in both supine and semi-recumbent positions (M = 33.5, SD = 13.0) and (M = 30.3, SD = 13.0), respectively. The mean erythema scores at the elbows and heels were fairly low when compared to those at the sacrum. To illustrate, mean erythema scores at the sacrum ranged between 26.9 and 41.8 units, and between 21.3–31.0 and 22.9–33.4 units at the elbows and heels, respectively. A large range of erythema scores was noted across anatomical testing sites (range: 2.0–71.0) with the lowest and highest scores recorded at the elbows and sacrum, respectively.

3.1.2. Descriptive results for melanin

There was minimal and, occasionally, no mean change in melanin scores across all timepoints at all anatomical testing sites and amongst individual participants. However, a wide range of melanin scores was noted (range: 1–99) between participants. When considering the measure of spread for melanin scores, the SDs were relatively large, indicating a high level of variance, with datapoints spread further away from the mean.

3.1.3. Descriptive results for stratum corneum hydration

There was minimal variation in mean scores of stratum corneum hydration across timepoints at all anatomical testing sites and amongst individual study participants. The highest mean score was recorded at the sacrum at $T60$ (uninterrupted) in both supine and semi-recumbent positions (M = 25.0, SD = 11.0) and (M = 27.9, SD = 13.9), respectively. On average, the sacrum registered marginally higher levels of hydration than the elbows and heels. To illustrate, mean hydration scores at the sacrum ranged from 16.9 to 27.9 units, while mean hydration scores at the elbows and heels ranged from 6.2 to 9.7 and 5.6–7.6 units, respectively. A large range of stratum corneum hydration scores was noted across anatomical testing sites (range: 0–66.0) with the lowest and highest scores recorded at the heels and sacrum, respectively.

3.1.4. Descriptive results for skin temperature

There was moderate variation in mean scores of skin temperature across timepoints at all anatomical testing sites and amongst individual study participants. The highest mean temperature score was recorded at the sacrum at $T60$ (uninterrupted) in both supine and semi-recumbent positions (M = 33.8, SD = 0.9) and (M = 34.3, SD = 0.9),

Table 10
Differences between methods and timepoints for erythema – Supine (n = 41).

	Variable	Estimate	95% Confidence Interval	*p* value
Right Elbow	Constant	23.7	(19.8, 27.5)	$p < .001$
	Interrupted compared to uninterrupted	−1.5	(-2.7, −0.4)	$p = .010$
	Final compared to baseline	0.5	(0.3, 0.6)	$p < .001$
Left Elbow	Constant	23.7	(19.7, 27.8)	$p < .001$
	Interrupted compared to uninterrupted	−1.4	(-2.7, −0.1)	$p = .038$
	Final compared to baseline	0.3	(0.1, 0.5)	$p = .013$
Right Heel	Constant	24.5	(20.8, 28.2)	$p < .001$
	Interrupted compared to uninterrupted	−1.0	(-2.0, −0.1)	$p = .037$
	Final compared to baseline	0.4	(0.3, 0.6)	$p = .001$
Left Heel	Constant	27.4	(23.6, 31.3)	$p < .001$
	Interrupted compared to uninterrupted	−1.9	(-3.0, −0.7)	$p = .002$
	Final compared to baseline	0.3	(0.1, 0.5)	$p < .003$
Sacrum	Constant	29.7	(25.2, 34.2)	$p < .001$
	Interrupted compared to uninterrupted	−1.8	(-3.5, −0.1)	$p = .040$
	Final compared to baseline	0.8	(0.6, 1.0)	$p < .001$

Table 11
Differences between methods and timepoints for melanin – Supine (n = 41).

	Variable	Estimate	95% Confidence Interval	*p* value
Right Elbow	Constant	22.2	(13.2, 31.1)	$p < .001$
	Interrupted compared to uninterrupted	0.1	(-0.6, 0.7)	$p = .831$
	Final compared to baseline	0.5	(-0.0, 0.2)	$p = .064$
Left Elbow	Constant	22.1	(13.2, 30.9)	$p < .001$
	Interrupted compared to uninterrupted	0.1	(-0.6, 0.9)	$p = .737$
	Final compared to baseline	0.0	(-0.1, 0.1)	$p = .866$
Right Heel	Constant	20.0	(11.6, 28.5)	$p < .001$
	Interrupted compared to uninterrupted	0.7	(-0.2, 1.5)	$p = .110$
	Final compared to baseline	0.1	(-0.0, 0.1)	$p = .303$
Left Heel	Constant	21.5	(13.0, 29.9)	$p < .001$
	Interrupted compared to uninterrupted	0.7	(-0.2, 1.7)	$p = .122$
	Final compared to baseline	0.1	(-0.0, 0.1)	$p = .315$
Sacrum	Constant	20.0	(17.3, 36.8)	$p < .001$
	Interrupted compared to uninterrupted	0.7	(-1.7, 1.1)	$p = .719$
	Final compared to baseline	0.1	(-0.0, 0.2)	$p = .089$

respectively. When compared to the elbows and heels, the sacrum registered higher maximum readings of skin temperature on each occasion. To illustrate, mean skin temperature scores at the sacrum ranged between 30.8 and 34.3 °C, and between 28.9–31.1 °C and 27.6–30.0 °C at the elbows and heels, respectively. A moderate range of skin temperature scores was noted across all anatomical testing sites; scores ranged from 25.9 to 31.2 °C at the elbows, 23.8–33.5 °C at the heels, and 29.0–35.9 °C at the sacrum.

Table 12
Differences between methods and timepoints for hydration – Supine (n = 41).

	Variable	Estimate	95% Confidence Interval	*p* value
Right Elbow	Constant	8.4	(6.1, 10.7)	$p < .001$
	Interrupted compared to uninterrupted	−1.1	(-1.7, −0.4)	$p < .001$
	Final compared to baseline	0.1	(0.0, 0.2)	$p = .021$
Left Elbow	Constant	8.1	(5.5, 10.7)	$p < .001$
	Interrupted compared to uninterrupted	−0.8	(-1.8, 0.1)	$p = .089$
	Final compared to baseline	0.1	(-0.1, 0.2)	$p = .310$
Right Heel	Constant	7.2	(2.8, 11.4)	$p < .001$
	Interrupted compared to uninterrupted	−0.0	(-0.7, 0.6)	$p = .913$
	Final compared to baseline	0.1	(-0.0, 0.2)	$p = .138$
Left Heel	Constant	7.0	(3.0, 11.4)	$p < .001$
	Interrupted compared to uninterrupted	−0.1	(-0.7, 0.5)	$p = .779$
	Final compared to baseline	0.1	(0.0, 0.2)	$p = .022$
Sacrum	Constant	22.3	(19.0, 25.6)	$p < .001$
	Interrupted compared to uninterrupted	−2.7	(-4.0, −1.4)	$p < .001$
	Final compared to baseline	0.9	(0.7, 1.1)	$p < .001$

Table 13
Differences between methods and timepoints for temperature – Supine (n = 41).

	Variable	Estimate	95% Confidence Interval	*p* value
Right Elbow	Constant	29.2	(19.8, 27.5)	$p < .001$
	Interrupted compared to uninterrupted	−0.3	(-0.4, −0.2)	$p < .001$
	Final compared to baseline	1.5	(1.4, 1.6)	$p < .001$
Left Elbow	Constant	29.4	(29.0, 29.7)	$p < .001$
	Interrupted compared to uninterrupted	−0.3	(-0.4, −0.1)	$p < .001$
	Final compared to baseline	0.4	(0.1, 0.3)	$p < .001$
Right Heel	Constant	28.2	(27.6, 28.8)	$p < .001$
	Interrupted compared to uninterrupted	−0.3	(-0.4, −0.2)	$p < .001$
	Final compared to baseline	0.3	(0.3, 0.3)	$p < .001$
Left Heel	Constant	28.2	(27.6, 28.8)	$p < .001$
	Interrupted compared to uninterrupted	−0.3	(-0.5, −0.2)	$p < .001$
	Final compared to baseline	0.3	(0.3, 0.3)	$p < .001$
Sacrum	Constant	31.7	(31.3, 32.0)	$p < .001$
	Interrupted compared to uninterrupted	−0.4	(-0.6, −0.3)	$p < .001$
	Final compared to baseline	0.4	(0.4, 0.5)	$p < .001$

3.2. Comparison of skin parameters across time and between method

The differences between the methods and timepoints for skin parameter appraisal are presented in Tables 10–13 (supine condition) and Tables 14–17 (semi-recumbent condition). On each occasion, the

Table 14
Differences between methods and timepoints for erythema – semi-recumbent (n = 41).

	Variable	Estimate	95% Confidence Interval	*p* value
Right Elbow	Constant	26.9	(30.7, 40.2)	$p < .001$
	Interrupted compared to uninterrupted	−2.2	(-3.5, −0.9)	$p < .001$
	Final compared to baseline	0.8	(0.6, 1.0)	$p < .001$
Left Elbow	Constant	27.1	(23.2, 31.0)	$p < .001$
	Interrupted compared to uninterrupted	−2.3	(-3.5, −1.1)	$p < .001$
	Final compared to baseline	0.9	(0.7, 1.1)	$p < .001$
Right Heel	Constant	29.1	(25.1, 33.1)	$p < .001$
	Interrupted compared to uninterrupted	−3.4	(-4.7, −2.2)	$p < .001$
	Final compared to baseline	0.8	(0.6, 1.0)	$p < .001$
Left Heel	Constant	31.0	(27.1, 34.9)	$p < .001$
	Interrupted compared to uninterrupted	−1.9	(-0.3, −0.7)	$p = .003$
	Final compared to baseline	0.3	(0.1, 0.5)	$p = .002$
Sacrum	Constant	35.4	(30.7, 40.2)	$p < .001$
	Interrupted compared to uninterrupted	−3.5	(-5.2, −1.8)	$p < .001$
	Final compared to baseline	1.4	(1.1, 1.7)	$p < .001$

Table 15
Differences between methods and timepoints for melanin – semi-recumbent (n = 41).

	Variable	Estimate	95% Confidence Interval	*p* value
Right Elbow	Constant	21.2	(12.4, 30.0)	$p < .001$
	Interrupted compared to uninterrupted	0.2	(-0.4, 0.9)	$p = .490$
	Final compared to baseline	0.0	(-0.1, 1.3)	$p = .607$
Left Elbow	Constant	21.3	(12.7, 30.0)	$p < .001$
	Interrupted compared to uninterrupted	−0.1	(-0.8, 0.7)	$p = .847$
	Final compared to baseline	0.1	(-0.0, 0.2)	$p = .108$
Right Heel	Constant	19.9	(11.7, 28.1)	$p < .001$
	Interrupted compared to uninterrupted	0.2	(-1.1, 1.4)	$p = .797$
	Final compared to baseline	−0.0	(-0.1, 0.1)	$p = .708$
Left Heel	Constant	20.5	(12.4, 28.6)	$p < .001$
	Interrupted compared to uninterrupted	−0.4	(-1.1, 0.3)	$p = .238$
	Final compared to baseline	0.1	(0.0, 0.2)	$p = .027$
Sacrum	Constant	26.4	(16.8, 36.1)	$p < .001$
	Interrupted compared to uninterrupted	−0.5	(-1.6, 0.5)	$p = .343$
	Final compared to baseline	0.1	(-0.0, 0.2)	$p = .070$

largest variation in skin parameter scores—when considering both methods—was found for the sacrum, with erythema and stratum corneum hydration scores observed to be most variable. In the supine condition, the mean of interrupted erythema measurements at the sacrum was 1.8 units lower than the mean of the uninterrupted measurements (95% CI -3.6, 0.1), while the mean of interrupted stratum corneum hydration measurements at the sacrum was 2.7 units lower than the mean of the uninterrupted measurements (95% CI -4.0, −1.4). Whilst stratum corneum hydration had slightly more variability

Table 16
Differences between methods and timepoints for epidermal hydration – semi-recumbent (n = 41).

	Variable	Estimate	95% Confidence Interval	*p* value
Right Elbow	Constant	8.8	(6.3, 11.2)	$p < .001$
	Interrupted compared to uninterrupted	−1.0	(-1.8, −0.2)	$p = .015$
	Final compared to baseline	0.1	(0.1, 0.4)	$p < .001$
Left Elbow	Constant	9.5	(7.0, 12.0)	$p < .001$
	Interrupted compared to uninterrupted	−1.5	(-2.2, −0.7)	$p < .001$
	Final compared to baseline	0.2	(0.1, 0.4)	$p < .001$
Right Heel	Constant	5.3	(0.4, 10.2)	$p < .001$
	Interrupted compared to uninterrupted	0.2	(-1.4, 1.9)	$p = .799$
	Final compared to baseline	0.1	(0.0, 0.2)	$p = .010$
Left Heel	Constant	5.1	(0.2, 10.0)	$p = .039$
	Interrupted compared to uninterrupted	0.6	(-1.1, 2.2)	$p = .498$
	Final compared to baseline	0.1	(0.0, 1.1)	$p = .037$
Sacrum	Constant	22.7	(18.9, 26.7)	$p < .001$
	Interrupted compared to uninterrupted	−1.7	(-2.9, −0.4)	$p < .001$
	Final compared to baseline	1.1	(0.9, 1.3)	$p < .001$

Table 17
Differences between methods and timepoints for temperature – semi-recumbent (n = 41).

	Variable	Estimate	95% Confidence Interval	*p* value
Right Elbow	Constant	24.5	(28.5, 29.5)	$p < .001$
	Interrupted compared to uninterrupted	−0.9	(-0.3, 0.1)	$p < .001$
	Final compared to baseline	0.3	(0.3, 0.4)	$p < .001$
Left Elbow	Constant	29.1	(28.7, 29.6)	$p < .001$
	Interrupted compared to uninterrupted	−0.1	(-0.4, 0.1)	$p < .001$
	Final compared to baseline	0.3	(0.3, 0.4)	$p < .001$
Right Heel	Constant	28.2	(27.6, 28.8)	$p < .001$
	Interrupted compared to uninterrupted	−0.3	(-0.4, −0.1)	$p < .001$
	Final compared to baseline	0.3	(0.3, 0.4)	$p < .001$
Left Heel	Constant	28.2	(27.7, 28.8)	$p < .001$
	Interrupted compared to uninterrupted	−0.3	(-0.4, −0.1)	$p < .001$
	Final compared to baseline	0.3	(0.3, 0.4)	$p < .001$
Sacrum	Constant	32.1	(31.8, 32.5)	$p < .001$
	Interrupted compared to uninterrupted	−0.6	(-0.8, −0.5)	$p < .001$
	Final compared to baseline	0.5	(0.4, 0.5)	$p < .001$

between methods than erythema in the supine condition, erythema was found to have more variability between methods in the semi-recumbent condition. To illustrate, in the semi-recumbent condition, the mean of the interrupted erythema measurements at the sacrum was 3.5 units lower than the mean of the uninterrupted measurements (95% CI -5.2, −1.8), while the mean of the interrupted stratum corneum hydration measurements at the sacrum was 1.7 units lower than the mean of the uninterrupted stratum corneum hydration measurements (95% CI -2.9, −0.4). However, these variations—when considered on a 0.99 scale—are minimal [72] and thus should not be considered as clinically significant. In relation to skin temperature assessment, the mean of interrupted measurements at the sacrum was 0.4 °C lower than the mean of the uninterrupted measurements (95% CI -0.6, −0.3) for the supine condition. In the semi-recumbent condition, the mean of the interrupted skin temperature measurements at the sacrum was 0.6 °C lower than the mean of the uninterrupted skin temperature measurements (95% CI -0.8, −0.5).

3.3. Intraclass correlation coefficient (ICC) for skin parameters

Of the 41 participants, there was a strong agreement in scores obtained from both the interrupted and uninterrupted methods, indicating minimal influence of the brief off-loading on skin parameters when adopting an interrupted measurement method. ICCs ranged from 0.72 to 0.99 (supine) and 0.62–0.99 (semi-recumbent) corresponding to a strong to near complete agreement [86] between methods. Note: ICC^1 is defined as test re-test reliability, and ICC^2 is defined as the agreement between method. Tables 18–21 and Tables 22–25 provide the intraclass correlations between skin parameter outcomes for the supine and semi-recumbent conditions, respectively.

3.3.1. ICC results for erythema

An ICC range of 0.83–0.92 (supine) and 0.83–0.90 (semi-recumbent) was noted for erythema across all anatomical testing sites (n = 41) translating to an excellent agreement [86] and was statistically significant between methods. In the supine condition, the lowest ICC^1 (test re-test reliability) and ICC^2 (agreement between method) values were both recorded at the sacrum at 0.87 (95% CI 0.79, 0.92) and 0.84 (95% CI 0.75, 0.90) respectively, while the highest ICC^1 and ICC^2 values were both recorded at the right heel at 0.92 (95% CI 0.88, 0.95) and 0.92 (95% CI 0.88, 0.95), respectively. For the semi-recumbent condition, the lowest ICC^1 and ICC^2 values were both recorded at the sacrum at 0.83 (95% CI 0.74, 0.89) and 0.83 (95% CI 0.74, 0.89) respectively, while the highest ICC^1 and ICC^2 values were both recorded at the left heel at 0.90 (95% CI 0.83, 0.94) and 0.89 (95% CI 0.82, 0.95), respectively.

3.3.2. ICC results for melanin

In both the supine and semi-recumbent condition, an ICC range of 0.99–0.99 (95% CI 0.99, 0.99) was noted for melanin across all anatomical testing sites (n = 41), corresponding to a near complete agreement [86] and was statistically significant between the methods.

3.3.3. ICC results for stratum corneum hydration

An ICC range of 0.81–0.98 (supine) and 0.88–0.99 (semi-recumbent)

Table 18
Intra-class correlations for erythema outcome measurements– Supine (n = 41).

Anatomical testing site	ICC^1 (95% CI)	ICC^2 (95% CI)
Right elbow	0.92 (0.87, 0.95)	0.90 (0.94, 0.95)
Left elbow	0.89 (0.82, 0.93)	0.88 (0.81, 0.93)
Right heel	0.92 (0.88, 0.95)	0.92 (0.88, 0.95)
Left heel	0.90 (0.83, 0.94)	0.89 (0.83, 0.93)
Sacrum	0.87 (0.79, 0.92)	0.84 (0.75, 0.90)

Note (see Method).
ICC^1 is defined as re-test reliability.
ICC^2 is defined as the agreement between methods.

Table 19
Intra-class correlations for melanin outcome measurements – Supine (n = 41).

Anatomical testing site	ICC^1 (95% CI)	ICC^2 (95% CI)
Right Elbow	0.99 (0.99, 0.99)	0.99 (0.99, 0.99)
Left Elbow	0.99 (0.99, 0.99)	0.99 (0.99, 0.99)
Right Heel	0.99 (0.99, 0.99)	0.99 (0.99, 0.99)
Left Heel	0.99 (0.99, 0.99)	0.99 (0.99, 0.99)
Sacrum	0.99 (0.99, 0.99)	0.99 (0.99, 0.99)

Table 20
Intra-class correlations for epidermal hydration outcome measurements – Supine (n = 41).

Anatomical testing site	ICC^1 (95% CI)	ICC^2 (95% CI)
Right Elbow	0.92 (0.87, 0.95)	0.92 (0.86, 0.95)
Left Elbow	0.92 (0.87, 0.95)	0.87 (0.79, 0.92)
Right Heel	0.98 (0.97, 0.99)	0.98 (0.96, 0.98)
Left Heel	0.99 (0.97, 0.99)	0.98 (0.97, 0.99)
Sacrum	0.84 (0.75, 0.90)	0.81 (0.70, 0.88)

Table 21
Intra-class correlations for temperature outcome measurements – Supine (n = 41).

Anatomical testing site	ICC^1 (95% CI)	ICC^2 (95% CI)
Right Elbow	0.86 (0.78.0.91)	0.86 (0.78.0.91)
Left Elbow	0.86 (0.79.0.91)	0.86 (0.79.0.91)
Right Heel	0.96 (0.93, 0.97)	0.96 (0.93, 0.98)
Left Heel	0.94 (0.90, 0.96)	0.94 (0.90, 0.96)
Sacrum	0.72 (0.60, 0.82)	0.72 (0.60, 0.82)

Table 22
Intra-class correlations for erythema outcome measures – Semi-recumbent (n = 41).

Anatomical testing site	ICC^1 (95% CI)	ICC^2 (95% CI)
Right Elbow	0.87 (0.80, 0.92)	0.87 (0.80, 0.92)
Left Elbow	0.89 (0.82, 0.92)	0.88 (0.80, 0.92)
Right Heel	0.86 (0.79, 0.92)	0.85 (0.76, 0.91)
Left Heel	0.90 (0.83, 0.94)	0.89 (0.82, 0.95)
Sacrum	0.83 (0.74, 0.89)	0.83 (0.74, 0.89)

Table 23
Intra-class correlations for melanin outcome measurements – Semi-recumbent (n = 41).

Anatomical testing site	ICC^1 (95% CI)	ICC^2 (95% CI)
Right Elbow	0.99 (0.99, 0.99)	0.99 (0.99, 0.99)
Left Elbow	0.99 (0.99, 0.99)	0.99 (0.99, 0.99)
Right Heel	0.99 (0.99, 0.99)	0.99 (0.99, 0.99)
Left Heel	0.99 (0.99, 0.99)	0.99 (0.99, 0.99)
Sacrum	0.99 (0.99, 0.99)	0.99 (0.99, 0.99)

Table 24
Intra-class correlations for epidermal hydration outcome measurements – Semi-recumbent (n = 41).

Anatomical testing site	ICC^1 (95% CI)	ICC^2 (95% CI)
Right Elbow	0.90 (0.88, 0.98)	0.89 (0.86, 0.95)
Left Elbow	0.90 (0.95, 0.98)	0.90 (0.91, 0.97)
Right Heel	0.98 (0.97, 0.99)	0.91 (0.91, 0.98)
Left Heel	0.99 (0.94, 0.99)	0.97 (0.95, 0.99)
Sacrum	0.88 (0.81, 0.92)	0.88 (0.81, 0.92)

Table 25
Intra-class correlations for temperature outcome measurements – Semi-recumbent (n = 41).

Anatomical testing site	ICC^1 (95% CI)	ICC^2 (95% CI)
Right Elbow	0.75 (0.78.0.88)	0.75 (0.70, 0.88)
Left Elbow	0.71 (0.71, 0.82)	0.71 (0.71, 0.82)
Right Heel	0.91 (0.88, 0.95)	0.91 (0.88, 0.95)
Left Heel	0.91 (0.87, 0.94)	0.91 (0.87, 0.94)
Sacrum	0.62 (0.60, 9.83)	0.62 (0.60, 0.83)

was noted for stratum corneum hydration across all anatomical testing sites (n = 41), corresponding to an excellent agreement [86] and was statistically significant between the methods. In the supine condition, the lowest ICC^1 and ICC^2 values were both recorded at the sacrum at 0.84 (95% CI 0.75, 0.90) and 0.81 (95% CI 0.70, 0.88) respectively, while the highest ICC^1 and ICC^2 values were both recorded at the left heel at 0.99 (95% CI 0.97, 0.99) and 0.98 (95% CI 0.97, 0.99), respectively. For the semi-recumbent condition, the lowest ICC^1 and ICC^2 values were both recorded at the sacrum at 0.88 (95% CI 0.81, 0.92) respectively, while the highest ICC^1 and ICC^2 values were both recorded at the left heel at 0.99 (95% CI 0.94, 0.99) and 0.97 (95% CI 0.95, 0.99), respectively.

3.3.4. ICC results for temperature

An ICC range of 0.72–0.96 (supine) and 0.62–0.91 (semi-recumbent) was noted for temperature across all anatomical testing sites (n = 41), translating to an excellent agreement [86] and was statistically significant between the methods. In the supine condition, the lowest ICC^1 and ICC^2 values were both recorded at the sacrum at 0.72 (95% CI 0.60, 0.82), respectively, while the highest ICC^1 and ICC^2 values were both recorded at the right heel at 0.96 (95% CI 0.93, 0.98), respectively. For the semi-recumbent condition, the lowest ICC^1 and ICC^2 values were both recorded at the sacrum at 0.62 (95% CI 0.60, 83) respectively, while the highest ICC^1 and ICC^2 values were recorded at the right heel at 0.91 (95% CI 0.88, 0.95) and at the left heel at 0.91 (95% CI 0.87.0.94), respectively.

4. Discussion

A pilot study was undertaken to examine changes in skin microclimate and skin colour in response to continual pressure-loading (immobilisation time 1 h) and during intermittent pressure relief. A convenience sample of 41 healthy adults aged 18–60 years was recruited. The main objective of this research was to determine if intermittent off-loading at pressure-prone areas impacted skin parameter results. To enable a comparison of results following brief consecutive off-loading, repeated measures were considered across an uninterrupted and interrupted method. In the uninterrupted method, measurements were taken at baseline and after 1 h, and 10 min apart in the interrupted method. Measurements were made using biophysical instruments by a trained examiner. Strong agreement emerged between data gathered across the uninterrupted and interrupted method, and scores obtained at end timepoints tended to be higher when compared to baseline for all skin parameter measures.

4.1. Distribution of skin parameters across pressure-prone areas

A large range of erythema scores (range: 2–71) and stratum corneum hydration scores (range: 0–66) was registered across all anatomical testing sites, implying large regional variations. When considered on a 0–99 scale, these results align with clinical interpretation of minimal to very high erythema [72] and very dry to over-hydrated skin [72]. There was a large range of melanin scores (range: 1–99) and a high level of variance which is strongly reflective of the multi-ethnic sample recruited. Because there was marginal change in melanin scores across all timepoints at all anatomical testing sites and amongst individual participants, future related studies may omit repeated measurements of melanin when assessing skin status.

A moderate range of skin temperature scores (range: 23.8–35.5 °C) was registered at all anatomical testing sites. Mean temperature scores at the elbows and heels ranged between 28.9–31.3 °C and 27.6–30.5 °C, respectively, while mean scores at the sacrum ranged between 30.8 and 34.3 °C. These findings can be translated as participants having greater variability and higher skin temperature at the sacral region. The mean baseline skin temperature scores at the elbows, heels and sacrum were recorded at 29.0 °C, 27.8 °C, and 31.0 °C respectively (supine), and at 29.2 °C, 27.8 °C and 31.1 °C respectively (semi-recumbent). These findings are consistent with those of Soares et al. (2019) [87] who sought to establish mean values for skin temperature at pressure-prone areas of the body in hospitalised patients at risk of PI development. The sacrum presented with the highest mean temperature of 34.2 ± 0.1 °C (range: 25.7–39.0 °C) while the elbows and heels presented with the lowest mean temperature of 31.2 ± 0.1 °C (range: 22.0–35.2 °C) and 28.2 ± 0.2 °C (range: 21.9–35.5 °C), respectively.

The largest variation between baseline and end scores of skin temperature was recorded at the sacrum; a difference of 2.6 °C for the supine condition, and 2.9 °C for the semi-recumbent condition. It is likely that an elevation in skin temperature at the sacral area region was caused by heat accumulation between at the skin support-surface interface [2,43,88] during periods of pressure-loading. It is probable that skin temperature would continue to rise if measurements were taken beyond 1 h [32]. Sae-Sia, Wipke-Tevis & Williams (2005) [34] in their study of hospitalised neurologically-impaired patients (n = 17) who were initially positioned supine (with pressure-loading on the sacral area) and then laterally (without pressure-loading at the sacral area) found that mean sacral temperature in patients who developed a PI was higher (p = .001) compared with those who did not. Sacral skin temperature was measured initially within 24–96 h of admission, and 48–72 h after the initial assessment. Patients who developed a PI were noted to have a higher sacral temperature (an increase of ~1.2 °C) at the 48–72 h re-assessment in both the supine (p = .001) and lateral positions (p = .002) compared with patients who did not develop PI. These findings are in agreement with those of Yusuf et al. (2015) [25] who evaluated skin microclimate and development of PIs and superficial skin changes in 71 patients on an acute care ward. They found that total mean difference in skin temperature was higher for patients with PIs and superficial skin changes (0.9 ± 0.6 °C) compared with controls (0.6 ± 0.8 °C) (p = 0·071) [25]. In a review by Lachenbruch (2005) [89], analysing findings from relevant animal [90,91] and human studies [92], it is recommended to limit sacral temperature, by means of cooling surfaces, to minimise the risk for sacral PIs. Overall, the results of the present study suggest that monitoring changes in sacral skin temperature can provide valuable clinical data that can be used by clinicians in their PI prevention efforts.

4.2. Differences in skin parameters between methods and timepoints

While no statistically significant difference emerged for melanin over time, significant differences ($p < .05$) were noted for erythema, stratum corneum hydration, and skin temperature between methods and timepoints. It is important to note that while these differences were statistically significant, the size of change is minor and suggests limited clinical relevance.

4.3. Comparison of results across evaluated lying conditions

The evaluated lying position had a negligible effect on skin parameters, with the endpoint results of the semi-recumbent condition only marginally higher compared to those of the supine condition. However, higher interface pressures have been reported in backrest elevation positions at the bony prominences. Barnett & Shelton (2008) [93] demonstrated that a backrest elevation of 30° in human subjects increased the loading of the pelvic region, producing elevated interface pressures in the sacral area. Additionally, Peterson et al. (1997) [94] *as cited in* Burk & Grap (2012) [95] found the lowest sacral pressures with backrest elevation of 0°, and significantly greater pressures with 30–75° backrest elevation in 14 healthy volunteers. In view of the above findings and the 1 h limit of pressure-loading in the current study, it is possible that compounding effects over time may exist and could be further investigated.

4.4. Relevance to clinical practice

It can be inferred from the current study data that brief off-loading of pressure-prone areas, over a period of 1 h, in healthy adults does not appear to impact skin parameter outcomes. Thus—when translating these findings into clinical practice—implementing an hourly off-loading regime for healthy patients lying continuously in the supine and semi-recumbent positions would not appear to clinically alter PI risk derived from the indirect effects of skin microclimate. However, considering that endpoint measures tended to be higher compared to baseline measures for all skin parameters, it is possible that beyond a 1 h period, a person may be approaching the threshold of skin changes that may place vulnerable skin at risk of PI development. Aside from this, there is scope to continue to explore the use of biophysical instruments in research with possible future applications in clinical practice, And to investigate the capacity of these instruments in providing an objective assessment of what has traditionally been a subjective assessment of skin microclimate and skin colour.

4.5. Limitations

A limitation of this study was a 1 h period to monitor for skin changes; this duration of appraisal was brief and, as such, this study cannot contribute to understanding longer-term changes. Future studies might consider a longer assessment duration. Additionally, participants were recruited from a university population; thus, results are specific to a predominantly younger active adult demographic, thereby restricting the ability to extrapolate skin changes to at risk populations such as the immobile elderly. Additionally, further research is required to determine when the level of erythema and stratum corneum hydration—when assessed at pressure-prone areas using biophysical instruments—should be deemed as clinically concerning. Finally, a correlation between skin parameter results and room temperature/relative humidity was not examined; future related studies should consider the influence of these variables on skin changes.

4.6. Conclusion

In general, this study has produced some important preliminary findings in a previously unaddressed area of the literature. The results provide valuable baseline data regarding the skin microclimate and skin colour of healthy persons as well as insights into skin variations during short periods of pressure-loading. This data may contribute to research investigating skin microclimate thresholds and how they ultimately relate to PI development. Findings indicate that brief off-loading of the elbows, heels, and sacral area at 10 min intervals to enable data collection did not impact results. Thus, researchers can employ this methodology to study skin changes (assessed repeatedly at 10 min increments over 1 h) without biased outcomes. Overall, the testing of the methods and procedures—specifically comparing skin results gathered during interrupted (brief off-loading) conditions and uninterrupted conditions—has provided the critical groundwork for development of a larger-scale investigation concerning biomechanical evaluation of skin manifestations in response to pressure and skin microclimate in healthy adults.

Author contributions

CB, CM and BM designed the experiments. CB completed data collection. CB, CM and DV analysed the data. CB, CM, BM and DV interpreted the data. CB drafted the manuscript. All authors edited and approved the final version.

Ethics statement

This research was conducted in full concordance with the ethical principles of the 1975 Declaration of Helsinki, and within Australian laws and regulations for Higher Research (National Statement on Ethical Conduct in Human Research, 2018). Institutional review and approval for this study (Ref. #HEC20300) was received from the Human Research Ethics Committee of the affiliated university.

Disclosures

All other authors have no disclosures.

Declaration of competing interest

The authors have no financial or other sources of conflict of interest relating to the submission of this manuscript pertaining to the study: Brief intermittent pressure off-loading on skin microclimate in healthy adults – a descriptive-correlational pilot study.

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5.3 Chapter Summary

This chapter incorporated one published work—a preliminary pilot study—that has provided context for the larger research. This published work discussed the aim of the first phase of this research (Studies I and II) and stipulated the background necessary to understand the scope of the second phase (Study III) of this research. The aim of Studies I and II was to generate data pertaining to whether intermittent pressure off-loading impacted skin parameters at the investigational anatomical sites over a one hour period of time. In doing so, Studies I and II explicitly addressed the following research questions:

i) How do erythema, melanin, stratum corneum hydration, and skin temperature measures when assessed using biophysical techniques at the skin overlying bony prominences change over time?

ii) Is there agreement between uninterrupted and interrupted methods when assessing erythema, melanin, stratum corneum hydration and skin temperature over time? That is, are the results from skin parameter measurement altered or relatively unchanged during brief off- loading of bony prominences?

The primary objective of Studies I and II was to assess for skin parameter differences between methods (interrupted and uninterrupted) and timepoints (baseline and final) to capture their associations with skin parameters. This, in turn, would enable consideration if momentarily shifting pressure to enable measurements (a method being considered for other research initiatives) altered skin parameter readings.

A descriptive-correlational design was employed to explore this aim. The results of the respective data analyses indicated that measured skin parameters in the study sample were not affected by intermittent pressure off-loading.

Overall, it can be inferred from the Studies I and II data that brief off-loading of pressure-prone areas, over a period of one hour, in healthy adults does not appear to impact skin parameter outcomes. Thus—when translating these findings into clinical practice—implementing a 10 minute off-loading regime for healthy patients lying continuously in the supine and semi-recumbent positions would not appear to clinically alter PI risk derived from the indirect effects of skin microclimate. However, considering that endpoint measures tended to be higher compared to baseline measures for all skin parameters, it is possible that beyond a one hour period, a person may be approaching the threshold of skin changes that may place vulnerable skin at risk of PI development.

5.4 Conclusion

In general, Studies I and II of this thesis have produced some important preliminary findings in a previously unaddressed area of the literature. The results provide valuable baseline data regarding skin colour and skin microclimate of healthy persons, as well as insights into skin parameter variations during short periods of pressure-loading. This data, in turn, may contribute to research investigating skin colour and skin microclimate thresholds, and how these thresholds ultimately relate to PI development. The findings of the first phase of this research indicate that brief off-loading of the elbows, heels and sacrum, at consecutive 10 minute intervals to enable data collection, did not impact results. Thus, researchers can employ this methodology to study skin changes (assessed repeatedly at 10 minute increments over one hour) without biased outcomes. Overall, the testing of the methodological aspects of this preliminary research phase has provided the critical groundwork for the design of a larger-scale investigation, which is addressed in the next chapter of this thesis. This next chapter describes the method for Study III in detail.

CHAPTER 6

Method – Study III

6.1 Chapter Overview

This chapter provides a comprehensive overview of the methods that were used to address the research aim, question, objectives and hypotheses of Study III of this thesis. The chapter begins by detailing ethical approval, followed by an overview of the research design together with the primary and secondary measures. The chapter then proceeds to outline the study eligibility criteria and recruitment procedures. The data collection instruments and their measurement principles are subsequently detailed. The chapter follows with an overview of the data collection procedures, and presents the statistical data analysis methods that were used to address the research aim, question and objectives of Study III. The chapter concludes by detailing the participant safety considerations applicable to this research.

6.2 Ethical Approval

This research was conducted in full conformance with the ethical principles of the 1975 Declaration of Helsinki, and within Australian laws and regulations for Higher Research (National Statement on Ethical Conduct in Human Research, 2018). Institutional review and approval for the conduct of Study III (reference no. HEC-20300) (Appendix B) was sought and received from the Human Research Ethics Committee of La Trobe University College of Science, Health and Engineering.

6.3 Research Design

Study III of this research used a quasi-experimental design with within-subject and between-condition comparisons across repeated measures. Study participants advanced consecutively, in a single visit, through three study conditions: condition 1: control, condition 2: heat intervention, and condition 3: moisture intervention. The terminology of 'condition' has been used in lieu of 'group' to accurately reflect the methodological concepts presented within this thesis, that is, all participants experienced all conditions rather than being allocated to a single condition and, in effect, acted as their own matched control. Advancement through the study conditions followed a non-randomised consecutive sequence. A standard 15 minute washout interval—for the cessation of residual effects from the previous condition—was used as a pragmatic interval between the control and heat

intervention, and between the heat and moisture intervention. A single researcher attended to and was responsible for all aspects of the relevant data collection processes.

The next section presents the aims, question, objectives and hypothesis pertaining to for Study III of this research.

6.4 Aims, Questions and Objectives

The aim of Study III was to examine the skin microclimate effects on clinical manifestations attributed to PI development in healthy adults. Specifically, Study III sought to assess for differences in erythema, stratum corneum hydration and skin temperature on exposure to heat and moisture at pressure-prone areas of the body of individuals lying in the semi-recumbent position, over time (one hour), with periodic pressure off-loading during the data collection.

Study III sought to answer the following research question:

1. How do erythema, stratum corneum hydration, and skin temperature measures—when assessed quantitatively at the skin overlying bony prominences of the body—change over time in response to local pressure and the controlled manipulation of skin microclimate at the skin-support surface interface?

In order to achieve the aim of Study III, the following objectives were adopted:

1. Establish a descriptive library of skin parameter scores—as measured over time and across pressure-prone areas—during intermittent pressure off-loading in the control, heat intervention, and moisture intervention;
2. Assess for differences in skin parameter scores, over time, and across pressure-prone areas between the control and heat intervention; and
3. Assess for differences in skin parameter scores, over time, and across pressure-prone areas between the control and moisture intervention.

Extending from the above research aims and objectives, a series of hypotheses were postulated for Study III. Study III was principally designed to test for differences in skin properties of heated versus unheated skin, and moist skin versus dry skin at the pressure-prone areas of healthy adults lying in the semi-recumbent position.

6.5 Research Hypotheses

It was hypothesised that in the heat intervention—when compared to the control—there would be significant increases in:

Erythema at the right and left elbow (Hypothesis One)

Erythema at the right and left heel (Hypothesis Two)

Erythema at the sacrum (Hypothesis Three)

Skin temperature at the right and left elbow (Hypothesis Five)

Skin temperature at the right and left heel (Hypothesis Six)

Skin temperature at the sacrum (Hypothesis Seven)

Null hypotheses were additionally stated. It was hypothesised that in the heat intervention—when compared to the control—there would be no significant increases in:

Stratum corneum hydration at the right and left elbow (Null Hypothesis Eight)

Stratum corneum at the right and left heel (Null Hypothesis Nine)

Stratum corneum hydration at the sacrum (Null Hypothesis Four)

It was hypothesised that in the moisture intervention—when compared to the control—there would be significant increases in:

Stratum corneum hydration at the sacrum (Hypothesis Ten)

Skin temperature at the sacrum (Hypothesis Eleven)

Null hypotheses were additionally stated in relation to the moisture intervention. It was hypothesised that in the moisture intervention—when compared to the control—there would be no significant increases in:

Erythema at the right and left elbow (Null Hypothesis Twelve)

Erythema at the right and left heel (Null Hypothesis Thirteen)

Erythema at the sacrum (Null Hypothesis Fourteen)

Stratum corneum hydration at the right and left elbow (Null Hypothesis Fifteen)

Stratum corneum hydration at the right and left heel (Null Hypothesis Sixteen)

Skin temperature at the left and right elbow (Null Hypothesis Seventeen)

Skin temperature at the right and left heel (Null Hypothesis Eighteen)

6.6 Primary and Secondary Measures

The primary measures in Study III included a series of skin parameters (erythema, stratum corneum hydration, and skin temperature) measured repeatedly, over time, at

pressure-prone areas of the body. Secondary measures included participants' demographic profile (age, gender, ethnicity) and health data (Fitzpatrick Skin Phototype Classification, baseline melanin measures, and body mass index (BMI)).

6.7 Study Eligibility

6.7.1 Inclusion Criteria

It was a condition of Study III eligibility criteria that healthy adults who were able to provide written informed consent comprised the study sample. To be eligible for the research, each person needed to be willing to participate and needed to satisfy the study inclusion criteria, as outlined below:

1. The person was able to provide written informed consent;
2. The person was a healthy adult over the age of 18 years; and
3. The person had intact skin at the elbows, heels and sacrum.

6.7.2 Exclusion Criteria

People were ineligible to participate if they met the below exclusion criteria:

1. The person was diagnosed with a cognitive impairment and/or disability that precluded their ability to provide informed consent;
2. The person had current wounds and/or dermatological pathology at the elbows, heels and/or sacrum;
3. The person had tattoos at the skin surface of the elbows, heels and/or sacrum; and
4. The person had current conditions/disorders that could affect their ability to lay stable for up to one hour at a time.

6.8 Study Recruitment

Recruitment for Study III commenced in November 2020 and concluded in June 2021. Participant recruitment was an ongoing process throughout the data collection period. In order to identify a healthy adult population, people were primarily recruited from the student population at La Trobe University and additionally via the researcher's personal networks. A minimum of 12 people and a maximum of 50 were sought. A power analysis was not completed for this study, given that a sample of 12 participants is usually sufficient for pilot investigations (Julious, 2005). Furthermore, a power analysis is not feasible in pilot studies in view of the unstable estimates of effect size (Moore et al., 2011).

6.8.1 Sampling Strategy

Convenience sampling was used to recruit eligible people from the target population. The primary means of identifying and inviting prospective participants to take part in this research was through paper-based and electronic study advertisements. These study advertisements were distributed by the researcher on La Trobe University Bundoora campus premises, primarily on bulletin boards in common study areas. Study advertisements were likely to be prospective participants' first time to hear about and/or be introduced to this research. Each advertisement included a brief statement of the study aim, eligibility criteria and participation requirements, allowing those interested to self-screen at their own convenience. This approach, thereby, reduced the number of people who did not meet the eligibility requirements contacting the researcher. This primary recruitment strategy was supplemented by electronic distribution of study advertisements through the Acting School Manager made to e-mails of HDR students at the School of Nursing and Midwifery, La Trobe University. Study participants were also recruited via personal networks such as the researcher's own social contacts. In all communications about the study, a contact telephone number for the researcher was provided to enable people interested to enquire about participating in the study.

6.8.2 Screening and Enrolment Process

When a person interested in participating made contact with the researcher, the person was asked to provide an e-mail address for the purpose of being sent a Participant Information Statement and Consent Form (PICF) (Appendix D) to read. As outlined in the PICF, if the person continued to convey an interest in participating, the person was asked to contact the researcher to complete a brief telephone screening checklist. The purpose of this telephone screening checklist was to ensure that the person was eligible to participate in the study. Screening calls were guided by a Telephone Screening Tool (Appendix E). An important component of this screening process was to identify characteristics that would preclude the person from inclusion in the study. Information regarding the person's age and skin status was relevant to study eligibility and, as such, was gathered during the initial telephone screening process. The researcher also used this time to reinforce to the person what study participation entailed. Once study eligibility for Study III had been established and inclusion criteria met, the person was sent an appointment vacancy schedule to nominate a preferred day and time for the study visit, and an appointment was booked.

It was explicitly requested that all eligible people avoid the topical application of products/preparations to the skin at the elbows, heels and sacrum for at least 24 hours prior to attending the study visit; a point emphasised in all preliminary study communications.

6.9 Instruments

6.9.1 The Norton Risk Assessment Scale (NRAS)

In this research, participants' risk of PI development was determined using the Norton Risk Assessment Scale (NRAS) (Norton, 1963) (Appendix G). The NRAS is a validated tool (Agency for Healthcare Research and Quality, 2015; Victorian Quality Council's Pressure Ulcer Point Prevalence Survey (PUPPS 3), 2006) which has applications within geriatric and acute care settings. The NRAS uses an ordinal scale to calculate an individual's predisposition to PI based on five separate items: physical condition, mental condition, activity, mobility, and incontinence. These items are then evaluated in terms of their severity; each item is assigned a numerical value ranging from a minimum value of 1 to a maximum value of 4 (4=best, 1=worst). The sum of the five separate items represents the total NRAS score, and indicates the level of PI risk; a score of 9 or less indicates significant risk; 10-13 high risk; 14-17 medium risk, and 18-20 low risk (Norton, 1962).

6.9.2 The Fitzpatrick Skin Phototype Classification

The Fitzpatrick Skin Phototype Classification (Fitzpatrick, 1988) (Appendix H) (abbreviated to Fitzpatrick Skin Type) was used in this research to guide the assessment of participants' skin type. The Fitzpatrick Skin Type is a recognised tool for dermatological research in human skin pigmentation (Taylor et al., 2006; Taylor et al., 2009). The scale uses a numerical classification schema which considers the amount of melanin pigment in the skin; this schema is determined by constitutional skin colour (white, brown, or black) and the effect of exposure to ultraviolet radiation (or tanning) (van Der Wal et al., 2013). The Fitzpatrick Skin Phototype scale also includes three grading items: genetic (physical traits), sensitivity (reaction to sun exposure), and intentional exposure (tanning habits). These items are further divided into sub-categories, where each item is ranked on an ordinal scale ranging from 0-4. The sub-category scores are then added together to generate an overall score; scores between 0-6 correspond to Type I: Pale white skin, 7-13 to Type II: Fair skin, 14-20 to Type III: Fair to beige skin, 21-27 to Type IV: Light brown skin, 28-34 to Type V: Brown skin, and 35-36 to Type VI: Dark brown to black skin.

6.9.3 The SD202 Skin Diagnostic (Courage+Khazaka Electronic GmbH)

In this research, a series of parametric skin measurements was undertaken on people eligible for data collection. Measurements were gathered using the SD202 Skin Diagnostic (Courage+Khazaka Electronic GmbH, Köln Germany) (Figure 6.1). The SD202 Skin Diagnostic is a portable battery-powered device, which combines the sensor technology of the Corneometer and Mexameter that measure stratum corneum hydration and skin colour, respectively. The SD202 Skin Diagnostic is used in Australia and overseas as a cosmetically and dermatologically relevant skin analysis aid used in the quantitative evaluation and monitoring of clients' skin health (Enviroderm, 2019).

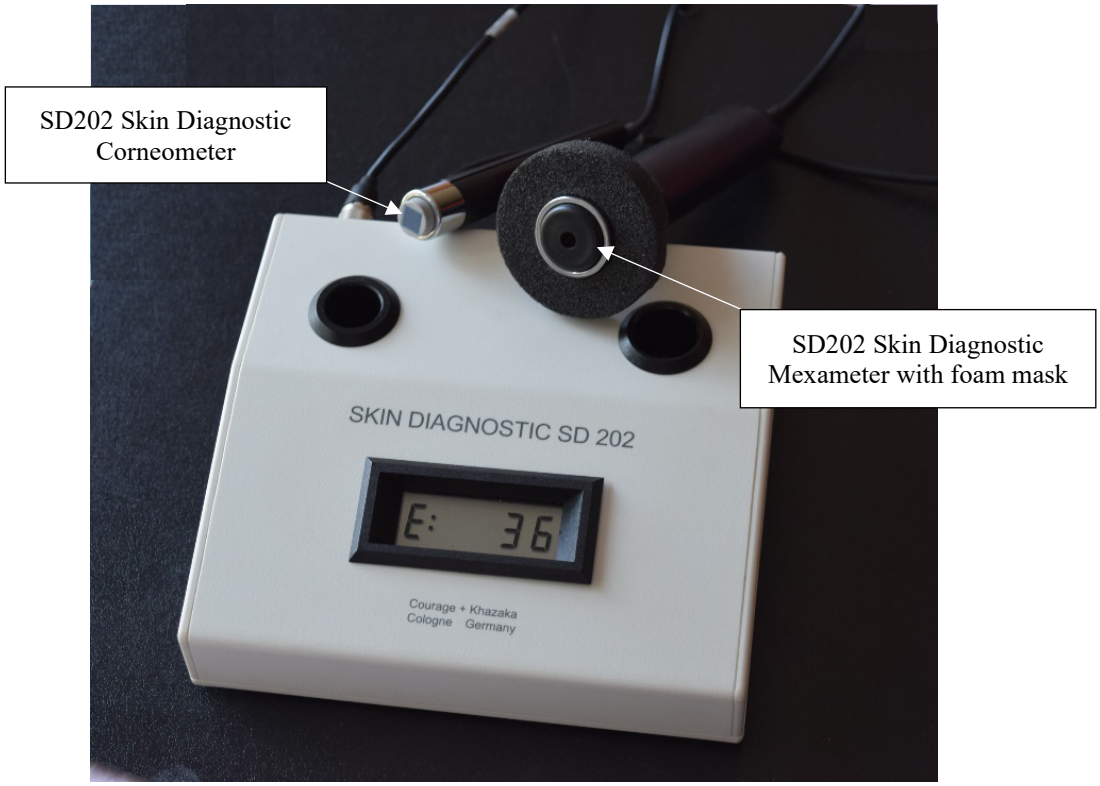

Figure 6.1 The SD202 Skin Diagnostic (Courage+Khazaka Electronic GmbH). Photo Credit: C. Borzdynski 2019

The reliability of the SD202 Skin Diagnostic was previously investigated in three independent studies (Borzdysnki, McGuiness & Miller, 2016; Huimin et al., 2017; Rowledge et al., 2016). Cronbach's α was obtained as a reflection of the SD202 Skin Diagnostic's internal consistency, that is, the consistency between single-site consecutive measurements attended to by the researchers. The reliability of the SD202 Skin Diagnostic was found to be exceptionally high with respect to its assessment of stratum corneum hydration and colour at periwound skin of venous leg ulcers (VLUs) (Huimin et al., 2017) and diabetic foot ulcers

(DFUs) (Rowledge, et al., 2016), as well as intact skin at pressure-prone areas of the body (Borzdynski, McGuiness & Miller, 2016).

6.9.4 The SD202 Skin Diagnostic Corneometer

In this research, stratum corneum hydration levels were measured using the SD202 Skin Diagnostic Corneometer (Courage+Khazaka Electronic GmbH, Köln Germany). The SD202 Skin Diagnostic Corneometer uses the high dielectric constant of water for analysing the water-related changes in the electric capacitance of the skin in the outermost layer (10-20 micrometres (μm) of the epidermis). When the device sensor is lightly depressed onto the skin surface and held in place for two seconds, a digital reading of stratum corneum hydration is automatically generated and appears on the device screen.

The SD202 Skin Diagnostic Corneometer measurements have been reported to be highly reliable (Borzdynski et al., 2016; Huimin et al., 2017; Rowledge et al., 2017). Readings are provided in system-specific arbitrary units, ranging from 0 to 99 (Enviroderm, 2019). The greater the water content of the skin, the greater the measuring capacity of the device; that is, a higher level of stratum corneum hydration reflects higher dielectric constants. As per the SD202 Skin Diagnostic Measurement Interpretation Guide (Barrett-Hill, 2015) (Figure 6.2), stratum corneum hydration readings between 0-23 correspond to a very dry stratum corneum, 24-45 to a dry stratum corneum, 46-64 to optimally balanced stratum corneum hydration, 65-82 to a higher level of stratum corneum hydration, and 83-99 to over-hydration of the stratum corneum (Barrett-Hill, 2015).

6.9.5 The SD202 Skin Diagnostic Mexameter

In this research, erythema (vascularity) and melanin were measured using the SD202 Skin Diagnostic Mexameter (Courage+Khazaka Electronic GmbH, Köln Germany). The SD202 Skin Diagnostic Mexameter quantifies melanin and erythema via a single probe with a precision sensor[9]. The probe has an alternate action when taking measurements. To illustrate, when the sensor is initially depressed onto the skin surface, it provides a melanin reading, and when the pressure is released before again being depressed onto the skin surface, the device registers erythema. The erythema index is calculated (range: 1-99) in

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[9] The SD202 Skin Diagnostic is supplied with a foam light mask for the Mexameter to reduce the amount of ambient light that falls onto the skin surface being measured. To avoid inaccurate readings, the measured area should always be under the shadow of the mask; this ensures that the precision sensor within the probe only reads the light which the light emitting diodes in the probe head project onto skin surface.

arbitrary units, of a circular $5mm^2$ area from the quantity of the reflected light when emitting light at wavelengths of 568 and 660nm (Kleesz, Darlenski & Fluhr, 2012). Readings of 1-18 indicate an absence of erythema, 19-36 minimal erythema, 37-54 diffused redness, 55-80 high erythema, and 80-99 extremely erythematous skin.

A melanin index is established through measurement of the amount of absorbed and reflected red and infrared light; a greater absorption of light at these wavelengths is indicative of increased melanin (Courage+Khazaka Electronic, 2019). Melanin readings correlate with the darkness of the skin as per Fitzpatrick Skin Type (Fitzpatrick, 1988); melanin readings of 1-8 are correlated with pale white skin (Fitzpatrick Type I), 9-13 fair skin (Fitzpatrick Type II), 14-21 fair to beige skin (Fitzpatrick Type III), 22-30 light brown skin (Fitzpatrick Type IV), 31-37 brown skin (Fitzpatrick Type V), and 38-99 dark brown to black skin (Fitzpatrick Type VI).

The reliability and validity of the SD202 Skin Diagnostic Mexameter has been explored in previous related studies, and was found to be exceptionally high with respect to the assessment of erythema at the peri-wound skin of venous leg ulcers (Huimin et al., 2017) and diabetic foot ulcers (Rowledge, et al., 2016), as well as intact skin at pressure-prone areas of the body (Borzdynski, McGuiness & Miller, 2016).

SD202 measure	Range	Reading interpretation
Stratum Corneum Hydration	0 – 23	Very dry
	24 – 45	Dry
	46 – 64	Optimal balanced zone
	65 – 82	Higher level of hydration
	83 – 99	Over-hydrated
Erythema	1 – 18	No erythema
	19 – 36	Minimal erythema
	37 – 54	Diffused redness
	55 – 80	High erythema
	80 – 99	Extreme erythema
Melanin	1 – 8	Pale white skin
	9 – 13	Fair skin
	14 – 21	Fair to beige skin
	22 – 30	Light brown skin
	31 – 37	Brown skin
	38 – 99	Dark brown to black skin

Figure 6.2 SD202 Skin Diagnostic Reading Interpretation Guide. Reprinted from "Pastiche Virtual Beauty Corporation: The Visual Skin Analysis Diagnostic Indicator Guide", Barrett-Hill, 2015 (http://www.virtualbeauty.co.nz/new/textbooks/visual-skin-analysis-diagnostic-indicator-guide/)

6.9.6 The Exergen DermaTemp DT-1001 RS Infrared Dermal Scanner

In this research, the Exergen DermaTemp DT-1001 RS Infrared Dermal Scanner (abbreviated to Exergen DermaTemp) (Exergen Corporation, Watertown, Massachusetts, USA) (Figure 6.3) was used to measure participants' skin temperature in degrees Celsius (°C). The Exergen DermaTemp is a high-precision device with a reported clinical accuracy of ±0.1°C. The device measures the heat naturally emitting from the body when held ~5mm from the skin surface, and is capable of detecting subtle skin temperature variations that may be caused, for example, by underlying perfusion variations (Exergen Corporation, 2019).

The Exergen DermaTemp's patented automatic emissivity[10] compensation system allows the detection of heat radiation emitted from the skin surface while avoiding error due to reflected energy from other sources. The cuplike mirror in the stainless steel probe head is made of germanium (an infrared-transmitting material) which 'traps' radiation emitted from a target site on the skin. This radiation is then transferred through an optical system, and converted to an electrical signal that can be read on the instrument's LCD display.

The Exergen DermaTemp has applications in both clinical settings (specifically plastic and vascular surgery) and in research (Exergen Corporation, Watertown, MA, 2019). Burnham et al. (2006) investigated the reliability and validity of the Exergen DermaTemp DT-1001 LN (Exergen Corporation, Watertown, MA, 2019) in its ability to measure skin temperature amongst healthy Caucasian volunteers (n=17) with a mean age of 29.5 years (*SD*=8.5). Skin temperature was recorded at the hand (dorsal surface), forearm (over the common extensor muscle belly), shoulder (over the lateral deltoid), foot (dorsal aspect), thigh (over the rectus femoris), and shin (over the tibialis anterior). Test-retest reliability was calculated using intraclass correlation coefficient (ICC). Pearson correlation assessed the relationship between the skin temperatures at the anatomical regions tested. The Exergen DermaTemp DT-1001 LN was found to be highly reliable. The test-retest reliability of the Exergen DermaTemp DT-1001 LN, as reported in Burnham et al.'s (2006) study, is concordant with a recent study (Mufti, Coutts & Sibbaled, 2015) that documented comparable results using the same DermaTemp model.

While there is no formal interpretation guide for skin temperature values published by the Exergen DermaTemp manufacturer, mean skin temperature values are often reported in clinical thermoregulation studies (Millington, 1983; Montalto et al., 2013). Assuming neutral (21-25°C) ambient temperatures and a core temperature of ~37.0°C, the temperature of human skin tends to vary between 33.5-36.9°C at the trunk, and between 23.5-35.6°C at the extremities. Skin temperature is considerably lower than core body temperature which tends to vary between 36.5-37.2°C (Burgess et al., 1978). Forehead skin temperature, for

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[10] Emissivity is a surface property which determines how well an object's temperature can be measured by an infrared device. Thus, emissivity can be considered as the measure of an object's ability to emit infrared energy. Emitted energy can have a value from 0 (shiny mirror) to 1.0 (black body) (Goldberg, 2003). In actuality, all real bodies (including human ones) have an emissivity between these two limits. Human skin is near but not equal to 1.0 and, if not accounted for, can introduce errors in the order of one to two degrees. The mirror used in the measuring sensor of the Exergen DermaTemp infrared scanner removes this source of error by trapping all of the radiation from the skin surface and, in effect, causes the skin surface to act like a black body with an emissivity of 1.0 (Exergen Cooperation, 2019).

example, is typically 2.0°C cooler than core temperature (Millington, 1983). Furthermore, unlike core body temperature, which is tightly-regulated, the temperature at the surface of the skin varies markedly as a function of environmental exposure (Sessler, 2008). Additionally, the temperature of the skin can change at a rapid rate in response to internal (physiological) influences such as unequal vascular supply and perfusion (Sessler, 2008) associated, for example, with peripheral vascular disease (Mufti, Coutts & Sibbald, 2015).

Figure 6.3 The DermaTemp DT1001-RS Infrared Thermographic Scanner. Photo Credit: C. Borzdynski 2019

This next section outlines the data collection procedures guided by the respective aims and objectives of Study III.

6.10 Procedure

Once the results for Studies I and II of this research were collated, processed and analysed, a protocol for Study III was established for which relevant institutional review and approval was sought (reference no. HEC-20300) (Appendix B). In Study III, the research team progressed with the data collection schedule as per the interrupted method initially employed in Studies I and II. Negligible variation in skin parameter measurements between the supine and semi-recumbent positions in Studies I and II (as was determined through statistical analyses) led the research team to consolidate its focus on the semi-recumbent position only in Study III. Finally, the absence of any measurable difference in melanin scores in the Studies I and II participant sample led to the omission of the melanin

component in Study III; however, melanin measures were attended to at the anatomical testing sites during baseline screening.

6.10.1 Study Setting

All data collection procedures relevant to Study III took place at La Trobe University, Bundoora campus, in the same nursing laboratory teaching-room designated for the conduct of Studies I and II. Prior to the participant's study visit, an analogue thermometer and hygrometer were used to gauge the ambient temperature and relative humidity within the laboratory room. Average room temperature was recorded at 22.9˚C (range: 20.6-25.8˚C) while relative humidity was recorded at 55% (range: 52-61%).

6.10.2 Participant Screening and Demographic Health Information

Participants attended a single study visit requiring approximately four hours of their time. At the commencement of the visit, the researcher reaffirmed the study's eligibility criteria to the person before offering enrolment. If the person met the inclusion criteria and agreed to participate in the study, a consent form was provided and signed, and the person was enrolled in the study. Participants were informed that they had the right to withdraw their consent and discontinue their participation at any time without giving any reason by signing a Withdrawal of Consent for use of Study Data Form (Appendix C). Upon provision of consent, each participant was assigned a unique participant identification number (PIN) to re-identify them for confidentiality purposes. The PIN was recorded in a log-book which listed each participant's title and full name. This log-book was stored separately to all other study data in secure storage as per the La Trobe University Study Data Management Plan.

Information regarding the participant's age, gender, ethnicity, body mass index (BMI)[11], Fitzpatrick Skin Type, baseline melanin measures and Norton Risk Assessment Scale (NRAS) score was gathered using the Participant Demographic and Health Data Form (Appendix J). If applicable, information regarding participants' history of cutaneous conditions was gathered in an open-ended response format.

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[11] Height and weight were measured using a stadiometer and digital scales, and BMI was calculated as weight (kg)/height (m^2). Participants were classed as *normal weight*, *underweight*, *overweight*, or *obese* based on internationally recognised cut-offs (BMI 18.5-24.9, <18.5, 25-29.9 or >30 kg/m^2, respectively) (WHO, 2001).

6.10.3 Standardisation Procedures and Participant Positioning

Participants were provided with a hospital-type back-tie exam gown (65% polyester 35% cotton) to apply over bare skin within a private space of the laboratory room. Next, the participant was instructed to lie on a hospital-grade mattress composed of high-density polyurethane foam (30kg/m^3 and 120mm in thickness) lined with a plastic nylon cover (Figure 6.4) and fitted with a cotton polyester bottom-sheet. This mattress is supported on a standard hospital-type electronic bed (915x1900) with a remote-operated crank that allows for adjusting the bedhead to a maximum 90° angle. The remote-operated controller allowed for adjusting the bedhead to a 45° angle in accordance with the Study III protocol. The angle of the bedhead was confirmed with a combination square tool.

As per the Study III protocol, the skin at the elbows, heels and sacrum needed to be barred at the support surface during periods of lying down. Care was also taken to ensure that the hospital gown was not underneath the participant's sacral area. A top-sheet and blanket covered the participant and were folded at the upper chest, however, the arms were exposed and extended along the midline of the body in the horizontal plane.

Figure 6.4 Laboratory bed with mattress used in this research. Note, the linen were removed for clarity. Photo Credit: C. Borzdynski 2019

6.10.4 Arrangement of Study Conditions

In Study III, all participants advanced consecutively in a single visit through three study conditions: condition 1: control, condition 2: heat intervention, and condition 3: moisture intervention. Advancement through the study conditions followed a non-randomised consecutive sequence. A standard 15 minute washout interval for the cessation of residual effects from the previous condition was used as a pragmatic interval between the control and heat intervention, and between the heat and moisture intervention conditions. Data gathered in the control served as a baseline against which the effects of the study intervention conditions were later evaluated, placing the results into context.

It should be noted that the heat intervention intentionally preceded the moisture intervention in view of the anticipated duration time that would be required for the re-establishment of participants' baseline skin condition. An incomplete return to baseline was anticipated in the case where the washout interval would proceed the moisture intervention. In this instance, any measured changes in skin properties at the investigational sites thereafter may have been attributable to carryover effects. In contrast, near complete return to baseline was anticipated in the instance where the washout interval would proceed the heat intervention condition, with consideration to the effect of heat dissipation upon the skin's exposure to the ambient environment (Brienza & Geyer 2005).

6.10.5 Data Collection Timepoints

In each study condition (control, heat intervention, and moisture intervention), skin parameter measurements were recorded at baseline (*T0*), interim 10 minute intervals (*T10, T20, T30, T40, T50*) and endpoint at 60 minutes *(T60*). These timepoints were selected following a pilot study (Borzdynski, Miller, Vicendese & McGuiness, 2021) that identified that brief off-loading between two consecutive intervals of 10 minutes over the course of one hour would not impact skin parameter results by virtue of data collection. The measurement intervals for each condition were monitored with the use of an electronic timer with audible beeping functionality. The data collection timepoints for skin parameter appraisal in Study III are summarised in Table 6.1.

In this study, a total loading period of one hour was used as an initial excursion to ensure participant safety in line with the *Prevention and Treatment of Pressure Ulcers Clinical Practice Guidelines* (EPUAP-NPUAP-PPPIA, 2019).

Table 6.1

Data collection timepoints for skin parameter appraisal (Study III)

Condition	Baseline measures *T0*	Interim measures *T10, T20, T30, T40, T50*	End measures *T60*
Control	☑	☑	☑
		Washout interval – *T5*, *T10*, *T15*	
Heat intervention	☑	☑	☑
		Washout interval – *T5*, *T10*, *T15*	
Moisture intervention	☑	☑	☑

T=time

6.10.6 Anatomical Testing Sites

Measurements of skin temperature, stratum corneum hydration and erythema were registered at the skin surface overlying the bony prominences of the sacrum (specifically the *medial sacral crest*), elbows (anatomically, *olecranon*) and heels (anatomically, *calcanei*), with measurements taken bilaterally at the elbows and heels. These bony prominences were selected as the anatomical testing sites as they represent common PI predilection areas in non-ambulatory individuals lying supine/semi-recumbent. As outlined previously in Chapter 2 of this thesis, PIs typically develop when soft tissue is compressed between a weight-bearing bony prominence and an external surface for an extended period of time.

The vulnerability of the skin at a bony prominence to PI formation is dependent on the individual's position (Larson et al., 2012). The sacral, elbow and heel bony prominences are the most weight-bearing bony prominences in supine/semi-recumbent positions. The tissues adjacent to these bony prominences are highly vulnerable to the effects of interface pressure and, therefore, termed 'pressure-prone areas' (Al Aboud & Manna, 2021). It has been widely-documented (Burk & Grap, 2018; Jaul et al., 2018; Santamaria et al., 2005; Schlüer et al., 2009; Wong & Stotts, 2003) that mechanical loads, in the form of internal

bodyweight forces, are typically transferred through the bony structures of the sacrum, elbow and heel into overlying soft tissue when an individual rests supine or semi-recumbent.

6.10.7 Procedure for Baseline Skin Parameter Appraisal

Immediately after the participant adjusted into a semi-recumbent position on the designated laboratory bed, baseline (*T0*) skin parameter measurement was attended at each of the anatomical testing sites. Measurements were registered by a single researcher using the designated biophysical instruments. The Exergen DermaTemp, SD202 Skin Diagnostic Corneometer and Mexameter measurement probes were consecutively applied to the skin surface of each anatomical testing site. At each data collection timepoint, a single measure of skin temperature, stratum corneum hydration and erythema was taken—in a 1cm radius area—at each target site. Readings were obtained within two seconds of applying the measurement probe to the skin.

Skin temperature preceded all other parameters with consideration to heat dissipation (Brienza & Geyer 2005; Petrofsky & Laymon, 2009) on exposure of the skin to the ambient environment. The elbows and heels were assessed prior to the sacrum given that attending to measures at the elbows and heels could be achieved without altering the position of the respective anatomical testing sites. First, the right elbow was assessed, with the participant instructed to briefly raise and flex their arm so that the elbow formed a 90° angle. A single reading of each skin parameter was taken and recorded. This measurement process was repeated for the left elbow. Next, the right heel was assessed, with the bedlinen at the end of the bed partially folded back to expose the right lower foot. The participant was instructed to briefly lift their right leg to an angle of between 15-20° with the foot dorsiflexed. After the right leg was lowered back onto the bed surface, the same measurement process was repeated for the left heel. To enable measurement at the sacrum, the participant was asked to briefly reposition onto their side. Once sacral measurements were attended to, the participant repositioned back into a semi-recumbent position. This processes of skin parameter appraisal was repeated in the same sequence at 10 minute intervals for the one hour period across all three study conditions. These three conditions—the control, heat intervention and moisture intervention—are described below.

6.10.7.1 Condition 1: Control

In the control, the participant lay semi-reclined on the designated laboratory bed for one hour, with skin parameter measurements attended to at interim timepoints (*T10, T20,*

T30, T40, T50) and endpoint *(T60*). Skin parameter appraisal in the control was conducted as per the procedure for baseline skin parameter appraisal described in Section 6.10.7.

A 15 minute washout interval applied after endpoint measurements of the control. Immediately following endpoint measurements, the participant was instructed to stand up from the bed, stretch, and walk at a self-selected comfortable walking pace back and forth along an 8 metre walkway in the laboratory space. After five minutes, the participant stood in a comfortable weight-bearing position while skin parameters were re-assessed at the sacrum, elbows and heels. To enable heel measures, the participant was asked to briefly flex their knee to a 90° angle. For the remainder of the washout interval, skin parameter measures were re-assessed at five minute intervals and monitored for their return to baseline.

The endpoint results of the first washout interval served as baseline results of the proceeding intervention. Immediately prior to the researcher attending to endpoint measures of the washout interval, the participant was instructed to resume lying semi-recumbent on the designated laboratory bed to prevent measurement accuracy being impacted by the angle of the skin measuring probe. The procedure for the heat intervention is described below.

6.10.7.2 Condition 2: Heat Intervention

In the heat intervention, the participant lay semi-reclined on the designated laboratory bed for one hour, with skin parameter measurements attended to at interim timepoints (*T10, T20, T30, T40, T50*) and endpoint (*T60*). However, this time, the participant lay on an electric heated blanket (160x130cm) (Ramesses® Electric Heated Blanket, Kingtex Inc). This heated blanket was pre-warmed 15 minutes prior to the participant lying down on the bed, with the temperature held constant thereafter at the desired heat-generating level of 30°C. To ensure a uniform distribution of heat at the posterior aspect of the participant's body, the heated blanket was spread across the surface of the bed. For hygiene purposes, a top-sheet was applied over the surface area of the heated blanket. Skin parameter appraisal at each timepoint of the heat intervention was repeated in an identical manner as for the procedure for baseline skin parameter appraisal described in Section 6.10.7.

A 15 minute washout interval applied following endpoint measurements in the heat intervention. The procedure for this washout period was duplicated as per the first washout interval. The endpoint results of the second washout interval served as baseline for the proceeding moisture intervention. Immediately prior to the researcher attending to endpoint

measures of the washout interval, the participant was instructed to resume lying semi-recumbent on the designated laboratory bed to prevent measurement accuracy being impacted by the angle of the device sensor. The procedure for the moisture intervention is described below.

6.10.7.3 Condition 3: Moisture Intervention

In the moisture intervention, in order to avoid any carryover effects in the form of residual heat remaining on the bed surface from the prior heat intervention, the participant was instructed to lay semi-recumbent on a neighbouring identical laboratory bed. The participant lay semi-reclined on the bed for one hour, with skin parameter measurement attended to at interim (*T10, T20, T30, T40, T50*) and endpoints (*T60*). However, this time, a skin wetting agent (moisture stimulus) in the form of an aqueous solution of 0.9% normal saline[12] (Baxter International Inc) was introduced at the sacral skin surface. The purpose of the skin wetting agent was to simulate an altered microclimate in the form of extreme moisture/liquid accumulation at the skin-support surface interface. Immediately prior to the participant lying down, the researcher poured 120ml of the normal saline solution onto the centre of a disposable absorbency pad (Halyard Clinical®) (approx. 40x55cm) with polyethylene backing. The participant was then positioned semi-recumbent on the bed with their lower back positioned directly over the soaked absorbency pad. Skin parameter appraisal at each timepoint of the moisture intervention was repeated in the same manner as per the procedure for baseline appraisal described in Section 6.10.7.

Upon completion of the moisture intervention, the participant was provided with a clean towel to remove any residual moisture from the skin, and was provided with privacy to re-dress. All participants received a small monetary incentive for their participation in the study as approved by the La Trobe University Human Research Ethics Committee.

The SD202 Skin Diagnostic and Exergen DermaTemp device were carefully wiped with a hospital-grade anti-bacterial wipes (ISOWIPE® 70% isopropyl alcohol) in-between participant use. All the biophysical instruments used in this research were stored in a locked cupboard when not in use.

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[12] Normal saline is an isotonic concentration of sodium chloride. Within each 100 ml of 0.9% sodium chloride, there are 154 mEq of sodium ions and 154 mEq of chloride ions. This means that normal saline is prepared by measuring out 0.9 g of sodium chloride and diluting this amount of sodium chloride to a final volume of 100 ml in water. The osmolarity of normal saline is 308 mOsmol/ and has a pH range of 4.5-7 (Chang & Holcomb, 2016).

Results for skin parameter appraisal in Study III were recorded onto a paper-based data collection form – Participant Data Collection Form for Repeated Skin Parameter Measurements (Study III) (Appendix M). This data collection form was identifiable only with the participant's unique identification number.

6.11 Statistical Procedures and Data Analysis

In order to address the research objectives of Study III, a series of descriptive and inferential statistical approaches were employed.

First, descriptive statistics were used to:

1. Provide the measures of central tendency (means, medians and modes) and variability (range, IQR, variance and SD) for erythema, stratum corneum hydration and skin temperature scores at baseline, interim (every 10 minute) and endpoints of control and intervention conditions; addressing objective 1 of Study III: To establish a descriptive library of skin parameter scores—as measured over time and across pressure-prone areas—during intermittent pressure off-loading in a control, heat intervention, and moisture intervention.

Second, a series of inferential statistical approaches were used to:

1. Test for differences by timepoint (that is, to test for differences in skin parameter scores simultaneously to each timepoint) and differences by condition (that is, to test for differences in skin measurements between heated versus unheated skin) at the elbows, heels and sacrum; addressing objective 2 of Study III: To assess for differences in skin parameter scores, over time, and across pressure-prone areas between the control and heat intervention; and
2. Test for differences by timepoint (that is, to test for differences in skin parameter scores simultaneously to each timepoint) and differences by condition (that is, to test for differences in skin measurements between wet versus dry skin) at the elbows, heels and sacrum; addressing objective 2 of Study III: To assess for differences in skin parameter scores, over time, and across pressure-prone areas between the control and moisture intervention.

6.11.1 Data Screening and Preparation

Data collection for Study III occurred between November 2020 and June 2021 following institutional review and approval from the Human Research Ethics Committee of

La Trobe University (reference no. HEC-20300) (Appendix B). Data were entered into the IBM Statistical Package for the Social Sciences (SPSS) analysis software (SPSS Version 25.0; IBM Corp., 2017). Once data entry was finalised, data were cleaned and checked for out-of-range values. Assumptions of normality, linearity and homoscedasticity were initially examined prior to proceeding with statistical data analyses. There was no evidence of violation of these assumptions or influential outliers. To confirm that there were no major violations of normality, values of skewness and kurtosis were reviewed. An examination of absolute skewness and kurtosis revealed that all model variables fell within skewness $\leq \pm 2.0$ and kurtosis $\leq \pm 7.0$ (West, Finch & Curran, 1995) suggesting univariate normality. Additionally, an inspection of histograms for continuous variables suggested that these data did not violate distributional assumptions. Using descriptive statistics, the study sample characteristics were described, including the frequencies, means, SDs, medians, and interquartile ranges (IQRs). Data were not missing at any timepoint. No participant cases or data required exclusion.

Baseline demographics (age, gender, ethnicity, BMI) and skin profile data (Fitzpatrick Skin Type and baseline melanin scores) were examined using bivariate correlations for their association with skin measurements. Pearson's product-moment correlation coefficient (r) was used to measure the strength of linear correlations between variables. As per Cohen's criteria, a correlation is considered 'weak' if the value of r varies between .1–.29, 'moderate' if the value of r varies between .3–.49 and 'strong' if r is equal to or exceeds .5. In Study III, any variable with an r value of $\geq .3$ was considered to be a covariate and, as such, would be incorporated into further analyses (Cohen, 1988). Spearman's Rho was used to assess the associations between ordinal variables (BMI, Fitzpatrick Skin Type and ethnicity) and continuous variables (skin parameter scores).

Linear mixed models (LMMs) were used to further analyse the data, with the key aim of testing for differences in skin parameter measurements, over the one hour period, between the control versus heat intervention, and the control versus moisture intervention. As the control and intervention conditions were mutually dependent, the estimates of the model were correlated *within* participants. As such, the data estimates generated by the LMMs provided an indication of the magnitude of change within interim timepoints of data collection for all participants, with a between-groups comparison for the control to the intervention. To determine variance over time, data gathered at baseline (*T0*) and interim

timepoints (*T10, T20, T30, T40* and *T50*) were compared against the endpoint timepoint at 60 minutes (*T60*) of each intervention.

A statistician was consulted through the La Trobe University Statistical Consultancy Platform to validate the study's statistical analysis plan. Clarification was specifically sought regarding how to control for the same participant being allocated to all three study conditions consecutively with consideration of the assumption of independence. Following discussions with a statistician, this was controlled for by including the Participant Identification Number (PIN) as a covariate in the statistical model. As such, the participant results gathered across all seven timepoints of data collection were matched to each PIN.

All LMM analyses were performed using the IBM Statistical Package for the Social Sciences (SPSS) Statistics (Version 25.0; IBM Corp., 2017). An overview of LMMs for repeated measures and their application to the data analysis procedures in the current study is detailed in the next section.

6.11.2 Linear Mixed Model (LMM) Analysis for Repeated Measures

Mixed models were first introduced by mathematician and statistician, Ronald Fisher, at the beginning of the 20th century and, to date, are widely-used methods of analysing repeated measures. A mixed model contains both fixed effects and random effects data (Fisher, 1954). These models are particularly useful in settings where repeated measures are made on the same statistical units, or where measurements are made on clusters of related statistical units (Suchmacher, Geller & Mauro, 2012).

Linear mixed models (LMMs) (also referred to as multi-level models, hierarchical linear models or random effect models) are known to provide a versatile approach to data analysis (Aarts et al., 2014; Audigé, Hanson & Kopjar, 2006; Kristensen & Hansen, 2004; McCulloch & Searle, 2008) where within-participant designs are common. LMMs comprise two types of terms: 'fixed effects' and 'random effects', hence the label 'mixed effects' (Magezi, 2015). The fixed effects term comprises exclusively fixed factors, and the fixed effect part of a LMM can vary in complexity depending on which terms are included. The random effects terms of LMMs are all the terms that include random factors; interactions between these fixed and random factors are considered in the random effects terms (Magezi, 2015). In a repeated measures design—where there are multiple measures from the same

subject—a term is needed for 'subjects' where, in almost all cases, 'subjects' are a 'random factor' while 'groups' are 'fixed' (McCulloch & Searle, 2008).

LMM is an extension of the general linear model (GLM) in which factors and covariates are assumed to have a linear relationship to the dependent variable. As such, the error terms and random effects are permitted to exhibit correlated and non-constant variability (Bell et al., 2019). LMM, therefore, provides the flexibility to model not only the mean of a response variable, but its covariance structure as well. In contrast to repeated measures analysis of variance (ANOVA), LMMs do not depend on limited assumptions about the variance-covariance matrix. Furthermore, LMMs provide the ability to include various configurations of grouping hierarchies, i.e., multiple nested groups (Magezi, 2015).

6.11.3 Bonferroni Adjustment

In this research, a large number of differences in dependent variables were explored using multiple analyses. When conducting multiple analyses on the same dependent variable, the chance of committing a Type I error increases, thereby increasing the likelihood of a significant result emerging by pure chance. To correct for this (i.e., to protect against Type I error), a Bonferroni adjustment (or Bonferroni correction) is conducted (Tabachnik & Fidell, 2007, p. 52). Bonferroni adjustment is one of the most commonly used correction procedures for multiple comparisons (Bland & Altman, 1986). This approach tries to control family-wise error rate in a very stringent criterion and compute the adjusted *p* values by directly multiplying the number of simultaneously tested hypotheses. The adjusted *p* values are compared with the pre-specified significance level $\alpha = .05$, and the statistical conclusion is altered before and after adjustment. Bonferroni adjustment has been well-acknowledged to be especially conservative when there are a large number of hypotheses being simultaneously tested and/or hypotheses are highly correlated (Chen, Feng & Yi, 2017).

In this research, as control condition data were used in subsequent analyses (in preparation for publication), a Bonferroni adjustment was applied to the alpha level to judge statistical significance. As two comparisons were simultaneously conducted, a maximum adjustment of $\alpha = .05 / 2$ was applied ($\alpha \leq .025$ – Bonferroni adjusted). As such, statistical significance was adjusted to the desired alpha (with 95% confidence) when running LMMs such that Type I errors could be corrected.

6.12 Ethical and Safety Considerations

During each study visit, the researcher stayed with each participant to ensure safety. Stopping protocol conditions applied in the event that a participant experienced discomfort during the study visit. Furthermore, during each off-loading interval, the researcher, having the experience of a Registered Nurse, inspected the skin surface of each anatomical testing site for signs of cutaneous irritation. For participants with deeply-pigmented skin, the anatomical testing sites were inspected for any blue/purple discolouration as indications of possible skin injury. The blanching status of each anatomical site was additionally assessed using brief fingertip application to ensure adequate tissue perfusion. In any instance where cutaneous irritation was apparent or where the skin did not respond as desired upon the blanch test, the study would be stopped and the area immediately off-loaded as per the current PI prevention guidelines: *Prevention and Treatment of Pressure Ulcers Clinical Practice Guidelines* (EPUAP-NPUAP-PPPIA, 2019).

6.13 Chapter Summary

This chapter has provided an outline of the quantitative methods that were used to address the research aims, questions, objectives and hypotheses of Study III of this thesis. An overview of the research design, together with the primary and secondary measures has been given. The study edibility criteria and recruitment procedures have been detailed, and the data collection instruments have been outlined. The relevant data collection procedures and statistical data analysis procedures have been explained. Finally, ethical considerations and safety measures have been discussed. In the next chapter, the descriptive data for the Study III sample are presented.

CHAPTER 7

Descriptive Results for Study III

7.1 Chapter Overview

In this chapter, the descriptive data for the Study III sample are presented. The chapter begins by providing an overview of the screening and preparation of the data, followed by a presentation of the participant sample with respect to their demographic and health data profile. Descriptive statistics are provided to profile erythema, stratum corneum hydration and skin temperature measures as gathered, over time, in the control and interventions. The chapter concludes with a summary of the descriptive results and conclusion.

7.2 Data Screening and Treatment

Demographic and health data, as well as skin parameter scores were explored using descriptive statistics and frequencies. A series of preliminary analyses were conducted to determine the degree of variability in the variables investigated, and to ensure there was no violation of the assumptions of normality, linearity and homoscedasticity. An inspection of histograms for continuous variables suggested these data did not violate distributional assumptions. Inspection of visual plots indicated nil evidence of influential outliers and nil violation of assumptions of linearity; that is, data followed a bivariate normal distribution.

7.3 Participant Recruitment and Sample

During the eight-month recruitment timeframe in Study III, 57 people contacted the researcher, expressing an interest to participate in the study after having viewed the study advertisement. During the telephone screening process, two people reported cutaneous conditions at one or more of the anatomical testing sites, and so did not meet the inclusion criteria and were ineligible to participate in the study. As such, 96% of enquiries translated into potentially eligible participants (n=55). Seven people chose to decline participation after reviewing the PICF, and so did not progress to be part of the study. All remaining eligible people (n=48) attended a baseline appointment and were part of the Study III sample. Of this sample, 46% of people (n=22) were former participants who had previously participated in the first phase of this research. None of the 48 participants subsequently withdrew from the study or were unable to complete the data collection. Figure 7.1 details the study participant

flow diagram illustrating recruited participants' involvement at baseline screenings with indication of withdrawals and inclusion in analyses.

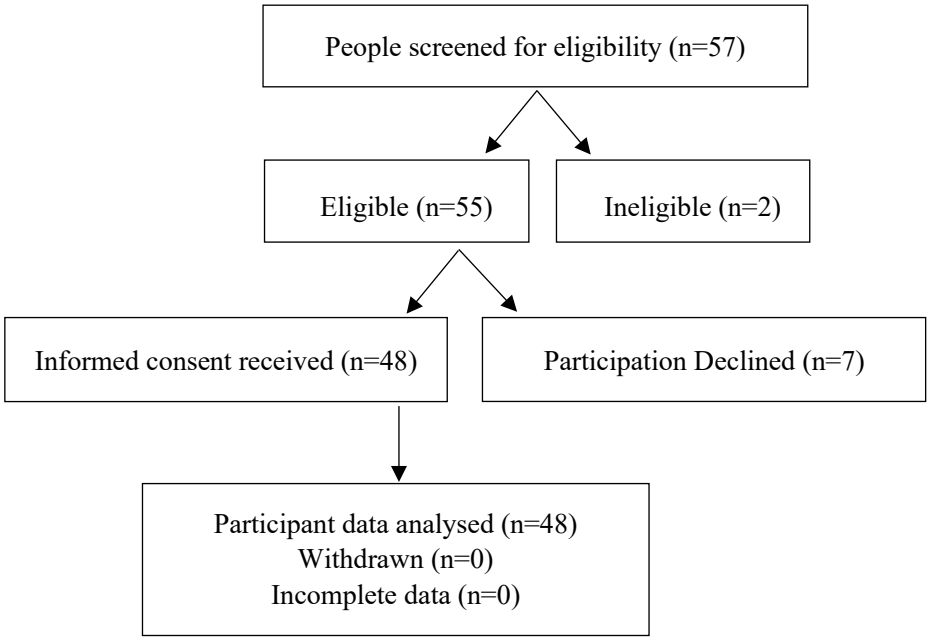

Figure 7.1 Study participant flow diagram illustrating recruited participants' involvement at baseline screenings, with indication of withdrawals and inclusion in analyses (Study III)

7.4 Participant Demographics

Table 7.1 provides an overview of participant demographic and clinical characteristics for Study III (n=48). The sample was predominately comprised of females (70.8%) with a mean participant age of 26.8 years (*SD*=9.5, *Min.*=18, *Max.*=62). The majority of participants (n=25) were of Caucasian ethnicity (52.1%) with a further 39.6% of participants of Asian ethnicity, and 4.2% of Hispanic and Middle-Eastern ethnicity, respectively. An average BMI[13] of 26.4 (*SD*=3.6) (*Min.*=19.3; *Max.*=32.6) was documented, with 68.8% (n=33) of participants reported to be within normal weight range and 31.3% (n=15) of participants reported to be overweight.

[13] Height and weight were measured using a stadiometer and digital scales, and BMI was calculated as weight (kg)/height (m^2). Participants were classed as *normal weight*, *underweight*, *overweight* or *obese* based on internationally recognised cut-offs (BMI 18.5-24.9, <18.5, 25-29.9 or >30 kg/m^2, respectively) (WHO, 2001).

Table 7.1

Participant demographic and health data profile (Study III) (n=48)

		Total n=48
Age (years)	M (SD)	26.8 (9.5)
	Med (IQR)	22.0 (11.0)
	Min.-Max.	18.0-62.0
Sex (% female)		70.8
Ethnicity (%)		
Caucasian		52.1
Asian		39.6
Hispanic		4.2
Middle-Eastern		4.2
Body Mass Index (kg/m^2)	M (SD)	24.6 (3.6)
	Med (IQR)	24.7 (5.7)
	Min.-Max.	19.3-32.6
% Within normal range (18.5-24.9)		68.8
% Underweight (<18.5)		-
% Overweight (25-29.9)		31.3
% Obese (>30 kg)		-

IQR=Interquartile range; M=mean; SD=standard deviation; Med=median; IQR=interquartile range; Min=minimum; Max=maximum.

Table 7.2 details participants' Fitzpatrick Skin Type scores obtained at baseline screening. Participants were classified into categories reflective of their calculated skin type. Almost half of the sample (41.7%) (n=20) had fair skin (Fitzpatrick Skin Type II), with the next highest category being pale white skin (Fitzpatrick Skin Type I) (22.9%) (n=11). No participants had dark brown to black skin (Fitzpatrick Skin Type VI). This variance in the Fitzpatrick Skin Type scores is reflective of the multi-ethnic study sample comprising Caucasian, Asian, Hispanic and Middle-Eastern ethnicities. The results of the Fitzpatrick Skin Type also align with the diverse range of baseline melanin scores recorded for the study sample. As revealed in the minimum and maximum data, the lowest melanin score was recorded at 3.0 units, while the highest melanin score was recorded at 74.0 units. An average baseline melanin score of 16.7 (*SD*=15.4) (*Min*.=4.0; *Max*.=73.0) was recorded at the right elbow, 16.5 (*SD*=15.3) (*Min*.=3.0; *Max*.=73.0) at the left elbow, 16.4 (*SD*=15.0) (*Min*.=3.0;

Max.=74.0) at the right heel, 16.5 (*SD*=15.2) (*Min*.=3.0; *Max*.=68.0) at the right heel, and 19.8 (*SD*=16.4) (*Min*.=4.0; *Max*.=73.0) at the sacrum. When considering the measure of spread for baseline melanin scores, the SDs were relatively large, indicating a high level of variance, with datapoints spread further away from the mean.

Table 7.2

Participant Fitzpatrick Skin Phototype Classification scores (Study III) (n=48)

	Total n=48
Fitzpatrick Skin Phototype Classification (%)	
Type I. Pale white skin	22.9
Type II. Fair skin	41.7
Type III. Fair to beige skin	6.3
Type IV. Light brown skin	16.7
Type V. Dark brown	12.4
Type V1. Dark brown or black skin	-

No participant self-reported having a history of PI. All participants received a score of 20 (*SD*=0.0) on the Norton Risk Assessment Scale (NRAS) indicative of negligible risk of PI development (Norton, 1962).

The next section provides the descriptive results for skin parameters gathered in the Study III sample (n=48). The descriptive results are organised into results for erythema (Section 7.5), stratum corneum hydration (Section 7.6) and skin temperature (Section 7.7) consecutively.

7.5 Descriptive Results for Erythema

A large range of erythema scores (1.0-63.0 units in the control, 1.0-78.0 in the heat intervention, and 1.0-79.0 in the moisture intervention) was registered across all anatomical sites, implying large regional variation for individuals in their erythema scores. These findings can be interpreted as participants having ‘nil erythema’ (1-18 units), ‘minimal erythema’ (19-36 units), ‘diffused redness’ (37-54 units) and ‘high erythema’ (55-80 units)

as per the SD202 Skin Diagnostic Measurement Interpretation Guide (Barrett-Hill, 2015) (see Figure 6.2 in Chapter 6).

7.5.1. Control versus Heat Intervention

Table 7.3 provides the descriptive results of erythema for the control as compared to the heat intervention. Results are presented by anatomical location for all participants (n=48) across all timepoints of data collection.

Table 7.3
Erythema measurements for the control versus heat intervention by anatomical location – Study III (n=48)

			Time (minutes)						
			0	10	20	30	40	50	60
Right Elbow	Control	M (SD)	20.4 (13.3)	21.6 (12.5)	22.3 (13.6)	21.8 (13.0)	21.7 (13.1)	22.6 (13.1)	21.9 (12.7)
		Med (IQR)	16.0 (21.5)	19.0 (19.8)	20.0 (21.0)	20.5 (2.5)	21.5 (17.8)	21.5 (18.5)	20.5 (21.0)
		Min.-Max.	2.0–50.0	2.0–44.0	1.0–50.0	2.0–50.0	2.0–50.0	1.0–54.0	2.0–48.0
	Heat	M (SD)	23.4 (13.3)	22.9 (12.9)	23.6 (14.0)	24.8 (14.2)	24.1 (13.0)	25.2 (14.3)	25.4 (13.7)
		Med (IQR)	23.0 (20.5)	21.0 (22.0)	25.0 (22.8)	22.0 (23.5)	20.5 (21.5)	22.0 (23.0)	21.0 (24.8)
		Min.-Max.	1.0–49.0	3.0–51.0	2.0–52.0	2.0–55.0	3.0–49.0	4.0–55.0	3.0–53.0
Left Elbow	Control	M (SD)	20.3 (12.7)	21.9 (13.1)	21.3 (13.8)	21.3 (13.2)	22.0 (13.6)	21.5 (12.3)	21.7 (12.5)
		Med (IQR)	19.5 (19.8)	20.0 (19.0)	19.5 (22.5)	17.5 (19.8)	18.0 (20.8)	20.5 (18.8)	19.5 (18.8)
		Min.-Max.	2.0–49.0	2.0–54.0	1.0–50.0	4.0–51.0	2.0–45.0	2.0–49.0	1.0–45.0
	Heat	M (SD)	22.1 (12.4)	23.9 (13.2)	25.4 (14.9)	24.4 (12.9)	24.8 (13.3)	24.8 (13.2)	25.9 (14.0)
		Med (IQR)	21.0 (21.0)	21.0 (19.0)	24.5 (23.5)	23.5 (22.8)	22.0 (21.5)	21.0 (22.5)	21.5 (23.0)
		Min.-Max.	2.0–50.0	2.0–50.0	2.0–55.0	5.0–56.0	3.0–55.0	4.0–50.0	3.0–55.0
Right Heel	Control	M (SD)	22.9 (12.8)	21.6 (12.4)	22.4 (10.9)	24.1 (12.3)	22.9 (11.9)	23.1 (12.3)	23.6 (12.1)
		Med (IQR)	21.0 (21.8)	17.5 (21.8)	21.5 (18.8)	25.0 (19.0)	21.5 (19.5)	22.0 (22.3)	24.0 (20.5)
		Min.-Max.	2.0–59.0	3.0–50.0	4.0–42.0	2.0–54.0	3.0–44.0	4.0–51.0	3.0–51.0
	Heat	M (SD)	25.0 (14.0)	26.5 (14.1)	27.3 (15.3)	26.7 (13.5)	26.0 (14.0)	27.7 (15.2)	28.1 (15.4)
		Med (IQR)	21.5 (21.5)	25.0 (21.5)	23.5 (30.5)	30.6 (19.5)	22.5 (22.8)	25.5 (22.0)	25.0 (25.3)
		Min.-Max.	3.0–55.0	4.0–55.0	7.0–55.0	3.0–59.0	2.0–55.0	4.0–60.0	40–56.0
Left Heel	Control	M (SD)	22.8 (11.8)	22.2 (11.0)	23.2 (11.9)	22.5 (11.5)	24.1 (12.3)	23.5 (12.2)	24.2 (12.8)
		Med (IQR)	22.0 (16.5)	21.0 (15.0)	23.0 (18.5)	22.5 (19.0)	23.0 (18.8)	21.0 (18.8)	22.5 (23.0)
		Min.-Max.	2.0–51.0	4.0–52.0	3.0–53.0	3.0–50.0	3.0–56.0	4.0–41.0	3.0–50.0
	Heat	M (SD)	24.7 (13.1)	26.6 (15.0)	27.8 (13.8)	26.6 (13.2)	26.8 (14.4)	28.1 (15.9)	28.1 (15.0)
		Med (IQR)	24.0 (20.8)	22.0 (24.3)	27.5 (23.5)	27.0 (20.5)	25.5 (21.8)	22.5 (24.3)	26.0 (24.5)
		Min.-Max.	2.0–52.0	3.0–55.0	5.0–550	3.0–55.0	4.0–55.0	3.0–57.0	3.0–57.0
Sacrum	Control	M (SD)	31.3 (13.5)	30.7 (13.7)	30.9 (12.4)	31.4 (13.4)	30.9 (12.7)	32.5 (13.5)	32.1 (13.1)
		Med (IQR)	32.0 (21.8)	31.0 (22.5)	30.0 (19.8)	31.0 (20.5)	31.0 (18.0)	31.5 (19.8)	33.0 (20.8)
		Min.-Max.	9.0–63.0	8.0–59.0	9.0–57.0	6.0–58.0	8.0–56.0	10.0–60.0	7.0–56.0
	Heat	M (SD)	36.0 (14.3)	37.4 (14.6)	37.0 (14.2)	38.9 (14.9)	38.1 (13.9)	40.6 (14.0)	41.0 (15.2)
		Med (IQR)	33.5 (24.5)	34.5 (25.8)	35.0 (24.5)	35.0 (23.8)	37.0 (20.5)	40.5 (21.5)	39.0 (21.3)
		Min.-Max.	12.0–72.0	13.0–67.0	13.0–67.0	14.0–78.0	14.0–70.0	16.0–74.0	18.0–77.0

As detailed in Table 7.3, there was minimal variation in mean scores of erythema across timepoints—in both the control and heat intervention—across all anatomical testing sites and amongst individual study participants (n=48). In the control, erythema scores appeared to oscillate with no discernible pattern across all anatomical testing sites, and were relatively consistent. In the heat intervention, the pattern of scores appeared to vary by anatomical region. To illustrate, while there were some oscillations in erythema observed at the elbows and heels (with scores generally tending to increase at the final timepoints), the sacral skin evidenced steady 10 minute incremental increases in erythema.

The lowest mean erythema scores were recorded at *T0* in the control for the left elbow (*M*=20.3, *SD*=12.7) and right elbow (*M*=20.4, *SD*=13.3). The highest mean erythema scores were recorded at *T50* in the control for the sacrum (*M*=32.5, *SD*=13.2) and at *T60* in the heat intervention for the sacrum (*M*=41.0, *SD*=15.2).

The greatest mean difference in erythema scores between baseline and end was recorded for the sacrum (1.5 units for the control, and 5.0 units for heat intervention). It should be noted that these differences, when considered on a scale ranging from 0 to 99, are relatively small and might not be clinically meaningful.

In both conditions, the mean erythema scores at the elbows and heels were consistently lower than erythema scores at the sacrum. In the control, mean erythema scores were recorded between 20.3-22.6 units at the elbows, 21.6-24.2 units at the heels, and 30.7-32.5 units at the sacrum. Conversely, in the heat intervention, mean erythema scores were recorded between 22.1-25.9 units at the elbows, 24.7-28.4 units at the heels, and 36.0-41.0 units at the sacrum.

There was a large range in erythema scores, as evidenced by the minimum and maximum data, at all anatomical testing sites for both conditions. The lowest erythema score was recorded at 1.0 units for the right elbow (in both the control and heat intervention), while the highest erythema scores were recorded at 63.0 units at the sacrum for the control, and at 78.0 units at the sacrum for the heat intervention.

7.5.2 Control versus Moisture Intervention

Table 7.4 provides the descriptive results of erythema for the control as compared to the moisture intervention. Results are presented by anatomical location for all participants (n=48) across all timepoints of data collection.

Table 7.4
Erythema measurements for the control versus moisture intervention by anatomical location – Study III (n=48)

			Time (minutes)						
			0	10	20	30	40	50	60
Right Elbow	Control	M (SD)	20.4 (13.3)	21.6 (12.5)	22.3 (13.6)	21.8 (13.0)	21.7 (13.1)	22.6 (13.1)	21.9 (12.7)
		Med (IQR)	16.0 (21.5)	19.0 (19.8)	20.0 (21.0)	20.5 (2.5)	21.5 (17.8)	21.5 (18.5)	20.5 (21.0)
		Min.-Max.	2.0–50.0	2.0–44.0	1.0–50.0	2.0–50.0	2.0–50.0	1.0–54.0	2.0–48.0
	Moisture	M (SD)	21.4 (12.9)	22.8 (12.1)	23.4 (13.4)	23.8 (13.4)	23.7 (13.9)	24.2 (13.4)	24.2 (14.1)
		Med (IQR)	20.0 (17.8)	20.0 (18.0)	21.0 (18.8)	21.5 (20.3)	19.5 (22.0)	21.0 (20.0)	20.5 (17.8)
		Min.-Max.	2.0–55.0	3.0–55.0	4.0–60.0	2.0–57.0	4.0–57.0	3.0–58.0	2.0–66.0
Left Elbow	Control	M (SD)	20.3 (12.7)	21.9 (13.1)	21.3 (13.8)	21.3 (13.2)	22.0 (13.6)	21.5 (12.3)	21.7 (12.5)
		Med (IQR)	19.5 (19.8)	20.0 (19.0)	19.5 (22.5)	17.5 (19.8)	18.0 (20.8)	20.5 (18.8)	19.5 (18.8)
		Min.-Max.	2.0–49.0	2.0–54.0	1.0–50.0	4.0–51.0	2.0–45.0	2.0–49.0	1.0–45.0
	Moisture	M (SD)	22.8 912.0)	22.6 (13.6)	23.3 (12.4)	24.1 (14.2)	23.3 (13.3)	24.4 (15.0)	24.0 (14.3)
		Med (IQR)	21.0 (17.0)	20.0 (21.0)	21.0 (21.5)	19.5 (23.2)	20.5 (20.8)	21.0 (23.7)	20.0 (22.5)
		Min.-Max.	4.0–52.0	1.0–56.0	2.0–55.0	3.0–55.0	2.0–57.0	2.0–69.0	4.0–6.0
Right Heel	Control	M (SD)	22.9 (12.8)	21.6 (12.4)	22.4 (10.9)	24.1 (12.3)	22.9 (11.9)	23.1 (12.3)	23.6 (12.1)
		Med (IQR)	21.0 (21.8)	17.5 (21.8)	21.5 (18.8)	25.0 (19.0)	21.5 (19.5)	22.0 (22.3)	24.0 (20.5)
		Min.-Max.	2.0–59.0	3.0–50.0	4.0–42.0	2.0–54.0	3.0–44.0	4.0–51.0	3.0–51.0
	Moisture	M (SD)	26.4 (14.6)	26.5 (14.2)	25.8 (13.4)	27.1 (14.2)	27.0 (14.6)	27.7 (14.7)	27.8 (15.6)
		Med (IQR)	21.5 (27.8)	28.0 (25.8)	21.6 (24.0)	28.0 (24.0)	26.5 (22.8)	28.5 (23.3)	26.5 (27.8)
		Min.-Max.	2.0–55.0	2.0–56.0	5.0–56.0	6.0–57.0	5.0–58.0	5.0–58.0	5.0–57.0
Left Heel	Control	M (SD)	22.8 (11.8)	22.2 (11.0)	23.2 (11.9)	22.5 (11.5)	24.1 (12.3)	23.5 (12.2)	24.2 (12.8)
		Med (IQR)	22.0 (16.5)	21.0 (15.0)	23.0 (18.5)	22.5 (19.0)	23.0 (18.8)	21.0 (18.8)	22.5 (23.0)
		Min.-Max.	2.0–51.0	4.0–52.0	3.0–53.0	3.0–50.0	3.0–56.0	4.0–41.0	3.0–50.0
	Moisture	M (SD)	25.3 (13.5)	26.7 (13.9)	27.6 (14.7)	27.6 (13.9)	26.9 (14.1)	27.7 (14.9)	27.8 (14.2)
		Med (IQR)	21.5 (21.5)	23.0 (23.8)	27.5 (25.0)	25.5 (22.8)	27.0 (22.8)	28.0 (24.8)	26.0 (21.5)
		Min.-Max.	3.0–52.0	6.0–57.0	4.0–57.0	6.0–57.0	6.0–57.0	3.0–57.0	5.0–58.0
Sacrum	Control	M (SD)	31.3 (13.5)	30.7 (13.7)	30.9 (12.4)	31.4 (13.4)	30.9 (12.7)	32.5 (13.5)	32.1 (13.1)
		Med (IQR)	32.0 (21.8)	31.0 (22.5)	30.0 (19.8)	31.0 (20.5)	31.0 (18.0)	31.5 (19.8)	33.0 (20.8)
		Min.-Max.	9.0–63.0	8.0–59.0	9.0–57.0	6.0–58.0	8.0–56.0	10.0–60.0	7.0–56.0
	Moisture	M (SD)	31.5 (17.8)	31.2 (17.0)	32.0 (16.1)	32.1 (17.1)	32.7 (16.2)	33.2 (17.7)	34.8 (17.5)
		Med (IQR)	25.0 (22.8)	26.5 (26.0)	28.5 (24.0)	27.5 (20.0)	28.0 (19.8)	28.0 (24.0)	29.0 (26.0)
		Min.-Max.	2.0–77.0	7.0–77.0	11.0–74.0	8.0–76.0	7.0–76.0	6.0–79.0	11.0–78.0

As detailed in Table 7.4, there was minimal variation in mean scores of erythema across timepoints—in both the control and moisture intervention—at all anatomical testing sites and amongst individual study participants. Overall, scores oscillated between timepoints with no discernible pattern and were relatively constant across all anatomical locations. However, scores of erythema generally tended to increase at the final timepoints.

The lowest mean erythema scores were recorded at *T0* in the control for the left elbow (*M*=20.3, *SD*=12.7) and right elbow (*M*=20.4, *SD*=13.3). The highest mean erythema scores were recorded at *T50* in the control for the sacrum (*M*=32.5, *SD*=13.2) and at *T60* in the moisture intervention for the sacrum (*M*=34.8, *SD*=17.52).

The greatest mean difference in erythema scores between baseline and end was recorded for the sacrum (1.5 units for the control, and 3.3 units for moisture intervention). It should be noted, however, that these differences, when considered on a scale of 0 to 99, are relatively small and might not be clinically meaningful.

In both conditions, the mean erythema scores at the elbows and heels were consistently lower and displayed less variability than erythema scores at the sacrum. To illustrate, in the control, mean erythema scores were recorded between 20.3-22.6 units at the elbows, 21.6-24.2 units at the heels, and 30.7-32.5 units at the sacrum. Conversely, in the moisture intervention, mean erythema scores were recorded between 21.4-24.4 units at the elbows, 25.3-27.8 units at the heels, and 31.2-34.8 units at the sacrum.

There was a large range in erythema scores, as evidenced by the minimum and maximum data, at all anatomical testing sites for both conditions. The lowest erythema score was recorded at 1.0 units for the elbows in both the control and moisture intervention, while the highest erythema scores were recorded at 63.0 units at the sacrum for the control, and at 79.0 units at the sacrum for the moisture intervention.

7.6 Descriptive Results for Stratum Corneum Hydration

A large range of stratum corneum hydration scores (0-77.0 units in the control, 0-78.0 units in the heat intervention, and 0-99.0 in the moisture intervention) was registered across all anatomical testing sites, implying large regional variations. These results imply that that participants had variably dry and excessively moist skin which—when considered on a scale ranging from 0 to 99 units—align with a clinical interpretation of ‘very dry skin’ (0-23

units), ‘dry skin’ (24-45 units), ‘optimal hydration’ (46-62 units), ‘higher level of hydration’ (65-82 units) to ‘over-hydrated skin’ (83-99 units) as per the SD202 Skin Diagnostic Measurement Interpretation Guide (Barrett-Hill, 2015).

7.6.1 Control versus Heat Intervention

Table 7.5 provides the descriptive results of stratum corneum hydration for the control as compared to the heat intervention. Results are presented by anatomical locations for all participants (n=48) across all timepoints of data collection.

Table 7.5

Stratum corneum hydration measurements for the control versus heat intervention by anatomical location – Study III (n=48)

			Time (minutes)						
			0	10	20	30	40	50	60
Right Elbow	Control	M (SD)	8.1 (8.9)	8.9 (9.5)	9.1 (9.4)	9.0 (8.1)	8.7 (8.3)	9.0 (8.2)	8.6 (7.6)
		Med (IQR)	8.0 (10.8)	7.0 (12.0)	7.5 (12.5)	7.5 (13.0)	6.5 (12.0)	8.0 (10.8)	6.5 (11.0)
		Min.-Max.	0.0–53.0	0.0–53.0	0.0–54.0	0.0–41.0	0.0–45.0	0.0–44.0	0.0–39.0
	Heat	M (SD)	9.3 (7.7)	8.7 (8.2)	8.7 (8.6)	9.6 (7.8)	10.0 (8.3)	9.7 (8.3)	9.7 (8.0)
		Med (IQR)	8.5 (11.0)	9.0 (12.0)	7.0 (12.0)	9.0 (9.3)	9.0 (10.8)	8.5 (11.5)	7.5 (10.8)
		Min.-Max.	0.0–40.0	0.0–42.0	0.0–40.0	0.0–44.0	0.0–46.0	0.0–49.0	0.0–45.0
Left Elbow	Control	M (SD)	9.6 (10.2)	8.5 (9.5)	9.5 (9.8)	8.5 (8.0)	9.1 (8.3)	8.6 (8.2)	9.2 (8.4)
		Med (IQR)	8.0 (11.75)	7.0 (12.0)	9.0 (11.8)	8.0 (9.8)	8.0 (10.7)	8.0 (10.8)	7.5 (12.8)
		Min.-Max.	0.0–52.0	0.0–40.0	0.0–49.0	0.0–41.0	0.0–44.0	0.0–42.0	0.0–42.0
	Heat	M (SD)	8.8 (8.1)	9.4 (8.4)	8.8 (7.3)	9.3 (8.0)	9.7 (8.3)	9.5 (7.6)	9.8 (8.4)
		Med (IQR)	8.0 (9.8)	7.0 (12.0)	8.5 (10.8)	8.5 (11.0)	8.0 (10.8)	8.0 (10.8)	8.0 (11.8)
		Min.-Max.	0.0–44.0	0.0–44.0	0.0–40.0	0.0–39.0	0.0–46.0	0.0–41.0	0.0–45.0
Right Heel	Control	M (SD)	8.9 (12.7)	8.8 (12.6)	9.3 (12.8)	8.0 (12.0)	8.3 (12.0)	8.7 (12.5)	8.7 (12.4)
		Med (IQR)	0.0 (16.0)	0.0 (12.0)	2.0 (15.0)	0.0 (13.8)	0.0 (16.0)	0.5 (12.0)	1.5 (13.8)
		Min.-Max.	0.0–43.0	0.0–44.0	0.0–50.0	0.0–48.0	0.0–45.0	0.0–46.0	0.0–53.0
	Heat	M (SD)	8.3 (11.2)	9.1 (12.1)	8.6 (12.5)	9.4 (12.1)	9.4 (12.3)	8.9 (12.2)	8.6 (11.8)
		Med (IQR)	0.5 (13.8)	1.0 (16.0)	0.0 (15.0)	2.0 (16.0)	2.0 (16.8)	0.0 (15.0)	0.5 (14.8)
		Min.-Max.	0.0–42.0	0.0–45.0	0.0–44.0	0.0–37.0	0.0–44.0	0.0–47.0	0.0–47.0
Left Heel	Control	M (SD)	9.1 (12.3)	8.9 (12.9)	8.7 (12.6)	8.8 (12.4)	8.7 (12.3)	8.4 (12.5)	8.4 (12.0)
		Med (IQR)	0.0 (12.3)	0.0 (14.0)	0.5 (14.0)	1.0 (13.5)	0.0 (15.0)	1.5 (14.5)	1.0 (13.0)
		Min.-Max.	0.0–42.0	0.0–48.0	0.0–44.0	0.0–43.0	0.0–41.0	0.0–41.0	0.0–45.0
	Heat	M (SD)	8.4 (11.3)	9.0 (12.3)	8.7 (12.2)	9.4 (12.7)	8.9 (12.2)	8.9 (12.1)	9.1 (12.0)
		Med (IQR)	1.0 (13.5)	0.0 (15.8)	1.0 (13.5)	1.0 (15.8)	0.5 (13.0)	2.0 (14.5)	0.5 (15.8)
		Min.-Max.	0.0–47.0	0.0–45.0	0.0–40.0	0.0–44.0	0.0–46.0	0.0–44.0	0.0–42.0
Sacrum	Control	M (SD)	27.1 (15.6)	27.6 (16.0)	28.0 (15.6)	27.4 (15.3)	28.4 (14.8)	29.2 (16.1)	29.3 (14.8)
		Med (IQR)	22.0 (16.0)	22.5 (12.5)	22.5 (14.0)	23.5 (17.8)	26.5 (16.8)	25.0 (19.2)	25.0 (19.8)
		Min.-Max.	10.0–77.0	7.0–72.0	9.0–77.0	7.0–72.0	7.0–70.0	7.0–76.0	9.0–70.0
	Heat	M (SD)	31.5 (17.8)	31.2 (17.0)	32.0 (16.1)	32.1 (17.1)	32.7 (16.2)	33.2 (17.7)	34.8 (17.5)
		Med (IQR)	25.0 (22.8)	26.5 (26.0)	28.5 (24.0)	27.5 (20.0)	28.0 (19.8)	28.0 (24.0)	29.0 (26.0)
		Min.-Max.	2.0–77.0	7.0–77.0	11.0–74.0	8.–76.0	7.0–76.0	6.0–78.0	11.0–78.0

As detailed in Table 7.5, there was minimal variation in mean scores of stratum corneum hydration across timepoints—in both the control and heat intervention—at all anatomical testing sites and amongst individual study participants (n=48). Across all anatomical testing sites, stratum corneum hydration scores appeared to oscillate with no discernible pattern, and were relatively consistent. The most discernible pattern was evidenced at the sacrum where, in general, scores tended to rise consistently but steadily between timepoints, and were notably higher at endpoints when compared to baseline.

The lowest mean stratum corneum hydration score was recorded at *T30* for the right heel in the control (*M*=8.0, *SD*=12.0), with the next lowest score recorded at *T0* for the right elbow in the control (*M*=8.1, *SD*=8.9). The highest mean stratum corneum hydration scores were recorded at *T60* for the sacrum in both the control (*M*=29.3, *SD*=14.8) and heat intervention (*M*=34.8, *SD*=17.5).

The greatest mean difference in stratum corneum hydration scores between baseline and end was recorded at the sacrum (2.2 units for the control, and 3.3 units for heat intervention). It should be noted, however, that these differences, when considered on a scale ranging from 0 to 99, are relatively small and might not be clinically meaningful.

In both conditions, the sacrum, on average, registered moderately higher levels of stratum corneum hydration than the elbows and heels. To illustrate, in the control, mean stratum corneum hydration scores were recorded between 8.1-9.5 units at the elbows, 8.0-9.3 units at the heels, and 27.1-29.3 units at the sacrum. In the heat intervention, mean stratum corneum hydration scores were recorded between 8.7-10.0 units at the elbows, 8.3-9.4 units at the heels, and 31.2-34.8 units at the sacrum.

There was a large range in stratum corneum hydration scores, as evidenced by the minimum and maximum data, at all anatomical testing sites for both conditions. To illustrate, the minimum stratum corneum hydration score was recorded at 0.0 units on at least one occasion in both conditions, while the maximum scores were recorded at 77.0 units and 78.0 units at the sacrum for the control and heat intervention, respectively.

7.6.2 Control versus Moisture Intervention

Table 7.6 provides the descriptive results of stratum corneum hydration for the control as compared to the heat intervention. Results are presented by anatomical location for all participants (n=48) across all timepoints of data collection.

Table 7.6

Stratum corneum hydration measurements for the control versus moisture intervention by anatomical location – Study III (n=48)

			Time (minutes)						
			0	10	20	30	40	50	60
Right Elbow	Control	M (SD)	8.1 (8.9)	8.9 (9.5)	9.1 (9.4)	9.0 (8.1)	8.7 (8.3)	9.0 (8.2)	8.6 (7.6)
		Med (IQR)	8.0 (10.8)	7.0 (12.0)	7.5 (12.5)	7.5 (13.0)	6.5 (12.0)	8.0 (10.8)	6.5 (11.0)
		Min.-Max.	0.0–53.0	0.0–53.0	0.0–54.0	0.0–41.0	0.0–45.0	0.0–44.0	0.0–39.0
	Moisture	M (SD)	8.7 (7.4)	9.8 (7.4)	9.3 (7.9)	9.1 (7.2)	9.6 (7.5)	9.9 (7.7)	9.8 (7.6)
		Med (IQR)	7.0 (10.5)	7.5 (11.0)	8.5 (12.0)	8.0 (10.0)	8.0 (10.8)	9.0 (9.8)	10.0 (11.0)
		Min.-Max.	0.0–31.0	0.0–32.0	0.0–40.0	0.0–35.0	0.0–36.0	0.0–37.0	0.0–36.0
Left Elbow	Control	M (SD)	9.6 (10.2)	8.5 (9.5)	9.5 (9.8)	8.5 (8.0)	9.1 (8.3)	8.6 (8.2)	9.2 (8.4)
		Med (IQR)	8.0 (11.75)	7.0 (12.0)	9.0 (11.8)	8.0 (9.8)	8.0 (10.7)	8.0 (10.8)	7.5 (12.8)
		Min.-Max.	0.0–52.0	0.0–40.0	0.0–49.0	0.0–41.0	0.0–44.0	0.0–42.0	0.0–42.0
	Moisture	M (SD)	9.4 (7.5)	9.2 (7.3)	9.2 (7.8)	10.1 (7.0)	9.9 (7.7)	9.4 (7.6)	10.1 (7.0)
		Med (IQR)	6.0 (11.8)	7.0 (11.0)	8.5 (9.8)	8.0 (9.8)	9.5 (12.0)	7.0 (10.8)	10.0 (8.8)
		Min.-Max.	0.0–32.0	0.0–33.0	0.0–39.0	0.0–36.0	0.0–34.0	0.0–33.0	0.0–35.0
Right Heel	Control	M (SD)	8.9 (12.7)	8.8 (12.6)	9.3 (12.8)	8.0 (12.0)	8.3 (12.0)	8.7 (12.5)	8.6 (12.4)
		Med (IQR)	0.0 (16.0)	0.0 (12.0)	2.0 (15.0)	0.0 (13.8)	0.0 (16.0)	0.5 (12.0)	1.5 (13.8)
		Min.-Max.	0.0–43.0	0.0–44.0	0.0–50.0	0.0–48.0	0.0–45.0	0.0–46.0	0.0–53.0
	Moisture	M (SD)	7.9 (10.7)	8.2 (10.6)	8.8 (11.3)	8.5 (11.1)	7.9 (10.7)	8.2 (11.4)	8.2 (11.3)
		Med (IQR)	0.0 (13.8)	0.5 (14.0)	2.0 (15.0)	3.0 (13.8)	1.0 (14.8)	1.5 (14.0)	2.0 (14.8)
		Min.-Max.	0.0–34.0	0.0–35.0	0.0–40.0	0.0–36.0	0.0–38.0	0.0–45.0	0.0–45.0
Left Heel	Control	M (SD)	9.1 (12.3)	8.9 (12.9)	8.7 (12.6)	8.8 (12.4)	8.7 (12.3)	8.4 (12.5)	8.4 (12.0)
		Med (IQR)	0.0 (12.3)	0.0 (14.0)	0.5 (14.0)	1.0 (13.5)	0.0 (15.0)	1.5 (14.5)	1.0 (13.0)
		Min.-Max.	0.0–42.0	0.0–48.0	0.0–44.0	0.0–43.0	0.0–41.0	0.0–41.0	0.0–45.0
	Moisture	M (SD)	8.6 (11.8)	8.6 (12.4)	8.6 (12.0)	8.3 (10.8)	8.0 (11.1)	7.6 (10.2)	8.6 (11.6)
		Med (IQR)	1.0 (17.0)	0.0 (13.8)	0.0 (14.0)	1.5 (15.0)	0.5 (13.8)	1.0 (14.5)	1.0 (14.8)
		Min.-Max.	0.0–45.0	0.0–54.0	0.0–45.0	0.0–35.0	0.0–45.0	0.0–34.0	0.0–45.0
Sacrum	Control	M (SD)	27.1 (15.6)	27.6 (16.0)	28.0 (15.6)	27.4 (15.3)	28.4 (14.8)	29.2 (16.1)	29.3 (14.8)
		Med (IQR)	22.0 (16.0)	22.5 (12.5)	22.5 (14.0)	23.5 (17.8)	26.5 (16.8)	25.0 (19.2)	25.0 (19.8)
		Min.-Max.	10.0–77.0	7.0–72.0	9.0–77.0	7.0–72.0	7.0–70.0	7.0–76.0	9.0–70.0
	Moisture	M (SD)	99.0 (0.3)	99.0 (0.4)	99.0 (0.3)	99.0 (0.2)	99.0 (0.3)	99.0 (0.3)	99.0 (0.5)
		Med (IQR)	99.0 (0.0)	99.0 (0.0)	99.0 (0.0)	99.0 (0.0)	99.0 (0.0)	99.0 (0.0)	99.0 (0.0)
		Min.-Max.	98.0–99.0	97.0–99.0	97.0–99.0	98.0–99.0	98.0–99.0	98.0–99.0	97.0–99.0

As detailed in Table 7.6, the lowest mean stratum corneum hydration score was recorded at *T30* for the right heel in the control (*M*=8.0, *SD*=12.0), with the next lowest score recorded at *T0* for the right elbow in the control (*M*=8.1, *SD*=8.9). The highest mean stratum corneum hydration score was recorded at *T60* for the sacrum in the control (*M*=29.3, *SD*=14.8). In the moisture intervention, mean stratum corneum hydration scores of 99.0 units were recorded across all measurement timepoints.

The greatest mean difference in stratum corneum hydration scores between baseline and end was recorded at the sacrum (2.2 units for the control, and 1.1 units for moisture intervention). It should be noted, however, that these differences, when considered on a scale ranging from 0 to 99, are relatively small and might not be clinically meaningful.

In both the control and moisture intervention, the mean scores of stratum corneum hydration at the elbows and heels were comparable. To illustrate, in the control, mean stratum corneum hydration scores were recorded between 8.1-9.5 units at the elbows and between 8.0-9.3 units at the heels. In the moisture intervention, mean stratum corneum hydration scores were recorded between 8.7-10.1 units and the elbows and between 7.6-8.8 units at the heels. However, when considering scores of stratum corneum hydration at the sacrum, scores were considerably higher in in the moisture intervention. To illustrate, mean scores of stratum corneum hydration ranged between 27.1-29.2 in the control, and between 99.0-99.0 units in the moisture intervention.

There was a large range in stratum corneum hydration scores, as evidenced by the minimum and maximum data, at all anatomical testing sites for both conditions. To illustrate, the minimum stratum corneum hydration score was recorded at 0.0 units on at least one occasion in both conditions, while the maximum scores were recorded at 77.0 units and 99.0 units at the sacrum for the control and moisture intervention, respectively.

7.7 Descriptive Results for Skin Temperature

A large range of skin temperature scores (21.8-8.5°C for the control, 22.0-42.8°C for the heat intervention, and 21.9-39.6°C in the moisture intervention) was registered across all anatomical testing sites with further variation observed across timepoints, implying large regional variations in skin temperature amongst study participants. While there is no formal reference range for skin temperatures of the human body (which can vary widely between person to person and by anatomical region) (Armstrong & Lavery, 1997), mean skin

temperature values are often reported in thermoregulation studies with human subjects. Assuming neutral (21–25°C) ambient temperatures and a core temperature of ~37.0°C, skin temperature at the trunk of the human body varies between 33.5–36.9°C, and between 23.5–35.6°C at the extremities (Millington, 1983). In the current study, the mean skin temperatures gathered for the control—when compared against the mean skin temperatures published by Millington (1983)—are largely comparable.

7.7.1 Control versus Heat Intervention

Table 7.7 provides the descriptive results of skin temperature for the control as compared to the heat intervention. Results are presented by anatomical location for all participants (n=48) across all timepoints of data collection.

Table 7.7

Skin temperature measurements for the control versus heat intervention by anatomical location – Study III (n=48)

			Time (minutes)						
			0	10	20	30	40	50	60
Right Elbow	Control	M (SD)	28.6 (2.3)	29.3 (2.3)	29.5 (2.4)	29.9 (2.5)	30.1 (2.4)	30.5 (2.5)	30.8 (2.6)
		Med (IQR)	29.3 (2.8)	29.7 (3.1)	29.9 (3.2)	30.1 (2.8)	30.4 (3.1)	30.9 (3.2)	31.0 (3.4)
		Min.-Max.	22.0–32.6	23.1–33.2	23.5–33.8	23.9–34.2	24.6–34.4	24.9–35.6	25.1–35.9
	Heat	M (SD)	29.0 (2.5)	30.1 (2.7)	30.9 (2.7)	31.9 (2.8)	32.5 (2.7)	33.4 (2.8)	34.2 (3.0)
		Med (IQR)	29.8 (3.1)	31.0 (3.4)	31.9 (3.5)	32.9 (3.1)	33.4 (3.0)	34.4 (3.3)	35.0 (3.6)
		Min.-Max.	22.2–33.4	23.5–34.2	24.1–35.2	25.3–35.9	26.0–37.2	26.5–37.9	26.7–38.7
Left Elbow	Control	M (SD)	29.0 (2.3)	29.3 (2.3)	29.6 (2.4)	29.8 (2.4)	30.3 (2.5)	30.4 (2.5)	30.7 (2.6)
		Med (IQR)	29.6 (2.8)	29.8 (2.9)	30.4 (2.8)	30.6 (3.1)	31.0 (3.1)	31.0 (3.2)	31.3 (3.3)
		Min.-Max.	22.5–32.7	22.9–33.0	23.2–33.6	23.9–33.9	23.9–34.0	24.1–35.0	24.5–35.8
	Heat	M (SD)	29.0 (2.5)	30.2 (2.6)	31.0 (2.6)	31.7 (2.7)	32.7 (2.8)	33.6 (2.9)	34.4 (3.0)
		Med (IQR)	30.0 (3.0)	31.0 (3.0)	31.9 (2.7)	32.6 (3.4)	33.7 (3.3)	34.8 (3.1)	35.4 (3.5)
		Min.-Max.	22.5–33	23.2–34.0	24.4–35.0	25.2–36.1	26.1–36.9	26.5–38.0	26.8–38.4
Right Heel	Control	M (SD)	26.4 (2.9)	26.7 (2.9)	27.0 (3.0)	27.6 (3.0)	27.7 (3.0)	28.1 (3.0)	28.5 (3.2)
		Med (IQR)	27.2 (5.8)	27.6 (5.5)	27.8 (5.8)	28.0 (5.9)	28.2 (5.8)	28.7 (6.0)	29.1 (5.8)
		Min.-Max.	21.9–32.3	22.3–32.4	22.5–32.5	22.8–33.2	22.7–33.8	22.6–33.9	22.7–34.4
	Heat	M (SD)	27.7 (3.3)	28.8 (3.3)	29.2 (3.4)	30.1 (3.5)	30.7 (3.5)	31.7 (3.5)	31.7 (3.5)
		Med (IQR)	28.4 (6.7)	29.1 (7.0)	30.0 (6.4)	30.5 (6.3)	30.1 (3.5)	31.6 (6.5)	31.6 (6.5)
		Min.-Max.	23.0–33.0	23.2–33.6	23.8–34.6	24.6–36.0	25.1–37.6	25.7–38.3	25.7–38.3
Left Heel	Control	M (SD)	26.4 (2.9)	26.8 (2.8)	27.1 (2.9)	27.5 (3.0)	27.8 (3.0)	28.0 (3.0)	28.5 (3.1)
		Med (IQR)	27.3 (5.9)	27.9 (5.6)	27.9 (6.0)	28.2 (6.0)	28.3 (5.5)	29.0 (5.8)	29.2 (5.9)
		Min.-Max.	21.8–32.1	22.4–32.3	22.6–32.6	23.0–33.9	23.2–34.1	23.0–34.2	23.5–34.9
	Heat	M (SD)	26.6 (3.0)	27.7 (3.4)	28.4 (3.4)	29.4 (3.6)	30.0 (3.5)	30.9 (3.6)	31.7 (3.6)
		Med (IQR)	27.4 (6.0)	28.6 (7.1)	29.5 (6.7)	30.4 (6.0)	30.8 (6.1)	31.6 (6.1)	31.9 (6.0)
		Min.-Max.	22.0–32.5	22.6–33.4	23.5–34.5	23.6–35.9	24.1–36.7	24.9–37.9	25.2–38.3
Sacrum	Control	M (SD)	32.3 (2.3)	32.7 (2.2)	33.1 (2.2)	33.6 (2.2)	33.9 (2.2)	34.5 (2.2)	35.0 (2.2)
		Med (IQR)	32.6 (2.3)	32.9 (3.2)	33.5 (3.4)	33.7 (3.6)	34.2 (4.1)	34.8 (3.9)	35.0 (3.9)
		Min.-Max.	26.3–35.8	26.7–35.9	27.9–36.8	28.2–37.2	28.8–37.5	29.5–38.0	29.8–38.5
	Heat	M (SD)	32.7 (2.3)	33.9 (2.2)	34.8 (2.3)	35.8 (2.2)	36.6 (2.1)	37.5 (2.2)	38.4 (2.2)
		Med (IQR)	33.0 (3.3)	34.0 (3.3)	34.9 (3.3)	36.0 (3.0)	36.9 (2.7)	37.9 (2.7)	38.6 (2.8)
		Min.-Max.	26.2–36.0	27.6–36.8	28.4–37.5	29.9–38.8	30.6–39.9	31.4–41.4	31.9–42.8

As detailed in Table 7.7, minimal to moderate variation emerged in mean scores of skin temperature across timepoints—in both the control and heat intervention—at all anatomical testing sites and amongst individual study participants (n=48). Skin temperature scores increased uniformly over time, with lowest scores observed at baseline (*T0*) and highest scores noted at endpoint at 60 minutes (*T60*) across all anatomical locations. The lowest mean skin temperature scores were recorded at *T0* in the control for the left heel (*M*=26.4, *SD*=2.9) and right heel (*M*=26.7, *SD*=2.9). The highest mean skin temperature scores were recorded at *T60* for the sacrum in both the control (*M*=35.0*, SD*=2.2) and heat intervention (*M*=38.4*, SD*=2.2).

The greatest mean difference in skin temperature scores between baseline and end was recorded at the sacrum (2.7°C for the control, and 5.7°C for the heat intervention). In both conditions, the mean skin temperature scores at the elbows and heels were consistently lower and had less variability than erythema scores at the sacrum. To illustrate, in the control, mean skin temperature scores were recorded between 28.6-30.7°C at the elbows, 26.4-28.5°C at the heels, and 32.3-35.0°C at the sacrum. In the heat intervention, mean skin temperature scores were recorded between 29.0-34.4°C at the elbows, 26.6-31.7°C at the heels, and 32.7-38.4°C at the sacrum among participants. This anatomical variation in skin temperature scores implies that participants, on average, experienced higher skin temperatures at the core of the body than at the extremities. Additionally, when considering mean scores of skin temperature in the heat intervention, a more pronounced variability in scores was evident at the sacrum than at the elbows and heels.

There was a moderate range in skin temperature scores, as revealed in the minimum and maximum data, at all anatomical testing sites for both conditions. In the control, the minimum skin temperature score was recorded at 21.8°C at the left heel, while the maximum skin temperature score was recorded at 38.5°C at the sacrum. In the heat intervention, the minimum skin temperature score was recorded at 22.0°C at the left heel, while the maximum skin temperature score was registered at 42.8°C at the sacrum.

7.7.2 Control versus Moisture Intervention

Table 7.8 provides the descriptive results of skin temperature for the control as compared to the moisture intervention. Results are presented by anatomical location for all participants (n=48) across all timepoints of data collection.

Table 7.8

Skin temperature measurements for the control versus moisture intervention by anatomical location – Study III (n=48)

			Time (minutes)						
			0	10	20	30	40	50	60
Right Elbow	Control	M (SD)	28.6 (2.3)	29.3 (2.3)	29.5 (2.4)	29.9 (2.5)	30.1 (2.4)	30.5 (2.5)	30.8 (2.6)
		Med (IQR)	29.3 (2.8)	29.7 (3.1)	29.9 (3.2)	30.1 (2.8)	30.4 (3.1)	30.9 (3.2)	31.0 (3.4)
		Min.-Max.	22.0–32.6	23.1–33.2	23.5–33.8	23.9–34.2	24.6–34.4	24.9–35.6	25.1–35.9
	Moisture	M (SD)	29.2 (2.5)	29.9 (2.6)	30.1 (2.5)	30.5 (2.3)	30.9 (2.4)	31.5 (2.5)	31.8 (2.4)
		Med (IQR)	29.9 (3.0)	30.7 (2.7)	31.1 (3.4)	31.4 (3.0)	31.7 (3.4)	32.0 (3.0)	32.4 (3.0)
		Min.-Max.	22.0–33.0	23.0–35.1	23.2–34.1	24.6–34.4	25.1–35.0	25.3–35.8	25.3–36.8
Left Elbow	Control	M (SD)	29.0 (2.3)	29.3 (2.3)	29.6 (2.4)	29.8 (2.4)	30.3 (2.5)	30.4 (2.5)	30.7 (2.6)
		Med (IQR)	29.6 (2.8)	29.8 (2.9)	30.4 (2.8)	30.6 (3.1)	31.0 (3.1)	31.0 (3.2)	31.3 (3.3)
		Min.-Max.	22.5–32.7	22.9–33.0	23.2–33.6	23.9–33.9	23.9–34.0	24.1–35.0	24.5–35.8
	Moisture	M (SD)	29.3 (2.5)	29.7 (2.5)	30.2 (2.5)	30.6 (2.5)	30.9 (2.4)	31.4 (2.4)	31.9 (2.5)
		Med (IQR)	30.0 (3.0)	30.4 (3.2)	31.0 (3.0)	31.4 (2.9)	31.5 (2.9)	32.0 (3.0)	32.6 (2.8)
		Min.-Max.	22.5–33.4	22.9–33.7	24.0–34.0	24.4–34.8	25.0–35.1	25.2–35.7	26.0–36.5
Right Heel	Control	M (SD)	26.4 (2.9)	26.7 (2.9)	27.0 (3.0)	27.6 (3.0)	27.7 (3.0)	28.1 (3.0)	28.5 (3.2)
		Med (IQR)	27.2 (5.8)	27.6 (5.5)	27.8 (5.8)	28.0 (5.9)	28.2 (5.8)	28.7 (6.0)	29.1 (5.8)
		Min.-Max.	21.9–32.3	22.3–32.4	22.5–32.5	22.8–33.2	22.7–33.8	22.6–33.9	22.7–34.4
	Moisture	M (SD)	26.7 (3.0)	27.1 (3.1)	27.4 (3.1)	27.9 (30)	28.4 (3.2)	28.7 (3.2)	29.3 (3.2)
		Med (IQR)	27.7 (5.9)	27.9 (6.5)	28.2 (6.4)	29.1 (6.4)	29.6 (6.3)	29.5 (6.0)	30.1 (6.0)
		Min.-Max.	21.9–33.1	22.7–33.5	22.8–33.9	22.9–34.0	23.5–35.2	23.6–36.9	24.1–37.2
Left Heel	Control	M (SD)	26.4 (2.9)	26.8 (2.8)	27.1 (2.9)	27.5 (3.0)	27.8 (3.0)	28.0 (3.0)	28.5 (3.1)
		Med (IQR)	27.3 (5.9)	27.9 (5.6)	27.9 (6.0)	28.2 (6.0)	28.3 (5.5)	29.0 (5.8)	29.2 (5.9)
		Min.-Max.	21.8–32.1	22.4–32.3	22.6–32.6	23.0–33.9	23.2–34.1	23.0–34.2	23.5–34.9
	Moisture	M (SD)	26.7 (3.0)	27.1 (3.0)	27.5 (3.0)	28.1 (3.1)	28.3 (3.1)	29.0 (3.1)	29.5 (3.2)
		Med (IQR)	27.5 (6.4)	27.9 (6.3)	28.4 (6.4)	29.0 (6.3)	29.0 (6.3)	29.5 (5.9)	29.9 (5.8)
		Min.-Max.	21.9–33.1	22.8–33.4	23.0–33.9	23.3–34.8	23.8–35.7	23.9–37.1	24.8–37.8
Sacrum	Control	M (SD)	32.3 (2.3)	32.7 (2.2)	33.1 (2.2)	33.6 (2.2)	33.9 (2.2)	34.5 (2.2)	35.0 (2.2)
		Med (IQR)	32.6 (2.3)	32.9 (3.2)	33.5 (3.4)	33.7 (3.6)	34.2 (4.1)	34.8 (3.9)	35.0 (3.9)
		Min.-Max.	26.3–35.8	26.7–35.9	27.9–36.8	28.2–37.2	28.8–37.5	29.5–38.0	29.8–38.5
	Moisture	M (SD)	32.0 (2.3)	33.5 (2.3)	34.4 (2.3)	34.8 (2.4)	35.0 (2.4)	35.3 (2.3)	35.8 (2.3)
		Med (IQR)	33.4 (3.1)	33.8 (3.4)	34.3 (3.5)	34.8 (3.7)	34.8 (3.6)	35.1 (3.3)	35.6 (3.5)
		Min.-Max.	26.2–36.7	26.3–36.8	26.7–37.9	27.5–38.4	27.6–38.4	28.2–39.1	29.3–39.6

As detailed in Table 7.8, minimal to moderate variation emerged in mean scores of skin temperature across timepoints—in both the control and moisture intervention—at all anatomical testing sites and amongst individual study participants (n=48). Skin temperature scores increased uniformly over time, with lowest scores observed at baseline (*T0*) and highest scores observed at endpoints (*T60*) across all anatomical locations. The lowest mean skin temperature scores were recorded at *T0* in the control for the left heel (*M*=26.4, *SD*=2.9) and right heel (*M*=26.7, *SD*=2.9). Conversely, the highest mean skin temperature scores were recorded at *T60* for the sacrum in both the control (*M*=26.4*, SD*=2.9) and moisture intervention (*M*=26.7*, SD*=2.9).

The greatest mean difference in skin temperature scores between baseline and end was recorded at the sacrum (2.7°C for the control, and 3.8°C for the moisture intervention). In both conditions, the mean skin temperature scores at the elbows and heels were consistently lower and had less variability than erythema scores at the sacrum. To illustrate, in the control, mean skin temperature scores were recorded between 28.6-30.7°C at the elbows, 26.4-28.5°C at the heels, and 32.3-35.0°C at the sacrum. In the moisture intervention, mean skin temperature scores were recorded between 29.2-31.9°C at the elbows, 26.7-29.5°C at the heels, and 32.0-35.8°C at the sacrum among participants. This anatomical variation in skin temperature scores implies that participants, on average, experienced higher skin temperatures at the core of the body than at the extremities.

There was a moderate range in skin temperature scores, as revealed in the minimum and maximum data, at all anatomical testing sites for both conditions. In the control, the minimum skin temperature score was recorded at 21.8°C at the left heel, while the maximum skin temperature score was recorded at 38.5°C at the sacrum. In the moisture intervention, the minimum skin temperature score was recorded at 21.9°C at the right heel, while the maximum score was registered at 39.6°C at the sacrum.

7.8 Summary of Descriptive Results

Objective 1 of Study III was to establish a descriptive library of skin parameter scores—as measured over time and across pressure-prone areas—during intermittent pressure off-loading in a control, heat intervention, and moisture intervention. Overall, Objective 1 was met with the establishment of a descriptive library of skin parameter scores at the elbows, heels and sacrum of healthy adults. As such, the collection of descriptive data results allowed the research team to address research gap IV of this thesis.

Quantitative measurements of erythema, stratum corneum hydration and skin temperature were gathered at the elbows, heels, and sacrum of participants. Measurements were considered across a control and two interventions; heat and moisture. In the heat intervention, the elbows, heels and sacrum were exposed to warmth generated from an electric heated blanket. In the moisture intervention, the sacrum was exposed to an absorbency pad saturated with normal saline. Descriptive data were generated to profile erythema, stratum corneum hydration and skin temperature measures at baseline, interim (10 minute) and final timepoints of each condition.

Among the 48 participants recruited in Study III, a large range of skin parameter scores was noted at all anatomical testing sites for the testing conditions, suggesting large regional variation in scores between participants. Conversely, when considering scores within participants, a minimal to moderate range of scores was observed at all anatomical testing sites, suggesting minimal to moderate regional variation for individual people. When examining the regional distributional characteristics of skin parameters, the elbows and heels were typically characterised by lower readings of erythema, stratum corneum hydration and skin temperature when compared to the sacrum, suggesting differences by anatomical location. On each measurement occasion, the sacrum registered the highest scores.

Minimal oscillations emerged between interim timepoints for erythema at the elbows and heels in all study conditions. However, in the heat intervention, erythema rose steadily over the 10 minute intervals, with endpoint scores notably higher than baseline scores. Minimal oscillations also emerged between interim timepoints for stratum corneum hydration; however, when considering the sacrum in the moisture intervention, maximum readings of 99.0 units for stratum corneum hydration were noted across all timepoints. Skin temperature displayed the most consistent incremental change of all skin parameters, with a gradual increase in scores noted upon consecutive readings. On average, skin parameter scores tended to be lowest at baseline and highest at end across at all anatomical testing sites and in all study conditions.

7.9 Chapter Summary

This chapter has presented the descriptive data for the study III participant sample. The chapter described the participant sample with respect to their demographic and health data profile. Descriptive statistics have been provided to profile erythema, stratum corneum hydration and skin temperature measures as gathered, over time, in control and intervention

conditions. Overall, the descriptive results generated in Study III provided a foundation for the linear mixed model (LMM) analyses extending from the first part of this analysis. In the next chapter, the results of the LMMs are presented.

CHAPTER 8

Linear Mixed Model Analyses Results for Study III

8.1 Chapter Overview

In this chapter, the linear mixed models (LMMs) results for Study III are presented. This chapter begins with overview of the screening and treatment of the data related to LMM analysis, including the extent to which data met assumptions of linearity as well as bivariate distribution. The results of LMM analyses are subsequently reported for each skin parameter variable by anatomical location for the participant sample. The chapter concludes with a summary of linear mixed model analyses results.

8.2 Overview of Analyses

Repeated measures of erythema, stratum corneum hydration, and skin temperature were collected from the 48 healthy participants positioned semi-recumbent in three consecutive conditions; condition 1: control, condition 2: heat intervention, condition 3: moisture intervention. Data collected in the control were compared to data gathered in the intervention conditions (in respective analyses) to examine differences in skin parameter measurements. These differences in skin parameter measurements were analysed using LMMs for the study sample (n=48), with analyses repeated for each anatomical testing site. The overall differences in the control versus heat intervention and control versus moisture intervention were considered, over time, between the conditions. Furthermore, data estimates—outputted for each 10 minute time increment over the one hour period—provided an indication of the magnitude of change by the interim timepoints of data collection against the final timepoint.

8.3 Data Screening and Treatment

A series of preliminary analyses were conducted to determine the degree of variability in the variables investigated, and to ensure there was no violation of the assumptions of normality, linearity and homoscedasticity. An inspection of histograms for continuous variables suggested these data did not violate distributional assumptions. Inspection of visual plots indicated nil evidence of influential outliers and nil violation of assumptions of linearity; that is, data followed a normal bivariate normal distribution.

8.3.1 Preliminary Analyses of Linear Mixed Model Variables

Baseline demographics (age, gender, ethnicity), clinical characteristics (BMI), and skin profile data (Fitzpatrick Skin Phototype and baseline melanin measures) were examined using bivariate correlations for their association with skin parameter measurements. These analyses would assist in identifying potential covariates to be considered in the LMM analyses.

Pearson's product-moment correlation coefficient (r) was used to measure the strength of linear correlations between variables. As per Cohen's criteria, a correlation is considered 'weak' if the value of r varies between .1–.29, 'moderate' if the value of r varies between .3–.49 and 'strong' if r is equal to or exceeds .5 (Cohen, 1988). In Study III, any variable with an r value of $\geq$.3 was considered as a covariate and, as such, would be incorporated into further analyses.

When examining bivariate relationships between age and skin parameter scores, with the exception of skin temperature at *T0* for the sacrum in the heat intervention (age r=-.4, p=.01), all bivariate relationships were weak (r<.3, p<.01). Upon inspection of correlations between gender and skin parameter scores, with the exception of skin temperature at *T0* for the sacrum in the heat intervention (gender r=.4, p=.01) and at *T30* in the control (gender r=.4, p=.01), all bivariate relationships were weak (r<.3, p<.01). Following an inspection of correlations between baseline melanin and skin parameter scores, all bivariate relationships were weak (r<3, p <.01). Spearman's Rho was used to assess the associations between ordinal variables (ethnicity, BMI and Fitzpatrick Skin Phototype) and continuous variables (skin parameter scores). All associations between these variables were found to be weak (r<.03, p<.01). Given the consistent weak associations between demographic/clinical variables and skin parameter scores, these variables were excluded from subsequent analyses and, as such, are not reported further.

The next section presents the LMM analyses results for the study sample (n=48). The LMM results for the control versus interventions are presented consecutively for erythema (Section 8.4), stratum corneum hydration (Section 8.5) and skin temperature (Section 8.6). Tables 8.1 to 8.10, 8.11 to 8.20, and 8.21 to 8.30 detail the results of the LMM analyses—including the SE, df, t test score, p values and 95% CI—for erythema, stratum corneum hydration and skin temperature, respectively.

8.4 Linear Mixed Model Analyses Results for Erythema by Anatomical Location

8.4.1 Control versus Heat Intervention

Tables 8.1 to 8.5 provide the LMM analysis results for erythema, by anatomical location, for the study sample (n=48). The results detail the differences between the control versus heat intervention, and further contrast the data gathered at baseline and interim (every 10 minute) timepoints against the final timepoint.

Table 8.1

Linear mixed model analysis results for erythema at the right elbow – control versus heat intervention (n=48)

Parameter	Estimate	SE	df	t	95% CI	*p* value
Intercept	24.9	1.9	99.4	13.1	(21.1, 28.6)	$p < .001$
Heat int.	-2.5	2.6	94	-0.9	(-7.7, 2.8)	$p = .357$
T0	-1.7	0.5	570	-3.6	(-2.7, -0.8)	$p < .001$
T10	-1.4	0.5	570	-2.8	(-2.3, -0.4)	$p = .005$
T20	-0.7	0.5	570	-1.4	(-1.6, 0.3)	$p = .173$
T30	-0.3	0.5	570	-0.7	(-1.3, 0.6)	$p = .475$
T40	-0.7	0.5	570	-1.6	(-1.8, 0.2)	$p = .114$
T50	-0.3	0.5	570	0.5	(-0.7, 1.1)	$p = .603$

$\alpha \leq .025$ for Bonferroni adjustment, T=Time, Int=Intervention

Table 8.2

Linear mixed model analysis results for erythema at the left elbow – control versus heat intervention (n=48)

Parameter	Estimate	SE	df	t	95% CI	*p* value
Intercept	25.3	1.9	100.5	13.4	(21.6, 29.0	$p < .001$
Heat int.	-3.0	2.6	94	-1.2	(-8.2, 2.1)	$p = .249$
T0	-2.6	0.5	570	-5.0	(-3.7, -1.6)	$p = .014$
T10	-0.9	0.5	570	-1.7	(-1.9, 0.1)	$p = .091$
T20	-0.4	0.5	570	-0.9	(-1.5, 0.6)	$p = .392$
T30	-0.9	0.5	570	-1.8	(-2.0, 0.9)	$p = .074$
T40	-0.4	0.5	570	-0.7	(-1.4, 0.6)	$p = .462$
T50	-0.6	0.5	570	-1.3	(-0.7, 0.4)	$p = .210$

$\alpha \leq .025$ for Bonferroni adjustment, T=Time, Int=Intervention

The LMM results for erythema are presented for the right elbow (Table 8.1) and left elbow (Table 8.2). No statistically significant differences in erythema scores were observed between the control and heat intervention for the right elbow [p=.357, 95% CI (-7.7, 2.8)] and left elbow [p=.249, 95% CI (-8.2, 2.1)]. These data suggest that the introduction of heat to the skin surface of semi-recumbent participants did not alter erythema scores at their elbows. Thus, the hypothesis that in the heat intervention—when compared to the control—there would be a significant increase in erythema at the right and left elbow (Alternative Hypothesis One) was not supported ($p > .025$).

The differences in erythema results at *T60* and those at *T0* and *T10* for the right elbow, and at *T0* for the left elbow were statistically significant ($\alpha \leq .025$). These data suggest that erythema scores changed from the initial measures to the last measure in the heat intervention.

Table 8.3

Linear mixed model analysis results for erythema at the right heel – control versus heat intervention (n=48)

Parameter	Estimate	SE	df	t	95% CI	*p* value
Intercept	27.7	1.9	102.6	14.7	(24.0. 31.5)	$p < .001$
Heat int.	-3.8	2.6	94	-1.4	(-9.0, 1.4)	$p = .150$
T0	-1.9	0.5	570	-3.2	(-3.1, -0.7)	$p < .001$
T10	-1.8	0.5	570	-3.0	(-3.0, -0.6)	$p < .001$
T20	-1.0	0.5	570	-1.7	(-2.1, 0.2)	$p = .092$
T30	-0.4	0.5	570	-0.7	(-1.6, 0.7)	$p = .454$
T40	-1.4	0.5	570	-2.4	(-2.6, -0.2)	$p = .018$
T50	-0.5	0.5	570	-0.8	(-1.6, 0.7)	$p = .434$

$\alpha \leq .025$ for Bonferroni adjustment, T=Time, Int=Intervention

Table 8.4

Linear mixed model analysis results for erythema at the left heel – control versus heat intervention (n=48)

Parameter	Estimate	SE	df	t	95% CI	*p* value
Intercept	28.1	1.9	100.2	15.0	(24.4. 31.8)	$p < .001$
Heat int.	-3.9	2.6	94	-1.5	(-8.9, 1.4)	$p = .148$
T0	-2.5	0.5	570	-4.9	(-3.5, -1.5)	$p < .001$
T10	-1.8	0.5	570	-3.6	(-2.8, -0.8)	$p < .001$
T20	-0.7	0.5	570	-1.4	(-1.7, 0.3)	$p = .163$
T30	-1.6	0.5	570	-3.2	(-2.6, 0.6)	$p < .001$
T40	-0.7	0.5	570	-1.5	(-1.8, 0.2)	$p = .134$
T50	-0.4	0.5	570	-0.8	(-1.4, 0.6)	$p = .411$

$\alpha \leq .025$ for Bonferroni adjustment, T=Time, Int=Intervention

The LMM results for erythema are presented for the right heel (Table 8.3) and left heel (Table 8.4). No statistically significant differences in erythema were observed between the control and heat intervention for the right heel [p=.150, 95% CI (-9.0, 1.4)] and left heel

[p=.148, 95% CI (-8.9, 1.4)]. These data suggest that the introduction of heat at the skin surface to semi-recumbent participants did not alter erythema scores at their heels. Thus, the hypothesis that in the heat intervention—when compared to the control—there would be a significant increase in erythema at the right and left heel (Alternative Hypothesis Two) was not supported ($p > .025$).

The differences in erythema results at *T60* and *T0* and *T10* for both the right and left heel were statistically significant ($\alpha \leq .025$) suggesting change in erythema measures from the initial first two measures to the last measure. However, fluctuations between timepoints were evident, with a significant difference also detected between *T60* and *T40* for the right heel (p=.018) and *T30* for the left heel (p<.001). This suggests a change in erythema measures from the measures obtained in the first 30 and 40 minutes (for the left and right heel, respectively) of the time period compared to the last measure at 60 minutes.

Table 8.5

Linear mixed model analysis results for erythema at the sacrum – control versus heat intervention (n=48)

Parameter	Estimate	SE	df	t	95% CI	*p* value
Intercept	40.1	2.0	100.2	20.4	(36.2, 44.0)	$p < .001$
Heat int.	-7.0	2.7	94	-2.6	(-12.4, -1.6)	$p = .012$
T0	-3.0	0.5	570	-5.5	(-4.0, -1.9)	$p < .001$
T10	-2.6	0.5	570	-4.8	(-3.6, -1.5)	$p < .001$
T20	-2.6	0.5	570	-5.0	(-3.7, -1.6)	$p < .001$
T30	-1.4	0.5	570	-2.7	(-2.4, -0.4)	$p = .007$
T40	-2.1	0.5	570	-3.9	(-3.1, -1.0)	$p < .001$
T50	-0.0	0.5	570	-0.1	(-0.1, 1.0)	$p = .938$

$\alpha \leq .025$ for Bonferroni adjustment, T=Time, Int=Intervention

The LMM results for erythema are presented for the sacrum in Table 8.5. The LMM results revealed statistically significant differences in erythema scores between the control and heat intervention at the sacrum [p=.012, 95% CI (-12.4, -1.6)]. Thus, the application of heat did alter scores of erythema at the sacral region. As per Table 8.5, the erythema

measurements collected in the control for the sacrum were 7.0 units lower than erythema measurements collected in the heat intervention.

The hypothesis that in the heat intervention—when compared to the control—there would be a significant increase in erythema at the sacrum (Alternative Hypothesis Three) was supported ($p \leq .025$).

With the exception of *T50*, the differences in erythema scores at the sacrum were statistically significant ($\alpha \leq .025$) across all timepoints, suggesting change in erythema measures over time.

8.4.2 Control versus Moisture Intervention

Tables 8.6 to 8.10 provide the LMM results for erythema, by anatomical testing site, for the study sample (n=48). The results detail the differences between the control versus moisture intervention, and further contrast the data gathered at baseline and interim timepoints against the final timepoint. It should be noted that in the moisture intervention, the moisture stimulus was only applied to participants' sacral skin as per the Study III protocol. However, erythema measurements in the moisture intervention were attended to at the elbows and heels as a control, and are reported accordingly.

Table 8.6

Linear mixed model analysis results for erythema at the right elbow – control versus moisture intervention (n=48)

Parameter	Estimate	SE	df	t	95% CI	*p* value
Intercept	23.8	1.9	99.9	12.7	(20.1, 27.5)	$p < .001$
Moisture int.	-1.6	2.6	94	-0.6	(-6.8, 3.6)	$p = .541$
T0	-2.1	0.5	570	-4.1	(-3.1, -1.1)	$p < .001$
T10	-0.8	0.5	570	-1.6	(-1.8, 0.2)	$p = .112$
T20	-0.2	0.5	570	-0.3	(-1.1, 0.8)	$p = .747$
T30	-0.2	0.5	570	-0.4	(-1.2, 0.8)	$p = .685$
T40	-0.3	0.5	570	-0.6	(-1.3, 0.7)	$p = .526$
T50	-0.4	0.5	570	0.8	(-0.6, 1.4)	$p = .435$

$\alpha \leq .025$ for Bonferroni adjustment, T=Time, Int=Intervention

Table 8.7

Linear mixed model analysis results for erythema at the left elbow – control versus moisture intervention (n=48)

Parameter	Estimate	SE	df	t	95% CI	*p* value
Intercept	23.9	1.9	101.0	12.6	(20.1. 27.7)	$p < .001$
Moisture int.	-2.1	2.6	94	-0.8	(-7.3, 3.1)	$p = .433$
T0	-1.3	0.5	570	-2.5	(-2.4, -0.3)	$p = .014$
T10	-0.6	0.5	570	-1.2	(-1.7, 0.4)	$p = .250$
T20	-0.2	0.5	570	-1.1	(-1.7, 0.5)	$p = .283$
T30	-0.2	0.5	570	-0.3	(-1.2, 0.9)	$p = .743$
T40	-0.4	0.5	570	-0.4	(-1.3, 0.9)	$p = .700$
T50	-0.4	0.5	570	0.1	(-1.0, 1.1)	$p = .945$

$\alpha \leq .025$ for Bonferroni adjustment, T=Time, Int=Intervention

The LMM results for erythema are presented for the right elbow (Table 8.6) and left elbow (Table 8.7). No statistically significant differences in erythema were observed between the control and moisture intervention for the right elbow [p=.541, 95% CI (-6.8,

3.6)] and left elbow [p=0.433, 95% CI (-7.3, 3.1)]. Thus, the hypothesis that in the moisture intervention—when compared to the control—there would be no significant increase in erythema at the right and left elbow (Null Hypothesis Twelve) failed to be rejected ($p > .025$).

The differences in erythema results between *T0* and *T60* for both the right and left elbow were statistically significant ($\alpha \leq .025$), suggesting change in erythema measures from the initial measure to the last measure.

Table 8.8

Linear mixed model analysis results for erythema at the right heel – control versus moisture intervention (n=48)

Parameter	Estimate	SE	df	t	95% CI	*p* value
Intercept	27.7	1.9	100.2	14.6	(23.9, 31.4)	$p < .001$
Moisture int.	-4.1	2.6	94	-1.6	(-9.3, 1.1)	$p = .123$
T0	-1.0	0.5	570	-1.8	(-2.0, 0.0)	$p = .060$
T10	-1.5	0.5	570	-3.0	(-2.6, -0.5)	$p = .003$
T20	-1.5	0.5	570	-3.0	(-2.5, -0.5)	$p = .003$
T30	-0.5	0.5	570	-1.0	(-1.5, 0.5)	$p = .342$
T40	-0.7	0.5	570	-1.3	(-1.7, 0.3)	$p = .196$
T50	-0.2	0.5	570	-0.4	(-1.2, 0.8)	$p = .671$

$\alpha \leq .025$ for Bonferroni adjustment, T=Time, Int=Intervention

Table 8.9

Linear mixed model analysis results for erythema at the left heel – control versus moisture intervention (n=48)

Parameter	Estimate	SE	df	t	95% CI	*p* value
Intercept	27.8	1.9	100.0	15.0	(24.1, 31.6)	$p < .001$
Moisture int.	-3.9	2.6	94	-1.5	(-9.0, 1.3)	$p = .137$
T0	-1.9	0.5	570	-3.8	(-2.9, -0.9)	$p < .001$
T10	-1.5	0.5	570	-3.0	(-2.5, -0.5)	$p = .003$
T20	-0.5	0.5	570	-1.0	(-1.5, 0.5)	$p = .304$
T30	-0.8	0.5	570	-1.7	(-1.8, 0.2)	$p = .098$
T40	-0.4	0.5	570	-0.8	(-1.4, 0.6)	$p = .452$
T50	-0.3	0.5	570	-0.7	(-1.3, 0.6)	$p = .502$

$\alpha \leq .025$ for Bonferroni adjustment, T=Time, Int=Intervention

The LMM results for erythema are presented for the right heel (Table 8.8) and left heel (Table 8.9). No statistically significant differences in erythema scores were observed between the control and moisture intervention for the right heel [p=.123, 95% CI (-9.3, 1.1)] and left heel [p=.137, 95% CI (-9.0, 1.3]. Thus, the hypothesis that in the moisture intervention—when compared to the control—there would be no significant increase in erythema at the right and left heel (Null Hypothesis Thirteen) failed to be rejected ($p > .025$).

The differences in erythema results at *T60* and *T10* and *T20* for the right heel, and at *T0* and *T10* for the left heel were statistically significant ($\alpha \leq .025$), suggesting change in erythema measures from the initial measures to the last measure.

Table 8.10

Linear mixed model analysis results for erythema at the sacrum – control versus moisture intervention (n=48)

Parameter	Estimate	SE	df	t	95% CI	*p* value
Intercept	35.9	1.9	101.0	18.9	(32.7, 40.4)	$p < .001$
Moisture int.	-4.3	2.7	94	-1.6	(-9.7, 0.9)	$p = .107$
T0	-1.1	0.6	570	-1.9	(-2.1, -0.0)	$p = .058$
T10	-1.1	0.6	570	-2.0	(-2.2, -0.5)	$p = .041$
T20	-1.8	0.6	570	-3.2	(-2.9, -0.7)	$p < .001$
T30	-0.4	0.6	570	-0.6	(-1.5, -0.7)	$p = .499$
T40	-1.0	0.6	570	-1.7	(-2.0, -0.1)	$p = .088$
T50	-0.7	0.6	570	-0.1	(-1.2, 1.0)	$p = .895$

$\alpha \leq .025$ for Bonferroni adjustment, T=Time, Int=Intervention

The LMM results for erythema are presented for the sacrum in Table 8.10. No statistically significant differences in erythema scores were observed between the control and moisture intervention [p=.107, 95% CI (-9.7, -0.9)]. Thus, the application of moisture did not alter scores of erythema at the sacral region. The hypothesis that in the moisture intervention—when compared to the control—there would be no significant increase in erythema at the sacrum (Alternative Hypothesis Fourteen) was supported ($p > .025$).

The differences in erythema results between *T20* and *T60* timepoints for the sacrum were statistically significant ($\alpha \leq .025$), suggesting change in erythema measures from the initial measure at 20 minutes to the last measure.

8.5 Linear Mixed Model Analyses Results for Stratum Corneum Hydration by Anatomical Location

8.5.1 Control versus Heat Intervention

Tables 8.11 to 8.15 provide the LMM results for stratum corneum hydration, by anatomical testing site, for the study sample (n=48). The results detail the differences between the control versus heat intervention, and further contrast the data gathered at baseline and interim timepoints against the final timepoint.

Table 8.11

Linear mixed model analysis results for stratum corneum hydration at the right elbow – control versus heat intervention (n=48)

Parameter	Estimate	SE	df	t	95% CI	*p* value
Intercept	9.4	1.2	103.0	7.8	(7.0, 11.7)	$p < .001$
Heat int.	-0.4	1.7	94	-0.2	(-3.7, 2.9)	$p = .817$
T0	0.2	0.4	570	0.6	(-0.5, 1.0)	$p = .521$
T10	-0.4	0.4	570	-0.9	(-1.1, 0.4)	$p = .347$
T20	-0.4	0.4	570	-1.0	(-1.2, 0.4)	$p = .307$
T30	0.4	0.4	570	1.1	(-0.3, 1.2)	$p = .271$
T40	0.1	0.4	570	0.3	(-0.6, 0.9)	$p = .788$
T50	0.3	0.4	570	0.7	(-0.7, 1.0)	$p = .468$

$\alpha \leq .025$ for Bonferroni adjustment, T=Time, Int=Intervention

Table 8.12

Linear mixed model analysis results for stratum corneum hydration at the left elbow – control versus heat intervention (n=48)

Parameter	Estimate	SE	df	t	95% CI	*p* value
Intercept	9.8	1.2	100.9	8.2	(7.4, 12.2)	$p < .001$
Heat int.	-0.7	1.7	94	-0.4	(-4.0, 2.6)	$p = .679$
T0	-0.9	0.3	570	-2.8	(-1.6, -0.3)	$p < .001$
T10	-0.6	0.3	570	-1.8	(-1.3, 0.1)	$p = .079$
T20	0.2	0.3	570	0.7	(-0.4, 1.0)	$p = .504$
T30	-0.4	0.3	570	-1.1	(-1.0, 0.3)	$p = .261$
T40	-0.2	0.3	570	-0.6	(-0.9, 0.5)	$p = .544$
T50	-0.3	0.3	570	-1.0	(-1.0, 0.3)	$p = .331$

$\alpha \leq .025$ for Bonferroni adjustment, T=Time, Int=Intervention

The LMM results for stratum corneum hydration are presented for the right elbow (Table 8.11) and left elbow (Table 8.12). No statistically significant differences in stratum corneum hydration were observed between the control and heat intervention for the right

elbow [p=.123, 95% CI (-9.3, 1.1)] or the left elbow [p=.137, 95% CI (-9.0, 1.3)]. These data suggest that the introduction of heat at the skin surface of semi-recumbent participants did not alter stratum corneum hydration scores at their elbows. Thus, the hypothesis that in the heat intervention—when compared to the control—there would be no significant increase in stratum corneum hydration at the right and left elbow (null Hypothesis Eight) failed to be rejected ($p > .025$).

The differences in stratum corneum hydration results between *T0* as compared to *T60* for the left elbow were statistically significant ($\alpha \leq .025$), suggesting change in stratum corneum hydration measures from the initial measure to the last measure.

Table 8.13

Linear mixed model analysis results for stratum corneum hydration at the right heel – control versus heat intervention (n=48)

Parameter	Estimate	SE	df	t	95% CI	*p* value
Intercept	8.7	1.8	96.7	5.0	(5.2, 12.2)	$p < .001$
Heat int.	-0.3	2.5	94	-0.1	(-5.2, 4.6)	$p = .911$
T0	0.3	0.3	570	0.9	(-0.3, 0.9)	$p = .362$
T10	0.4	0.3	570	1.2	(-0.2, 1.0)	$p = .216$
T20	0.5	0.3	570	1.5	(-0.1, 1.1)	$p = .134$
T30	0.3	0.3	570	0.1	(-0.6, 0.6)	$p = .922$
T40	0.2	0.3	570	0.7	(-0.4, 0.9)	$p = .474$
T50	0.2	0.3	570	0.6	(-0.4, 0.8)	$p = .536$

$\alpha \leq .025$ for Bonferroni adjustment, T=Time, Int=Intervention

Table 8.14

Linear mixed model analysis results for stratum corneum hydration at the left heel – control versus heat intervention (n=48)

Parameter	Estimate	SE	df	t	95% CI	*p* value
Intercept	8.9	1.8	96.3	5.0	(5.3, 12.3)	$p < .001$
Heat int.	-0.3	2.5	94	-0.1	(-5.2, 4.6)	$p = .914$
T0	-0.1	0.3	570	-0.2	(-0.7, 0.5)	$p = .805$
T10	0.2	0.3	570	0.8	(-0.3, 0.8)	$p = .418$
T20	0.0	0.3	570	0.1	(-0.5, 0.6)	$p = .888$
T30	0.4	0.3	570	1.3	(-0.2, 1.0)	$p = .192$
T40	0.1	0.3	570	0.5	(-0.4, 0.7)	$p = .622$
T50	0.1	0.3	570	0.2	(-0.6, 0.5)	$p = .832$

$\alpha \leq .025$ for Bonferroni adjustment, T=Time, Int=Intervention

The LMM results for stratum corneum hydration are presented for the right heel (Table 8.13) and left heel (Table 8.14). No statistically significant differences in stratum corneum hydration scores were observed between the control and heat intervention for the

right heel [p=.911, 95% CI (-5.2, 4.6)] or the left heel [p=0.914, 95% CI (-5.2, 4.6)]. These data suggest that the introduction of heat at the skin surface of semi-recumbent participants did not alter stratum corneum hydration scores at their heels. Thus, the hypothesis that in the heat intervention—when compared to the control—there would be no significant increase in stratum corneum hydration at the right and left heel (null Hypothesis Nine) failed to be rejected ($p > .025$).

When considering differences in stratum corneum hydration scores over time at the heels, none of the model variables reached statistical significance. This suggests that there were nil changes in stratum corneum hydration measures from the initial measure to the last measure.

Table 8.15

Linear mixed model analysis results for stratum corneum hydration at the sacrum – control versus heat intervention (n=48)

Parameter	Estimate	SE	df	t	95% CI	*p* value
Intercept	34.4	2.3	98.4	14.7	(29.7, 39.0)	$p < .001$
Heat int.	-4.5	3.3	94	-1.4	(-11.0, 2.0)	$p = .175$
T0	-2.5	0.5	570	-4.7	(-3.6, -1.4)	$p < .001$
T10	-3.0	0.5	570	-5.5	(-4.0, -1.9)	$p < .001$
T20	-2.9	0.5	570	-4.3	(-3.3, -1.2)	$p < .001$
T30	-2.5	0.5	570	-4.7	(-3.6, -1.4)	$p < .001$
T40	-1.6	0.5	570	-3.0	(-2.7, -0.6)	$p < .001$
T50	-1.0	0.5	570	-1.9	(-2.1, 0.2)	$p = .055$

$\alpha \leq .025$ for Bonferroni adjustment, T=Time, Int=Intervention

The LMM results for stratum corneum hydration are presented for the sacrum in Table 8.15. No statistically significant differences in stratum corneum hydration scores were observed between the control and hat intervention [p=0.175, 95% CI (-11.0, -2.0)]. These data suggest that the application of heat to the sacrum of semi-recumbent participants did not alter scores of stratum corneum hydration at their sacrum. Thus, the hypothesis that in the heat intervention—when compared to the control—there would be no significant increase in stratum corneum hydration at the sacrum (Hypothesis Four) failed to be rejected ($p > .025$).

With the exception of *T50* (p=.055), stratum corneum hydration at the sacrum changed significantly at all timepoints ($\alpha \leq .025$), suggesting change in stratum corneum hydration measures from the initial measures to the last measure.

8.5.2 Control versus Moisture Intervention

Tables 8.16 to 8.20 provide the LMM results for stratum corneum hydration, by anatomical testing site, for the study sample (n=48). The results detail the differences between the control versus moisture intervention, and further contrast the data gathered at baseline and interim timepoints against the final timepoint. It should be noted that in the moisture intervention, the moisture stimulus was only applied to participants' sacral skin as per the Study III protocol. However, stratum corneum measurements were attended to at participants' elbows and heels as a control, and are reported accordingly.

Table 8.16

Linear mixed model analysis results for stratum corneum hydration at the right elbow – control versus moisture intervention (n=48)

Parameter	Estimate	SE	df	t	95% CI	*p* value
Intercept	9.4	1.2	102.8	8.1	(7.0, 11.7)	$p < .001$
Moisture int.	-0.8	1.6	94	-0.3	(-3.6, 2.8)	$p = .791$
T0	-0.8	0.4	570	0.23	(-0.8, 0.6)	$p = .821$
T10	1.2	0.4	570	-0.4	(-0.6, 0.9)	$p = .676$
T20	-0.1	0.4	570	-0.6	(-0.9, 0.5)	$p = .577$
T30	0.1	0.4	570	0.3	(-0.6, 0.8)	$p = .759$
T40	-0.1	0.4	570	-0.3	(-0.9, 0.6)	$p = .738$
T50	0.3	0.4	570	0.8	(-0.4, 1.0)	$p = .403$

$\alpha \leq .025$ for Bonferroni adjustment, T=Time, Int=Intervention

Table 8.17

Linear mixed model analysis results for stratum corneum hydration at the left elbow – control versus moisture intervention (n=48)

Parameter	Estimate	SE	df	t	95% CI	*p* value
Intercept	10.1	1.1	100.3	8.9	(7.9, 12.4)	$p < .001$
Moisture int.	-0.4	1.6	94	-0.5	(-4.0, 2.3)	$p = .596$
T0	-1.0	0.3	570	-3.1	(-1.6, -0.3)	$p = .002$
T10	-1.0	0.3	570	-3.1	(-1.6, -0.3)	$p = .090$
T20	-0.5	0.3	570	-1.4	(-1.1, 0.8)	$p = .090$
T30	-1.7	0.3	570	-0.5	(-0.8, 0.4)	$p = .595$
T40	-0.4	0.3	570	-1.3	(-1.0, 0.2)	$p = .206$
T50	-0.4	0.3	570	-1.4	(-1.0, 1.2)	$p = .173$

$\alpha \leq .025$ for Bonferroni adjustment, T=Time, Int=Intervention

The LMM results for stratum corneum hydration are presented for the right elbow (Table 8.16) and left elbow (Table 8.17). No statistically significant differences in stratum corneum hydration scores were observed between the control and moisture intervention for the right elbow [p=.791, 95% CI (-3.6, 2.8)] and left elbow [p=0.596, 95% CI (-4.0, 2.3)]. Thus, the hypothesis that in the moisture intervention—when compared to the control—there would be no significant increase in stratum corneum hydration at the right and left elbow (null Hypothesis Fifteen) failed to be rejected ($p > .025$).

The differences in stratum corneum hydration results at *T60* and between *T0* and *T10* for the left elbow were statistically significant ($\alpha \leq .025$), suggesting change in stratum corneum hydration measures from the first two initial measures to the last measure. No statistically significant changes in stratum corneum hydration scores were observed over time at the right elbow. This suggests that there were no changes in stratum corneum hydration measures from the initial measure to the last measure.

Table 8.18

Linear mixed model analysis results for stratum corneum hydration at the right heel – control versus moisture intervention (n=48)

Parameter	Estimate	SE	df	t	95% CI	*p* value
Intercept	8.2	1.7	97.4	4.9	(4.9, 11.5)	$p < .001$
Moisture int.	0.4	2.4	94	0.2	(-4.2, 5.1)	$p = .870$
T0	0.3	0.3	570	1.0	(-0.3, 1.0)	$p = .327$
T10	0.1	0.3	570	0.4	(-0.5, 0.8)	$p = .713$
T20	0.6	0.3	570	1.9	(-0.0, 1.3)	$p = .058$
T30	-0.1	0.3	570	-0.4	(-0.8, 0.5)	$p = .713$
T40	-0.3	0.3	570	-1.0	(-1.0, 0.3)	$p = .327$
T50	-0.5	0.3	570	0.1	(-0.6, 0.7)	$p = .902$

$\alpha \leq .025$ for Bonferroni adjustment, T=Time, Int=Intervention

Table 8.19

Linear mixed model analysis results for stratum corneum hydration at the left heel – control versus moisture (n=48)

Parameter	Estimate	SE	df	t	95% CI	*p* value
Intercept	8.3	1.7	97.2	4.9	(5.0, 11.7)	$p < .001$
Moisture int.	0.4	2.4	94	0.2	(-4.3, 5.1)	$p = .860$
T0	-0.1	0.3	570	-0.3	(-0.8, 0.5)	$p = .730$
T10	0.2	0.3	570	0.5	(-0.5, 0.8)	$p = .594$
T20	0.2	0.3	570	0.5	(-0.5, 0.8)	$p = .638$
T30	-0.0	0.3	570	0.1	(-0.6, 0.7)	$p = .900$
T40	-0.1	0.3	570	-0.4	(-1.8, 0.5)	$p = .661$
T50	0.0	0.3	570	-1.6	(-1.2, 0.1)	$p = .110$

$\alpha \leq .025$ for Bonferroni adjustment, T=Time, Int=Intervention

The LMM results for stratum corneum hydration are presented for the right heel (Table 8.18) and left heel (Table 8.19). No statistically significant differences in stratum corneum hydration were observed between the control and moisture intervention for the right

heel [p=.870, 95% CI (4.9, 11.5)] or the left heel [p=.860, 95% CI (5.0, 11.7)]. Thus, the hypothesis that in the moisture intervention—when compared to the control—there would be no significant increase in stratum corneum hydration at the right and left heel (null Hypothesis Sixteen) failed to be rejected ($p > .025$).

When considering differences in stratum corneum hydration scores over time at participants' heels, none of the model variables reached statistical significance. This suggests that there were no changes in stratum corneum hydration measures from the initial measure to the last measure.

Table 8.20

Linear mixed model analysis results for stratum corneum hydration at the sacrum – control versus moisture intervention (n=48)

Parameter	Estimate	SE	df	t	95% CI	*p* value
Intercept	99.5	1.6	98.3	63.7	(96.4, 102.6)	$p < .001$
Moisture int.	-70.8	2.2	94	-32.4	(-75.0, -66.4)	$p < .001$
T0	-1.1	0.4	570	-3.1	(-1.8, -0.4)	$p = .002$
T10	-0.8	0.4	570	-2.4	(-1.5, -0.1)	$p = .018$
T20	-0.6	0.4	570	-1.7	(-.1.3, 0.1)	$p = .083$
T30	-0.9	0.4	570	-2.6	(-1.6, -0.2)	$p = .011$
T40	-0.5	0.4	570	-1.3	(-1.2, 0.2)	$p = .196$
T50	-0.0	0.4	570	-0.1	(-0.7, 0.7)	$p = .930$

$\alpha \leq .025$ for Bonferroni adjustment, T=Time, Int=Intervention

The LMM results for stratum corneum hydration are presented for the sacrum in Table 8.20. Statistically significant differences in stratum corneum hydration scores were observed between the control and moisture intervention [p<.001, 95% CI (-75.0, -66.4)]. These data suggest that the application of moisture did alter scores of stratum corneum hydration at the sacral region. As per Table 8.20, the stratum corneum hydration measurements collected in the control for the sacrum were 70.8 units lower than stratum corneum hydration measurements collected in the moisture intervention.

Thus, the hypothesis that in the moisture intervention—when compared to the control—there would be a significant increase in stratum corneum hydration at the sacrum (Alternative Hypothesis Ten) was supported ($p \leq .025$).

Differences in stratum corneum hydration results between *T60* and those at *T0*, *T10* and *T30* for the sacrum were statistically significant ($\alpha \leq .025$). These data suggest a change in stratum corneum hydration measures from the measures obtained in the first 30 minutes of the time period compared to the last measure at 60 minutes.

8.6 Linear Mixed Model Analyses Results for Skin Temperature by Anatomical Location

8.6.1 Control versus Heat Intervention

Tables 8.21 to 8.25 provide the LMM results for skin temperature, by anatomical testing site, for the study sample (n=48). The results detail the differences between the control versus heat intervention, and further contrast the data gathered at baseline and interim timepoints against the final timepoint.

Table 8.21

Linear mixed model analysis results for skin temperature at the right elbow – control versus heat intervention (n=48)

Parameter	Estimate	SE	df	t	95% CI	*p* value
Intercept	33.4	0.4	101.4	88.9	(32.7, 34.1)	$p < .001$
Heat int.	-1.9	0.5	94	-3.7	(-3.0, -0.1)	$p < .001$
T0	-3.5	0.1	570	-31.8	(-3.7, -3.3)	$p < .001$
T10	-2.8	0.1	570	-25.1	(-3.0, -2.6)	$p < .001$
T20	-2.6	0.1	570	-20.3	(-2.5, -2.0)	$p < .001$
T30	-1.6	0.1	570	-14.5	(-1.8, -1.4)	$p < .001$
T40	-1.2	0.1	570	-10.5	(-1.4, -1.0)	$p < .001$
T50	-0.6	0.1	570	-0.5	(-0.8, 0.4)	$p < .001$

$\alpha \leq .025$ for Bonferroni adjustment, T=Time, Int=Intervention

Table 8.22

Linear mixed model analysis results for skin temperature at the left elbow – control versus heat intervention (n=48)

Parameter	Estimate	SE	df	t	95% CI	*p* value
Intercept	33.4	0.4	101.5	89.7	(32.8, 34.3)	$p < .001$
Heat int.	-1.9	0.5	94	-3.6	(-2.9, -0.9)	$p < .001$
T0	-3.6	0.1	570	-32.0	(-3.8, -3.3)	$p < .001$
T10	-2.9	0.1	570	-25.7	(-3.1, -2.6)	$p < .001$
T20	-2.3	0.1	570	-20.7	(-2.5, -2.1)	$p < .001$
T30	-1.9	0.1	570	-16.3	(-2.0, -1.6)	$p < .001$
T40	-1.1	0.1	570	-10.0	(-1.3, -0.9)	$p < .001$
T50	-0.6	0.1	570	-5.1	(-0.8, 0.3)	$p < .001$

$\alpha \leq .025$ for Bonferroni adjustment, T=Time, Int=Intervention

The LMM results for skin temperature are presented for the right elbow (Table 8.21) and left elbow (Table 8.22). Statistically significant differences in skin temperature scores were observed between the control and heat intervention for the right elbow [$p<.001$, 95% CI (-3.0, -0.1)] and left elbow [$p<.001$, 95% CI (-2.9, -0.9)]. These data suggest that the introduction of heat to semi-recumbent participants did alter scores of skin temperature scores at their elbows. As per tables 5.29 and 5.30, skin temperature measurements gathered in the control for the right and left elbow were 1.9°C lower, respectively, than skin temperature measurements gathered in the heat intervention.

Overall, the hypothesis that in the heat intervention—when compared to the control—there would be a significant increase in skin temperature at the right and left elbow (Alternative Hypothesis Five) was supported ($p \leq .025$).

The difference in skin temperature results at *T60* for the elbows were statistically significant ($\alpha \leq .025$) compared to all other timepoints, suggesting change in skin temperature measures over time.

Table 8.23

Linear mixed model analysis results for skin temperature at the right heel – control versus heat intervention (n=48)

Parameter	Estimate	SE	df	t	95% CI	*p* value
Intercept	31.0	0.5	101.0	67.7	(30.1, 31.9)	$p < .001$
Heat int.	-1.8	0.6	94	-2.8	(-3.0, -0.5)	$p = .006$
T0	-3.6	0.1	570	-27.7	(-3.9, -3.4)	$p < .001$
T10	-2.9	0.1	570	-22.0	(-3.1, -2.6)	$p < .001$
T20	-2.4	0.1	570	-18.2	(-2.6, -2.1)	$p < .001$
T30	-1.7	0.1	570	-13.1	(-2.0, -1.5)	$p < .001$
T40	-1.3	0.1	570	-9.6	(-1.5, -1.0)	$p < .001$
T50	-0.8	0.1	570	-5.8	(-1.0, 0.5)	$p < .001$

$\alpha \leq .025$ for Bonferroni adjustment, T=Time, Int=Intervention

Table 8.24

Linear mixed model analysis results for skin temperature at the left heel – control versus heat intervention (n=48)

Parameter	Estimate	SE	df	t	95% CI	*p* value
Intercept	31.0	0.5	101.5	67.5	(30.1, 31.9)	$p < .001$
Heat int.	-1.8	0.6	94	-2.8	(-3.0, -0.6)	$p = .005$
T0	-3.6	0.1	570	-26.4	(-3.9, -3.4)	$p < .001$
T10	-2.8	0.1	570	-20.9	(-3.1, -2.6)	$p < .001$
T20	-2.1	0.1	570	-15.8	(-2.4, -2.9)	$p < .001$
T30	-1.5	0.1	570	-11.2	(-1.8, -1.3)	$p < .001$
T40	-1.1	0.1	570	-8.2	(-1.4, -0.9)	$p < .001$
T50	-0.6	0.1	570	-4.2	(-0.8, -0.3)	$p < .001$

$\alpha \leq .025$ for Bonferroni adjustment, T=Time, Int=Intervention

The LMM results for skin temperature are presented for the right heel (Table 8.23) and left heel (Table 8.24). Statistically significant differences in skin temperature scores were observed between the control and heat intervention for the right heel [p=-.006, 95% CI

(-3.0, -0.5)] and left heel [p=-.005, 95% CI (-3.0, -0.6)]. These data suggest that the introduction of heat to semi-recumbent participants did alter scores of skin temperature at their heels. As per tables 8.23 and 8.24, skin temperature measurements gathered in the control for the right and left heel were 1.8°C lower, respectively, than skin temperature measurements gathered in the heat intervention.

Thus, the hypothesis that in the heat intervention—when compared to the control—there would be a significant increase in skin temperature at the right and left heel (Alternative Hypothesis Six) was supported ($p \leq .025$).

The differences in skin temperature results at *T60* for the heels were statistically significant ($\alpha \leq .025$) compared to all other timepoints, suggesting change in skin temperature measures over time.

Table 8.25

Linear mixed model analysis results for skin temperature at the sacrum – control versus heat intervention (n=48)

Parameter	Estimate	SE	df	t	95% CI	*p* value
Intercept	37.8	0.3	104	117.1	(37.1, 38.4)	$p < .001$
Heat int.	-2.0	0.4	94	-4.6	(-2.9, -1.2)	$p < .001$
T0	-4.3	0.1	570	-38.9	(-4.5, -4.0)	$p < .001$
T10	-3.4	0.1	570	-31.1	(-3.7, -3.2)	$p < .001$
T20	-2.8	0.1	570	-25.4	(-3.0, -2.6)	$p < .001$
T30	-2.1	0.1	570	-18.9	(-2.3, -1.9)	$p < .001$
T40	-1.4	0.1	570	-13.0	(-1.6, -1.2)	$p < .001$
T50	-0.8	0.1	570	-7.0	(-1.0, -0.5)	$p < .001$

$\alpha \leq .025$ for Bonferroni adjustment, T=Time, Int=Intervention

The LMM results for skin temperature are presented for the sacrum in Table 8.25. Statistically significant differences in skin temperature scores were observed between the conditions [p<.001, 95% CI (-2.9, -1.2)]. Thus, the introduction of heat to semi-recumbent participants did alter scores of skin temperature at their sacral region. As per tables 8.24 and

8.25, skin temperature measurements gathered in the control for the sacrum were 2.0°C lower than skin temperature measurements gathered in the heat intervention.

Overall, the hypothesis that in the heat intervention—when compared to the control—there would be a significant increase in skin temperature at the sacrum (Alternative Hypothesis Seven) was supported ($p \leq .025$).

The difference in skin temperature results at *T60* for the sacrum were statistically significant ($\alpha \leq .025$) compared to all other timepoints, suggesting change in skin temperature measures over time.

8.6.2 Control versus Moisture Intervention

Tables 8.26 to 8.30 provide the LMM analysis results for skin temperature by anatomical testing site for the study sample (n=48). The results detail the differences between the control versus moisture intervention, and further contrast the data gathered at the baseline and interim timepoints against the final timepoint. It should be noted that in the moisture intervention, the moisture stimulus was only applied to participants' sacral skin as per the Study III protocol. However, skin temperature measurements in the moisture intervention were attended to at participants' elbows and heels as a control, and are reported accordingly.

Table 8.26

Linear mixed model analysis results for skin temperature at the right elbow – control versus moisture intervention (n=48)

Parameter	Estimate	SE	df	t	95% CI	*p* value
Intercept	31.6	0.3	98.9	90.9	(31.0, 32.4)	$p < .001$
Moisture int.	-0.8	0.5	94	-1.6	(-1.7, 0.2)	$p = .115$
T0	-2.2	0.1	570	-26.6	(-2.4, -2.1)	$p < .001$
T10	-1.7	0.1	570	-20.1	(-1.9, -1.5)	$p < .001$
T20	-1.5	0.1	570	-17.3	(-1.6, -1.3)	$p < .001$
T30	-1.1	0.1	570	-13.2	(-1.3, -1.0)	$p < .001$
T40	-0.8	0.1	570	-9.1	(-0.9, -0.6)	$p < .001$
T50	-0.3	0.1	570	-4.0	(-0.5, -0.2)	$p < .001$

$\alpha \leq .025$ for Bonferroni adjustment, T=Time, Int=Intervention

Table 8.27

Linear mixed model analysis results for skin temperature at the left elbow – control versus moisture intervention (n=48)

Parameter	Estimate	SE	df	t	95% CI	*p* value
Intercept	31.7	0.4	96.5	90.4	(31.0, 32.4)	$p < .001$
Moisture int.	-0.6	0.5	94	-1.3	(-1.6, 0.3)	$p = .198$
T0	-2.2	0.1	570	-36.7	(-2.4, -2.1)	$p < .001$
T10	-1.9	0.1	570	-30.9	(-2.0, -1.8)	$p < .001$
T20	-1.5	0.1	570	-34.5	(-1.6, -1.4)	$p < .001$
T30	-1.1	0.1	570	-18.5	(-1.2, -1.0)	$p < .001$
T40	-0.8	0.1	570	-12.9	(-0.9, -0.7)	$p < .001$
T50	-0.4	0.1	570	-7.3	(-0.6, -0.3)	$p < .001$

$\alpha \leq .025$ for Bonferroni adjustment, T=Time, Int=Intervention

The LMM results for skin temperature are presented for the right elbow (Table 8.26) and left elbow (Table 8.27). No statistically significant differences in skin temperature scores were observed between the control and moisture intervention condiitons for the right

elbow [p=-.115, 95% CI (-1.7, -0.2)] or the left elbow [p=-.198, 95% CI (-1.6, 0.3)]. Thus, the hypothesis that in the moisture intervention—when compared to the control—there would be no significant increase in skin temperature at the right and left elbow (Alternative Hypothesis Seventeen) was supported ($p > .025$).

The differences in skin temperature results at *T60* for the elbows were statistically significant ($\alpha \leq .025$) compared to all other timepoints, suggesting change in skin temperature measures over time.

Table 8.28

Linear mixed model analysis results for skin temperature at the right heel – control versus moisture intervention (n=48)

Parameter	Estimate	SE	df	t	95% CI	*p* value
Intercept	29.2	0.5	99.0	66.9	(28.3, 30.0)	$p < .001$
Moisture int.	-0.5	0.6	94	-0.9	(-1.7, 0.7)	$p = .383$
T0	-2.3	0.1	570	-21.9	(-2.5, -2.1)	$p < .001$
T10	-2.0	0.1	570	-18.6	(-2.2, -1.8)	$p < .001$
T20	-1.7	0.1	570	-15.9	(-2.0, -1.5)	$p < .001$
T30	-1.2	0.1	570	-11.1	(-1.4, -1.0)	$p < .001$
T40	-0.9	0.1	570	-8.2	(-1.1, -0.7)	$p < .001$
T50	-0.6	0.1	570	-0.5	(-0.8, -0.4)	$p < .001$

$\alpha \leq .025$ for Bonferroni adjustment, T=Time, Int=Intervention

Table 8.29

Linear mixed model analysis results for skin temperature at the left heel – control versus moisture intervention (n=48)

Parameter	Estimate	SE	df	t	95% CI	*p* value
Intercept	29.2	0.4	97.6	67.6	(28.4, 30.1)	$p < .001$
Moisture int.	-0.5	0.6	94	-0.9	(-1.7, 0.7)	$p = .370$
T0	-2.4	0.1	570	-27.0	(-2.6, -2.2)	$p < .001$
T10	-2.0	0.1	570	-22.7	(-2.2, -1.9)	$p < .001$
T20	-1.7	0.1	570	-18.8	(-1.9, -1.5)	$p < .001$
T30	-1.1	0.1	570	-12.6	(-1.3, -1.0)	$p < .001$
T40	-0.9	0.1	570	-9.9	(-1.1, -0.7)	$p < .001$
T50	-0.4	0.1	570	-4.6	(-0.6, -0.2)	$p < .001$

$\alpha \leq .025$ for Bonferroni adjustment, T=Time, Int=Intervention

The LMM results for skin temperature are presented for the right heel (Table 8.28) and left heels (Table 8.29). No statistically significant differences in skin temperature scores were observed between the control and moisture intervention for the right heel [p=-.383, 95% CI (-1.7, -0.7)] or the left heel [p=-.370, 95% CI (-1.7, 0.7)]. Thus, the hypothesis that in the moisture intervention—when compared to the control—there would be a significant increase in skin temperature at the right and left heel (Alternative Hypothesis Eighteen) was supported ($p > .025$).

The differences in skin temperature results at *T60* for the heels were statistically significant ($\alpha \leq .025$) compared to all other timepoints, suggesting change in skin temperature measures over time.

Table 8.30

Linear mixed model analysis results for skin temperature at the sacrum – control versus moisture intervention (n=48)

Parameter	Estimate	SE	df	t	95% CI	*p* value
Intercept	35.9	0.3	98.5	107.5	(35.3, 36.6)	$p < .001$
Moisture int.	-0.8	0.5	94	-1.7	(-1.7, 0.1)	$p = .087$
T0	-2.9	0.8	570	-37.3	(-3.0, -2.7)	$p < .001$
T10	-2.5	0.8	570	-31.9	(-2.6, -2.3)	$p < .001$
T20	-2.0	0.8	570	-26.2	(-2.1, -1.9)	$p < .001$
T30	-1.6	0.8	570	-20.3	(-1.7, -1.4)	$p < .001$
T40	-1.1	0.8	570	-14.2	(-1.3, -1.0)	$p < .001$
T50	-0.7	0.8	570	-8.9	(-0.8, -0.5)	$p < .001$

$\alpha \leq .025$ for Bonferroni adjustment, T=Time, Int=Intervention

The LMM results for skin temperature are presented for the sacrum in Table 8.30. No statistically significant differences in skin temperature scores were observed between the control and moisture intervention [p=.087, 95% CI (-1.7, 0.1)]. Thus, the application of moisture at the sacrum of semi-recumbent participants did not alter scores of skin temperature at their sacral region. The hypothesis that in the moisture intervention—when compared to the control—there would be a significant increase in skin temperature at the sacrum (Alternative Hypothesis Eleven) was not supported ($p > .025$).

The differences in skin temperature results at *T60* for the sacrum were statistically significant ($\alpha \leq .025$) compared to all other timepoints, suggesting change in skin temperature measures over time.

8.7 Summary of Linear Mixed Model Analysis Results

The aim of Study III was to test for differences in investigated skin parameters between heated and unheated skin, and between dry and wet skin at pressure-prone areas of the body. LMMs were used to test for differences in the skin parameters of interest, over time, and between i) the control and heat intervention, and ii) control and moisture intervention. As such, the results of the LMMs enabled the research team to address

Objectives 1 and 2 of Study III and research gaps IV, V and VI, as well as address the research hypotheses formulated and outlined in Chapter 6 of this thesis.

No statistically significant differences were found for erythema by condition for any of the anatomical testing sites, with the exception of the sacrum (p=.012) in the heat intervention. These results suggest that while warming the skin at the heels and elbows over one hour does not appear to impact erythema in semi-recumbent healthy adults, warming of the sacrum significantly increases sacral erythema over this period of time. Upon further inspection of the LMMs, changes in erythema by timepoint emerged among participants; however, these changes were broadly registered between the initial timepoints to the final timepoint. This observation applied to both the elbows and heels, regardless of the study condition. These data indicate that over a one hour period (particularly in the early stages) of lying semi-recumbent on a standard hospital bed, notable changes in skin redness may appear at the elbows and heels. These findings are in contrast to erythema at the sacrum. When considering sacral erythema, timepoint changes appeared to differ based on the study condition. In the moisture intervention, changes in erythema at the sacrum were similar to changes in erythema at the heels and elbows. Over the course of one hour, erythema gradually became more pronounced at the sacrum, as it did for the elbows and heels. Conversely, in the heat intervention, erythema at the sacrum increased exponentially at 10 minute intervals, indicating a much more rapid and notable magnitude of change when compared to the moisture intervention.

With the exception of the sacrum (p<.001) in the moisture intervention, no statistically significant differences were found for stratum corneum hydration by condition for any of the anatomical testing sites. These results suggest that the introduction of a moisture significantly impacted scores of stratum corneum hydration at the sacral skin of semi-recumbent healthy adults over the course one hour. Given that the heels and elbows were not exposed to the wetting agent in the moisture intervention, the results cannot be considered for the elbows and heels. In the heat intervention, aside from some occasional inconsistent time effects, there was no major change in stratum corneum hydration by timepoint for the elbows or heels over the one hour period. That is, skin warming did not appear to impact measures of stratum corneum hydration at the elbows and heels at any of the timepoints. In regard to the sacrum, stratum corneum hydration appeared to change readily at 10 minute intervals on exposure to heat and later on exposure to the wetting agent.

These changes in stratum corneum hydration over time, however, were much more pronounced in the heat intervention.

Statistically significant differences emerged for skin temperature for the elbows ($p<.001$), heels ($p=-.006$) and sacrum ($p<.001$) between the control and heat intervention. These results imply that the introduction of heat at pressure-prone areas significantly impacts (increases) skin temperature over the course of one hour. No statistically significant differences in skin temperature scores were observed between the control and moisture intervention for any of the anatomical testing sites. These results imply that skin wetness, over a period of one hour, does not appear to impact skin temperature at the elbows, heels or sacrum of semi-recumbent healthy adults. When considering differences in skin temperature by timepoint, significant changes were observed ($\alpha \leq .025$) at the 10 minute intervals over the one hour period, at all anatomical testing sites among the study participants in both the heat and moisture intervention conditions.

8.8 Chapter Summary

This chapter has presented the results of the LMM analyses for the Study III participant sample. It has detailed the steps taken to screen and prepare these data, and has outlined the preliminary assumption checking analyses. The results of LMM analyses have been reported for each skin parameter variable by anatomical location for the sample. The next chapter will review the findings of this research with respect to the research aim, objectives and hypotheses, and contrast the results with evidence from the literature. The results of the current study will then be interpreted in relation to their clinical significance and their potential contribution to clinical practice. Finally, the strengths and limitations of this research will be considered and recommendations for future research will be proposed.

CHAPTER 9
Discussion

9.1 Introduction

In this chapter, the results of this research are discussed and interpreted, and their significance with reference to the published existing literature examined. The chapter begins with a summary of all three studies to provide context to the discussion. Subsequently, the chapter compares and interprets the descriptive data gathered in Studies I and II and in the control of Study III against the current literature. This is followed by a discussion pertaining to skin manifestations in response to the brief intermittent pressure-loading and alterations in skin microclimate in healthy adults; these discussions are considered separately for skin manifestations in response to heat, and in response to moisture. The clinical implications of the study results are considered as they relate to the study hypotheses and, more broadly, to the research aims and objectives. The strengths and limitations of this research are then identified. Finally, recommendations for clinical practice and in relation to further research are provided.

9.2 Appraisal of Skin Manifestations Associated with Pressure Injury Risk

Although many PIs are preventable, they continue to represent a substantial burden in healthcare institutions, and have been associated with increased mortality and morbidity rates (Khor et al., 2014; Lozano et al., 2012; NPUAP, 2019). The presence of PIs has a significant impact on affected individuals' quality of life (Gorecki et al., 2009), and from an economic perspective, it is considered more expensive to treat than to prevent PIs (Spetz et al., 2013). PI prevention is critical in reducing the prevalence of these chronic wounds. Although the international guidelines for PI prevention are available and many initiatives have been implemented to reduce their incidence, the number of individuals suffering from PIs globally remains unacceptably high.

To date, an assessment of skin status in individuals at risk of PI development is the primary element of prevention measures (Moore & Patton, 2019; Posnett & Franks, 2008; Yang et al., 2020). The National Pressure Injury Advisory Panel, European Pressure Injury Advisory Panel, and Pan Pacific Pressure Injury Alliance (NPUAP-EPUAP-PPPIA) Global 2019 Clinical Practice Guideline states that the condition of the patient's skin overlying a bony prominence can be informative of impending PI (NPUAP-EPUAP-PPPIA, 2019).

Thus, an appraisal of the skin condition represents a focal point in the contemporary PI prevention care pathway used to inform the care provided (Dealey et al., 2015; Falloon et al., 2018; NPUAP, 2019), and is considered a key nursing responsibility (NPUAP, 2019). Nurses have traditionally been taught to inspect the patient's skin overlying the pressure-prone areas for erythematous discolouration (NPUAP, 2019; Romanelli et al., 2006; Vanderwee et al., 2007). In addition to the assessment of erythema, an assessment of skin moisture and skin temperature forms an integral component of contemporary PI risk assessment (NPUAP, 2019). The parameters of skin moisture and skin temperature represent key variables in the theories and evidence pertaining to the concept of the skin microclimate (Agrawal & Chauhan, 2012; Clark et al., 2010).

9.3 The Theory of Skin Microclimate in Pressure Injury Development

A review of the literature in Chapter 3 explored the contextual elements of the skin microclimate as a concept with theoretical and empirical associations with PI development. The term 'microclimate' describes the local skin environment (Clark et al., 2010), encompassing the parameters of skin moisture and/or humidity and skin temperature at the skin-support surface interface (Gefen, 2011; Roaf, 2006). It should be noted that the parameter of skin moisture can refer both to the moisture at the skin surface, or to the moisture within the deeper tissue layers. In this thesis, the concept of skin moisture is denoted by the descriptor 'stratum corneum hydration', reflective of the water content within the outermost layers of the skin. As outlined in Chapter 3 of this thesis, stratum corneum hydration is regarded as a biophysical skin property (Clarys & Barel, 1995) and a relevant dimension in the contemporary understanding the construct of the skin microclimate (Derler et al., 2012; Yusuf et al., 2015).

Skin microclimate data, although increasingly discussed and referred to as a fundamental element in the maintenance of skin and tissue integrity, is seldom reported in the literature. The characteristics of an 'optimal' skin microclimate are still a matter of ongoing research. Few human-based studies (Bates-Jensen et al., 2007; Borzdynski, McGuiness & Miller, 2016; Denzinger et al., 2020; Falloon et al., 2018; Huimin et al., 2017; Kottner et al., 2015; Kottner et al., 2018; Lachenbruch, 2005 ; Lichterfeld-Kottner et al., 2021; Sae-Sia, Wipke-Tevis & Williams, 2005; Schwartz et al., 2020; Zhong et al., 2008) report biophysical measures of skin microclimate data at pressure-prone areas using immobilisation protocols. However, there is generally limited published longitudinal in-vivo data regarding the phenomena of how sub-optimal microclimate conditions influence skin properties of

erythema, stratum corneum and skin temperature at pressure-prone areas of loaded skin. This paucity of the literature presents a fertile area for further research and has provided the impetus for this thesis.

In view of the limited volume of in-vivo studies and an absence of a large longitudinal monitor of microclimate skin changes in healthy adults, this study contributes rich descriptive and inferential data, albeit limited to one hour as an initial excursion to ensure participant safety. The thesis investigations have provided valuable insight into the behaviour of skin colour and skin microclimate properties, over time, at weight-bearing regions of the body of healthy adult volunteers lying in the supine and semi-recumbent positions. In doing so, this research has generated clinically important benchmark data against which skin parameter results of future related studies can be compared and contrasted. Furthermore, this research contributes new evidence to inform the current evidence-base regarding skin sensing technologies as a novel and innovative method of capturing PI-associated skin parameters in healthy adults.

9.4 Overarching Research Aim

The overarching aim of this thesis was to explore skin manifestations over time, with intermittent pressure-loading, in response to alterations in the skin microclimate (i.e., controlled manipulation of heat and moisture at the posterior aspect of the body) at common PI predilection sites of healthy adults with no existing skin damage. Specifically, this thesis sought to add evidence describing the biophysical properties of skin colour (erythema and melanin) and skin microclimate (skin moisture and skin temperature) and changes occurring during one hour of immobilisation with periodic pressure off-loading.

9.5 Summary of Research Studies

This research was undertaken in two phases. The first phase, referred to as Studies I and II, represented elements of pilot investigations that informed the protocol of the second and final research phase, referred to as Study III. A repeated measures descriptive-correlational pilot design was employed in Studies I and II, while Study III was conducted using a repeated measures quasi-experimental approach. The next sections provide a summary of the results gathered in Studies I and II (Section 9.5.1) and in Study III (Section 9.5.2) of this research.

9.5.1 Studies I and II

Studies I and II are reported as a publication in Chapter 5 of this thesis, as published by the *Journal of Tissue Viability*, titled "Brief intermittent pressure off-loading on skin microclimate in healthy adults – A descriptive-correlational pilot study" (Borzdynski, Miller, Vicendese & McGuiness, 2021).

A convenience sample of 41 healthy adults with no existing skin damage, and with a mean age of 27.9 years (*SD*=10.6) participated in Studies I and II. Due to the exploratory nature of this study, a formal sample size calculation was not performed. However, the expected number of participants was considered sufficient for the relevant statistical analyses. The aim of studies I and II was to examine microclimate changes to the skin as a result of pressure-loading over a one hour period. Skin measurements were taken immediately after off-loading of the elbows, heels and sacrum whilst participants lay supine; 0° incline (Study I) and then semi-recumbent; 45° incline (Study II) over one hour. To allow a comparison, repeated measures were taken across uninterrupted and interrupted methods. The detection of minimal differences in skin measurements between methods would confirm that intermittent pressure off-loading did not interfere with the study outcome measures. Conversely, moderate to large differences would require the research team to continue to explore avenues to measure skin parameters without altering skin status as a by-product of the data collection method.

Objective 1 of Studies I and II was to establish a descriptive library of skin parameter scores—as measured over time and across pressure-prone areas—during intermittent pressure off-loading. Descriptive data were generated to profile measures of erythema, melanin, stratum corneum hydration and skin temperature at baseline (*T0*), interim 10 minute intervals (*T10*, *T20*, *T30*, *T40*, *T50*) and endpoint at 60 minutes (*T60*) of the supine and semi-recumbent conditions. Among the 41 participants recruited into Studies I and II, a large range of skin parameter scores was noted at all anatomical testing sites, suggesting large regional variation in scores between participants. Conversely, when considering scores within participants, a minimal to moderate range of scores was observed at all anatomical testing sites, suggesting minimal to moderate regional variations for individuals.

When examining the distributional characteristics of skin parameters, the elbows and heels were typically characterised by lower readings of erythema, stratum corneum hydration and skin temperature when compared to the sacrum, suggesting differences by anatomical

location. On each measurement occasion, the sacrum registered the highest scores. However, when considering mean scores for skin parameters, there was minimal variation in erythema and stratum hydration between the anatomical testing sites. Conversely, moderate variation in mean scores of skin temperature emerged between the anatomical testing sites. Descriptive data gathered in Studies I and II have been compared and contrasted to subsequent data gathered in Study III. These comparisons have been integrated into the later discussions in this chapter.

Objective 2 of Studies I and II was to compare differences in skin parameter scores between timepoints (baseline to final) over a one hour period as measured at pressure-prone areas amongst participants. At the same time, this approach sought to address the methodological gap by examining the agreement between the two methods (uninterrupted and interrupted) when attending to skin parameter measurements. Strong agreement between the interrupted and uninterrupted method was observed, with intra-class correlations coefficients (ICCs) ranging from 0.72 to 0.99 (Study I - supine) and 0.62 to 0.99 (Study II - semi-recumbent). These ICC results indicated that brief off-loading (interruptions) to enable skin parameter measurement do not alter skin readings; thus, the researchers could proceed with some confidence regarding the use of this protocol as an effective research technique to monitor changes in skin status in the subsequent study. Furthermore, the absence of any measurable difference in melanin scores in the Studies I and II participant sample led to the omission of the melanin component in Study III. However, baseline melanin measures were attended to at the anatomical testing sites to describe the Study III sample.

Objective 3 of Studies I and II sought to compare differences in skin parameter scores at anatomical testing sites when assessed in the supine position (Study I) and semi-recumbent position (Study II); two common positions adopted in the care of hospitalised patients. The datasets in Study I and Study II were comparable; the results indicated that the evaluated lying positions had a negligible effect on skin parameters, with the endpoint results of the semi-recumbent condition only marginally higher when compared to endpoint results of the supine condition. Clinically, these findings suggest that the incline of the head of the bed may not impact skin changes associated with PI development. Thus, with consideration to the results of Studies I and II, relying on skin changes as an assessment criteria for the success of any changes in patient positioning is not supported. Nevertheless, in view of the one hour limit of pressure-loading in the current study, it is possible that compounding effects over time may exist and could be further investigated. Overall, negligible variation

between the evaluated positions led the research team to consolidate the focus on the semi-recumbent position only in Study III.

Overall, it can be inferred from the Studies I and II data that brief off-loading of pressure-prone areas, over a period of one hour, in healthy adults does not appear to impact skin parameter outcomes. Thus—when translating these findings into clinical practice—implementing a 10 minute off-loading regime for healthy patients lying continuously in the supine and semi-recumbent positions would not appear to clinically alter PI risk derived from the indirect effects of skin microclimate. However, considering that endpoint measures tended to be higher compared to baseline measures for all skin parameters, it is possible that beyond a one hour period, a person may be approaching the threshold of skin changes that may place vulnerable skin at risk of PI development.

9.5.2 Study III

Study III of this research program was conducted with the aim of exploring the impact of skin microclimate on clinical manifestations often attributed to PI development in healthy adults. Specifically, the study sought to examine changes in repeated measures of erythema, stratum corneum hydration and skin temperature in response to the application of thermal and moisture stimuli at pressure-prone areas of the body. A series of research hypotheses were further generated pertaining to Study III of this research. These hypotheses will be explored against the Study III results later in the chapter.

A convenience sample of 48 healthy adults with no existing skin damage, and with a mean age of 26.8 years (*SD*=9.5), participated in a quasi-experimental study. Of the Study III sample, 46% of people (n=22) were former participants who had previously participated in Studies I and II. The data trends across Studies I and II and Study III were relatively comparable between these 22 participants. This has not only conveyed consistency and validity in the overall results, but has also imparted further confidence in the study findings. Due to the exploratory nature of this study, a formal sample size calculation was not performed. However, the expected number of participants was considered sufficient for the relevant statistical analyses.

Skin parameters (in the order of skin temperature, stratum corneum hydration, and erythema) were gathered at the elbows, heels and sacrum of healthy adults lying in a semi-recumbent position (45° incline) on a standard hospital bed. Measures were taken

immediately after off-loading of the pressure-prone areas, every 10 minutes, over one hour. Skin parameters were considered across three conditions: condition 1: control, condition 2: heat intervention, and condition 3: moisture intervention. In-between measurements, the skin of the investigational areas was barred to and in direct contact with the support surface.

All participants advanced consecutively, in a non-randomised order, through the study conditions in a single visit and, in effect, acted as their own matched control. A standard fifteen minute pragmatic washout interval separated each condition to ensure a robust baseline. In the heat intervention, participants lay posteriorly on an electric heated blanket; as such, the posterior aspect of the body was exposed to warmth generated from the blanket over one hour. In the moisture intervention, participants lay posteriorly with their sacrum positioned directly over an absorbency pad saturated with 0.9% sodium chloride (normal saline). In effect, the sacral area was exposed to a wetted surface over one hour.

Objective 1 of Study III was to establish a descriptive library of skin parameter scores—as measured over time and across pressure-prone areas—during intermittent pressure off-loading in a control, heat intervention, and moisture intervention. Descriptive data were generated to profile erythema, stratum corneum hydration and skin temperature measures at baseline, interim (10 minute) and final timepoints of each condition. Among the 48 participants recruited in Study III, a large range of skin parameter scores was noted at all anatomical testing sites in each condition, suggesting large regional variation in scores between participants. Conversely, when considering scores across time for individuals, a minimal to moderate range of scores was observed at all anatomical locations, suggesting minimal to moderate regional variations for individual participants. When examining the distributional characteristics of skin parameters, the elbows and heels were typically characterised by lower readings of erythema, stratum corneum hydration and skin temperature when compared to the sacrum, implying differences by anatomical location. On each measurement occasion, the sacrum registered the highest scores. These findings were consistent between Studies I-III, as will be discussed in later sections of this chapter.

On average, skin parameter scores tended to be lowest at baseline and highest at endpoints for all anatomical testing sites and in all study conditions. Minimal oscillations, with relatively nil discernable patterns, emerged between interim timepoints for erythema at the elbows and heels in all study conditions. However, in the heat intervention, a more evident pattern emerged, with erythema rising steadily over the interim intervals, and with

endpoint scores notably higher than baseline scores. Similarly, skin temperature displayed uniform increase between consecutive timepoints. Erythema and skin temperature scores were highest at the sacral region. In both conditions, minimal oscillations emerged between interim timepoints for stratum corneum hydration. However, when considering the sacrum in the moisture intervention, maximum readings (range: 97-99 units) were noted across timepoints. These results have been interpreted in Section 9.7 of this chapter.

Objectives 2 and 3 of Study III was to assess for differences in skin parameter scores, over time, and across pressure-prone areas between the control and heat intervention (Objective 2) and the control and moisture intervention (Objective 3). Linear mixed models (LMMs) were used to test for differences in the skin parameter scores, over time, and between the control and intervention conditions. Analyses were repeated for each anatomical testing site. The results of the LMM analyses enabled the research team to determine if the research hypotheses were supported or disproved.

Statistically significantly differences for erythema were found between the control and heat intervention for the sacrum (p=.012). Statistically significant differences were also evidenced for skin temperature between the control and heat intervention for the elbows (p<.001), heels (p=.006) and sacrum (p<.001). Additionally, statistically significant differences emerged for stratum corneum hydration between the control and moisture intervention for the sacrum (p<.001). No statistically significant differences were found for erythema and stratum corneum hydration between the control and heat intervention for the elbows and heels.

When considering changes in skin parameters by timepoint, significant changes emerged for erythema ($\alpha \leq .025$) and stratum corneum hydration ($\alpha \leq .025$) at the sacrum in heated skin across all timepoints. Significant changes by timepoint also emerged for stratum corneum hydration in wet skin at the sacrum ($\alpha \leq .025$). Finally, significant changes for skin temperature emerged at the elbows, heels and sacrum ($\alpha \leq .025$) at the 10 minute off-loading intervals, over the one hour, in both heated skin and wet skin among the study participants.

The inferential findings of Study III of this thesis both supported and disproved the research hypotheses that were previously outlined in Chapter 6. These findings and how they have addressed these research hypotheses will be considered in subsequent sections of this chapter.

9.6 Comparison of Skin Manifestation Findings with the Current Literature

This section provides a comparison and interpretation of the descriptive data gathered in the Studies I and II and in the control of Study III. Comparisons are made utilising the existing related literature. Due to negligible variation in skin parameter scores between the uninterrupted and interrupted conditions of Studies I and II, only the interrupted condition has been compared and contrasted against the control of Study III. For organisational purposes, these discussions are presented by skin parameter; erythema (Section 9.6.1), stratum corneum hydration (Section 9.6.2) and skin temperature (Section 9.6.3).

9.6.1 Skin Colour (Erythema)

Erythema displayed an oscillatory behaviour between timepoints with no discernible pattern. When considering the mean ranges for erythema measurements at the anatomical testing sites across Studies I-III, relatively similar trends emerged among participants. The mean range of erythema scores for the elbows was recorded between 21.3–22.8 units (Study I), 23.1–27.2 units (Study II) and 20.3–21.9 units (Study III - control). For the heels, the mean range of erythema scores was documented between 23.0–24.4 units (Study I), 24.3–27.8 units (Study II), and 22.8–23.6 units (Study III - control). For the sacrum, the mean range of erythema scores was noted between 26.9–30.3 units (Study I), 30.1–35.0 units (Study II) and 30.7–32.5 units (Study III - control). These data indicate consistent trends in mean erythema scores at the elbows, heels and sacrum from the perspective of all three studies. Study III had slightly higher mean erythema scores with a smaller range compared to Studies I and II.

The documented mean ranges for erythema at the elbows, heels and sacrum among participants in Studies I-III correspond to the category of 'minimal erythema' (reference range: 19-36 units) as per the SD202 Skin Diagnostic Measurement Interpretation Guide (Barrett-Hill, 2015) (see Figure 6.2 in Chapter 6). The findings of minimal erythema registered at the skin surface in the majority of participants may be explained by the fact that the sample were comprised of predominantly younger, healthy adults without any skin compromise. However, should the same study be replicated in a sample comprising aged and acutely ill individuals, variations in erythema intensity could be observed.

When considering the minimal and maximum scores of erythema scores across the anatomical testing sites, all three studies were characterised by a wide range of scores (range: Study I: 3.0–61, Study II: 3.0–63.0, Study III: 2.0–79.0), with higher maximum scores

observed at the sacrum. When interpreted against the SD202 Skin Diagnostic Measurement Interpretation Guide for erythema (reference range: 1-99 units), these findings imply that participants had 'nil erythema' (reference range: 1-18 units), 'minimal erythema' (reference range: 19-36 units), 'diffused redness' (reference range: 37-54 units) and 'high erythema' (reference range: 55-80 units) (Barrett-Hill, 2015). Clinically, this variability in scores suggests that 'hues' of erythema may vary among healthy adults, and suggests the need for an individualised regional assessment of erythema at pressure-prone areas of the body. This recommendation will be discussed in Section 9.10.1 of this chapter.

There is an evident lack of comparative data within the literature against which the result of the current study could be considered. In studies that employ similar protocols, discrepancies related most often to standardisation of skin measures. Furthermore, immobilisation protocols and participant positioning differed between studies (Kottner et al., 2015; Soares et al., 2019), limiting data comparisons.

An example of a study with the most comparability to the current research is by Kottner et al. (2015) that emerged concurrent to the early phases of the current research program. Kottner et al. (2015) examined pressure-loading and site-dependent changes for erythema, stratum corneum hydration and skin temperature among healthy ambulant older females (n=20) with a mean age of 70 years (*SD*=3.4) (age range: 60-80 years). The possible effects of pressure-loading on skin status were considered at the sacrum and heel. Only the right heel was investigated given that empirical evidence suggests symmetry of cutaneous properties between contralateral body parts. Under controlled conditions, the participants followed a standardised immobilisation protocol of 90 minutes in the supine position on a standard hospital bed/mattress. After baseline skin parameters had been recorded, the participants were instructed to remain supine for 90 minutes. After 90 minutes, participants underwent a twenty minute recovery (washout) period.

It should be noted that in Kottner et al.'s (2015) study, the immobilisation protocol was thirty minutes longer in comparison to the immobilisation protocol employed in the current studies. Unlike the current study, Kottner et al.'s (2015) study did not employ brief interruptions to measure skin parameters, rather, the immobilisation interval was continuous. Furthermore, erythema measurements at the anatomical testing sites were registered using the Mexameter MX 18 (Courage+Khazaka, GmbH), which is a different device model to the SD202 Mexameter (Courage+Khazaka, GmbH) used in the current research. Although the

measurement principles between both Mexameter devices are consistent, the outputted erythema indices are not standardised to the same scale. That is, the erythema indices outputted by the Mexameter MX 18 are interpreted against a scale ranging from 0-999, whereas the erythema indices outputted by the SD202 Mexameter are interpreted against a scale ranging from 1-99. Thus, for the purpose of this discussion, comparisons in erythema results gathered in the current studies and comparable study are discussed in relative terms.

When considering baseline and end erythema scores at the sacrum in Kottner et al.'s (2015) study, erythema was recorded at 174 units at baseline and rose to 277 units (indicating an increase of 59%) after 90 minutes of immobilisation in the supine position. After the twenty minute washout period, erythema decreased to 236 units, indicating an incomplete return to baseline. Although direct comparisons between erythema data gathered in Kottner et al.'s (2015) study and the current studies cannot be made, results are comparable when considered in relative terms; that is, in both studies, erythema at the sacrum was observed to increase between baseline and end scores. However, in contrast to the current studies, erythema did not completely return to baseline following the twenty minute washout period. Furthermore, in the current studies, the difference between baseline and end scores for erythema at the sacrum was smaller. To illustrate, in Study I (supine), the mean erythema value at the sacrum was recorded at 26.9 units at baseline, and rose to 30.3 units after one hour, indicating a limited mean difference of 3.4 units (an increase of 12.6%). Similarly, in Study II (semi-recumbent), the mean erythema value at the sacrum was recorded at 30.1 units at baseline, and rose to 35.0 units after one hour, indicating a limited mean difference of 4.9 units (an increase of 16.3%). In Study III, the mean erythema value at the sacrum was recorded at 30.7 units at baseline, and rose to 32.5 units after one hour, indicating a mean difference of only 1.8 units (an increase of 5.8%).

When considering baseline and end scores of erythema at the right heel in Kottner et al.'s (2015) study, erythema was recorded at 166 units at baseline and rose to 221 units after 90 minutes of immobilisation in the supine position (indicating an increase of 33.1%). After the twenty minute washout period, erythema was recorded at 207 units, indicating that it had not completely returned to the first baseline of 166 units (denoting a decrease of 6.3%). These findings for erythema at the heels are in contrast to the current study findings; that is, erythema at the heels remained relatively stable from baseline to end over the one hour, returning to near baseline after the conclusion of the fifteen minute washout period. It is possible that the observed discrepancies in skin parameter results between the current studies

and Kottner et al.s (2015) study could have been attributed to protocol variances, for example, when considering the duration of the immobilisation intervals.

9.6.2 Stratum Corneum Hydration

Similarities emerged between participants when considering mean ranges for stratum corneum hydration scores at the anatomical testing sites in all three studies. The mean range of stratum corneum hydration scores for the elbows was recorded between 6.2-7.0 units (Study I), 6.2-6.7 units (Study II) and 8.1-9.5 units (Study III). For the heels, the mean range of stratum corneum hydration scores was noted between 7.0-7.6 units (Study I), 6.3-6.6 units (Study II), and 8.0-9.3 units (Study III). For the sacrum, the mean range of stratum corneum hydration scores was noted between 16.9-22.5 units (Study I), 19.7-25.5 units (Study II) and 27.1-29.3 units (Study III). These data indicate that across all three studies, the mean ranges for stratum corneum hydration were relatively consistent for the elbows and heels. When considering the sacrum, the mean stratum corneum hydration values were moderately higher in Study III, and had a smaller range when compared to Studies I and II.

The findings of this research indicate that, on average, there was minimal variation in mean scores of stratum corneum hydration among study participants. When interpreted against the SD202 Skin Diagnostic Measurement Interpretation Guide (Barrett-Hill, 2015) (see Figure 6.2 in Chapter 6), participants' skin can be described as 'very dry' (reference range: 0-23 units) at the elbows and heels, and as 'dry' (reference range: 24-45 units) at the sacral region. Thus, the results for stratum corneum hydration for this particular sample suggest that participants, on average, presented with under-hydrated skin overlying common pressure-prone areas of the body. Furthermore, the extremities were noted to be slightly more under-hydrated than the trunk of the body.

The finding of under-hydrated skin overlying the investigated bony prominences may be attributed to the distinct skin and tissue properties at these regions. The bony prominences represent weight-bearing regions that continually bear mechanical loads as individuals interact with the environment (Boyle et al., 2019). Thus, the skin overlying the bony prominences tends to be stiffer and drier (Dobos et al., 2015), with a relatively thicker stratum corneum layer (Cichowitz et al., 2009). A thicker stratum corneum acts as a protective mechanism thought to enhance tissue tolerance to continual mechanical loads (Derler, 2012). Furthermore, the stratum corneum overlying the bony prominences tends to have significantly fewer sebaceous glands (Ludovici et al., 2018) and, therefore, limited

intracellular lipids (Imokawa et al., 1989; Imokawa et al., 1991), which further contributes to a drier skin surface. Thus, it is likely that that deficiencies in intracellular lipids at participants' elbows, heels and sacrum had accounted for lower stratum corneum hydration readings registered by the SD202 Skin Diagnostic Corneometer. As such, it can be argued that the present study findings of under-hydrated skin at the elbows and heels were attributed to the skin morphology and mechanical properties of these anatomical regions.

Of particular note is the wide range of minimum and maximum scores of stratum corneum hydration at the anatomical testing sites across all three studies (Study I: 0-61.0 units; Study II: 0-67.0 units; Study III: 0-77.0 units). These ranges, when interpreted against the SD202 Skin Diagnostic Measurement Interpretation Guide for stratum corneum hydration (reference range: 0-99 units), imply that participants had variably dry and well-hydrated skin. When considered on a scale ranging from 0-99 units, these results align with a clinical interpretation of 'very dry skin' (reference range: 0-23 units), 'dry skin' (reference range: 24-45 units), 'optimal hydration' (reference range: 46-62 units) to a 'higher level of hydration' (reference range: 65-82 units) (Barrett-Hill, 2015). This variability in scores of stratum corneum hydration may suggest clinical benefit of attending to an individualised assessment of moisture-related skin changes in semi-reclined healthy adults, as will be discussed in Section 9.10 of this chapter.

The descriptive results for stratum hydration recorded in the control of Study III have been compared to those gathered by Kottner et al. (2015), whose study was overviewed in the previous section. In brief, Kottner et al. (2015) investigated loading and site-dependent changes for stratum corneum hydration in ambulant healthy older females (n=20) over a 90 minute immobilisation interval in the supine position. In both Kottner et al.'s (2015) study and the current studies, stratum corneum hydration was registered at the anatomical sites using the Courage & Khazaka Corneometer CM 85 (Courage & Khazaka Electronic GmbH) (range: 0-99 units). Thus, comparisons for stratum corneum hydration results from the current studies are possible.

When considering stratum corneum hydration at the sacrum in both Kottner et al.'s (2015) 90 minute immobilisation protocol and the 60 minute immobilisation protocol with brief interruptions (off-loading) in the current studies, stratum corneum hydration remained relatively stable over the loading intervals. In Kottner et al.'s (2015) study, the mean stratum corneum hydration value at the sacrum was recorded at 40.0 units at baseline and rose to 45.1

units after 90 minutes of immobilisation in the supine position. After the twenty minute washout period, the mean stratum corneum hydration value decreased negligibly to 43.1 units and was relatively within baseline.

Compared to Kottner et al.'s (2015) study, the current studies (also employing an immobilisation interval that was thirty minutes shorter), revealed that the sacrum exhibited moderately lower mean scores of stratum corneum hydration, but a wider variation between baseline and end; that is, mean baseline and end scores were recorded at 16.9 units and 22.5 units, respectively (Study I), at 19.7 units and 25.5 units, respectively (Study II), and at 27.1 units and 29.3 units, respectively (Study III). Thus, the degree of change in scores of stratum corneum hydration between baseline and end was inconsistent between the current studies and the comparable study. However, the mean scores for all studies, when considered against the SD202 Skin Diagnostic Interpretation Guide (Barrett-Hill, 2015) (See Figure 6.2 in Chapter 6), correspond to the categories of 'very dry skin' (reference range: 0-23 units) and 'dry skin' (reference range: 24-45 units).

In Kottner et al.'s (2015) study, the mean stratum corneum hydration value at the right heel was recorded at 17.8 units at baseline and rose to 19.2 units after 90 minutes of immobilisation in the supine position. After the twenty minute washout period, the mean stratum corneum hydration value decreased negligibly to 18.5 units, being relatively close to the baseline value.

Measurements of stratum corneum hydration documented at the heels in the current studies, when compared against those of Kottner et al. (2015), were lower and displayed a more limited increase over time. To illustrate, in Study I, the mean baseline and end scores for stratum corneum hydration at both the right and left heel were recorded at 7.0 units and 7.6 units, respectively. In Study II, the mean baseline and end scores at the right heel were recorded at 5.7 units and 6.5 units, respectively, and at 6.3 units and 6.6 units at the left heel, respectively. In Study III, the mean baseline and end scores at the right heel were recorded at 8.6 units and 8.9 units, respectively, and at 9.1 units and 8.4 units at the left heel, respectively. Despite these variances, when considered against the SD202 Skin Diagnostic Interpretation Guide (Barrett-Hill, 2015), the mean stratum corneum hydration scores at the heels in both the current and comparable study correspond to the same category of 'very dry skin' (reference range: 0-23 units). These findings have been interpreted in terms of their clinical significance in Section 9.7.3 of this chapter.

Comparisons with regard to stratum corneum hydration between Kottner et al.'s (2015) study and the current studies must be viewed with caution when considering the differences pertaining to the sample characteristics. In Kottner et al.'s (2015) study, the mean age of the sample was 70 years (*SD*=3.4), comprising older females, whilst in the current study, the mean age of the sample was 26.8 years (*SD*=9.5), comprising predominantly younger females and males. It is, therefore, possible that the skin parameter scores documented in the comparable study, in some respect, may have been influenced by participant intrinsic factors, such as age-related skin changes.

9.6.3 Skin Temperature

The trends for the mean ranges for skin temperature scores at the anatomical testing sites across Studies I-III were relatively consistent among the sample. The mean range of skin temperature scores for the elbows was recorded between 28.9–30.4°C (Study I), 28.9–30.8°C (Study II) and 28.6–30.7°C (Study III). For the heels, the mean range of skin temperature scores was documented between 27.6–29.2°C (Study I), 27.7–30.1°C (Study II) and 26.4–28.5°C (Study III). For the sacrum, the mean range of skin temperature scores was noted between 30.8–33.4°C (Study I), 30.9–33.6°C (Study II) and 32.3–35.0°C (Study III). These data indicate that across all studies, the mean scores for skin temperature were similar for the elbows and heels. In contrast, the sacral skin yielded moderately higher mean skin temperature scores in Study III when compared to Studies I and II.

All three studies in this research evidenced moderate ranges for minimum and maximum scores of skin temperature (Study I: 24.1–35.5°C; Study II: 24.3–35.9°C; Study III 21.8-38.5°C) across the anatomical testing sites. Higher maximum scores of skin temperature were apparent at the sacrum when compared to the elbows and heels. As such, it can be inferred from the skin temperature data that, on average, the trunk (sacrum) presented with moderately higher skin temperatures than the extremities (elbows and heels). These regional variations in the skin temperature scores amongst study participants have yielded important recommendations for clinical practice that will be discussed in Section 9.10 of this chapter.

While there is no formal reference range for skin temperatures of the human body (which can vary widely between person to person and by anatomical region) (Armstrong & Lavery, 1997), mean skin temperature values are often reported in thermoregulation studies with human subjects. The descriptive data for skin temperature in the current studies, with consideration to the anatomical distribution of scores, are in relative agreement with the skin

temperature data reported by Millington (1983). Assuming neutral ambient temperatures of 21–25°C and a core temperature of ~37.0°C, Millington (1983) reported that, on average, skin temperature at the trunk of the human body would vary between 33.5–36.9°C, and between 23.5–35.6°C at the extremities. These regional skin temperature characteristics are congruent with those reported in the current studies; that is, higher skin temperatures were noted at the trunk of the body than at the extremities. To illustrate, in the current studies, the range of mean skin temperature scores at the sacrum (anatomically situated at the trunk of the body) was recorded between 30.8–33.9°C (Study I), 30.9–33.6°C (Study II) and 32.3–35.0°C (Study III), which was moderately higher than the range for mean skin temperature scores recorded at the elbows and heels (Study 1: 28.9–30.4°C and 27.6–29.2°C, respectively, Study II: 28.9–30.8°C and 27.7–29.6°C, respectively, and Study III: 28.6–30.7°C and 26.4–28.5°C, respectively). When compared to the range of mean skin temperature scores at the trunk of the body reported by Millington (1983), the mean range for skin temperature in the current studies was notably smaller when considering the extremities.

Overall, the control data for skin temperature gathered in Studies I-III suggest that participants experienced, on average, moderately higher skin temperatures at the core of the body than the peripheries, supporting the results of the aforementioned thermoregulation study by Millington (1983). These findings are expected when considering the distribution of adipose tissue at the core of the body compared to the peripheries. Adipose tissue is known to store more heat (Arner et al., 2011; Picard, 2011); thus, anatomical regions with a greater abundance and relative thickness of adipose tissue tend to exhibit a warmer skin surface (Agrawal & Pardasani, 2016; Chait & den Hartigh, 2020). Anatomically, the sacrum is situated at the core of the human body (Martin et al., 2003), and has propensity to store more adipose tissue than the peripheries (van Pelt et al., 2002). It can, therefore, be argued that higher skin temperatures at the participants' sacrum were attributed to the composition to the underlying tissue. Furthermore, the median sacral artery supplies the sacral vertebra (Scanlon & Sanders, 2012) which, in this study, would have enabled an increase in the amount of the heat transported via the blood to the skin surface (Heising & Werner, 1985).

The descriptive results for skin temperature values gathered in the current studies are relatively consistent with the results of a recent study by Soares et al. (2019). In their cross-sectional study, Soares et al. (2019) examined mean values for skin temperature at pressure-prone areas of the body in hospitalised surgical patients (n=230) with a mean age of 69.3 years (*SD*=6.9) (age range: 18–88 years) and low risk of PI development. An infrared

thermometer was used to measure skin temperature at the elbows, heels and sacrum of participants once a day. The sacrum presented with the highest mean skin temperature of 34.2°C (range: 25.7–39.0°C), while the elbows and heels presented with the lowest mean skin temperature of 31.2°C (range: 22.0–35.2°C) and 28.2°C (range: 21.9–35.5°C), respectively. The mean skin temperature score of 34.2°C at the sacrum, as reported by Soares et al. (2019), was moderately higher when compared to the mean baseline skin temperature scores at the sacrum in the current studies (Study I: 30.8°C , Study II: 30.9°C , Study III: 32.3°C). In Soares et al.'s (2019) study, the mean skin temperature score of 31.2°C at the elbows was also moderately higher when compared to the mean baseline skin temperature scores at the elbows in the current studies (Study I: 28.9°C, Study II: 28.9°C, Study III: 28.6°C). The mean skin temperature score of 28.2°C at the heels, as reported by Soares et al. (2019), was slightly higher when compared to the mean baseline skin temperature scores at the heels in the current studies (Study I: 27.6°C, Study II: 27.7°C, Study III: 26.4°C).

The results for minimum and maximum scores of skin temperature at the sacrum, as reported by Soares et al. (2019), are relatively comparable with the results of the current studies. In Soares et al.'s (2019) study, the range for minimum and maximum scores for skin temperature at the sacrum was noted at 25.7–39.0°C. In the current studies, the range of minimum and maximum scores for skin temperature for the sacrum was noted between 29.0–35.5°C (Study I), 29.7–35.3°C (Study II), and 26.3–38.5°C (Study III). Thus, Studies I-III had higher minimum skin temperature scores but lower maximum skin temperature scores. In general, the minimum and maximum range for sacral skin temperature scores was generally smaller in the current studies than the comparable study.

The range of minimum and maximum scores for skin temperature for the elbows in the current studies was also comparable to those reported by Soares et al. (2019) (i.e., 22.0–35.2°C). To illustrate, in the current studies, the range of minimum and maximum scores for skin temperature for the right elbow was noted between 26.0–32.9°C (Study I), 26.3–32.5°C (Study II), and 22.0–35.9°C (Study III). For the left elbow, the range of minimum and maximum scores for skin temperature was documented between 25.9–32.8°C (Study I), 26.3–32.7°C (Study II), and 24.0–35.8°C (Study III). Thus, the current studies had higher minimum skin temperature scores, but relatively comparable maximum skin temperate scores. The range of minimum and maximum skin temperature scores was generally more

limited for the elbows in Studies I and II while comparable in Study III when considered against those of Soares et al. (2019).

Comparisons emerged for the range of minimum and maximum scores for skin temperature at the heels in the current studies and Soares et al.'s (2019) study. To illustrate, in the current studies, the range of minimum and maximum scores for skin temperature for the right heel was documented between 24.1–33.1°C (Study I), 24.6–32.3°C (Study II), and 21.9-34.4°C (Study III). For the left heel, the range of minimum and maximum scores for skin temperature was documented between 23.8–33.2°C (Study I), 24.3–32.5°C (Study II), and 21.8-34.9°C (Study III). When compared to the range for minimum and maximum scores for skin temperature at the heels (i.e., 21.9–35.5°C) reported by Soares et al. (2019), Studies I and II had slightly higher minimum skin temperature scores and relatively comparable maximum skin temperature scores. The range of skin temperature scores for the heels was generally more limited in Studies I and II, although comparable in Study III when considered against those reported by Soares et al. (2019).

The descriptive results for sacral and heel skin temperature reported in the current studies are relatively aligned with those gathered by Kottner et al. (2015). Kottner et al. (2015) investigated loading and site-dependent changes for skin temperature in ambulant healthy older females (n=20) over a 90 minute immobilisation interval in the supine position. Skin temperature was measured in degrees Celsius (°C) using a skin thermometer (Courage+Khazaka Electronic GmbH) based on the infrared technique. Thus, comparisons for skin temperature data gathered by Kottner et al. (2015) can be made against the skin temperature data gathered in the current studies.

In Kottner et al.'s (2015) study, the mean skin temperature value at the sacrum was recorded at 31.8°C at baseline and rose to 34.0°C after 90 minutes of immobilisation in the supine position, indicating a mean difference of 2.2°C from baseline. After the twenty minute washout period, the mean skin temperature value was recorded at 32.0°C, indicating an almost complete return to baseline of 31.8°C.

In Study I, the mean baseline and end scores at the sacrum were recorded at 30.8°C and 33.4°C, respectively, indicating a mean difference of 2.6°C. In Study II, the mean baseline and end scores at the sacrum were recorded at 30.9°C and 33.6°C, respectively, indicating a mean difference of 2.7°C. In Study III, the mean baseline and end scores at the

sacrum were recorded at 32.3°C and 35.0°C, respectively, indicating a mean difference of 2.7°C. These mean differences are comparable to the mean difference of 2.2°C between baseline and end over 90 minutes noted in Kottner et al.'s (2015) study. In Study III, the mean skin temperature scores were similar when compared to Kottner et al.'s (2015) study, however, mean endpoint values were slightly higher in Study III (over a shorter loading interval). In Studies I and II, the mean baseline skin temperature values were slightly lower when compared to Kottner et al.'s (2015) study, however, relatively similar in terms of the mean endpoint scores.

In Kottner et al.'s (2015) study, the mean skin temperature value at the right heel was recorded at 28.6°C at baseline and rose to 30.3°C after 90 minutes of immobilisation in the supine position, indicating a mean difference of 1.7°C from baseline to end. After the twenty minute washout period, this mean skin temperature value remained unchanged at 30.3°C, indicating that it had not completely returned to the baseline of 28.6°C. Although the starting baseline mean value for skin temperature at the heels was slightly lower in the current studies when compared to those reported by Kottner et al. (2015), the elevation in skin temperature at the heels was comparable. To illustrate, in both Studies I and II, the mean baseline and end scores for skin temperature for the right and left heel were recorded at 27.6°C and 29.2°C, indicating a mean difference of 1.6°C. In Study II, the mean baseline and end scores for skin temperature at the right heels were recorded at 27.7°C and 30.1°C, respectively, and at 27.7°C and 29.6°C at the left heel, respectively. Thus, mean differences of 2.4°C and 1.9°C were recorded for the right and left heel, respectively. In Study III, the mean baseline and end scores for both the right and left heel were recorded at 26.4°C and 28.5°C, respectively, reflecting a mean difference of 2.1°C. These mean differences are comparable to the mean difference of 1.7°C between baseline and end over 90 minutes noted in the comparable study.

In both Kottner et al.s (2015) study and the current studies, skin temperature at the anatomical testing sites gradually increased in response to time. In the current studies, elevations in skin temperature were also noted upon each 10 minute measurement timepoint at all the anatomical testing sites. A plausible explanation for these incremental elevations in skin temperature may be related to heat accumulation at the skin-support surface interface. Under normal circumstances, the body constantly produces a certain amount of heat that is naturally transferred, via convection, from the skin surface to the environment (Flam et al., 1995). When a patient lies on a support surface, this natural flow of heat is impeded,

restricting heat dissipation (Ootsuka et al., 2015) and contributing to skin warming (Patel et al., 1999, Yusuf et al., 2015). Thus, in the context of the current studies, the occlusive and/or insulating effects of the support surface (foam mattress) would have limited thermal diffusivity and air convection, leading to 'heat trapping' at the skin-support surface interface (Webb et al., 2015; Werner et al., 1992). This cumulative effect of heat trapping—compounded by loading time—may have accounted for the gradual elevations in skin temperature observed in the participant sample.

In both Kottner et al.'s (2015) study and the current studied, similarities emerged with regard to the washout interval data for the sacrum. In the current studies and comparable study, skin temperatures at the sacrum returned to near baseline after the fifteen and twenty minute off-loading intervals, respectively. This notable return to baseline was most likely attributed to the effect of heat dissipation (Brienza & Geyer, 2005) upon the skin's exposure to the ambient environment during the off-loading. The washout interval data in the current study has not been examined in this thesis; however, the contribution of these data to nursing care may be an important consideration of future related studies.

9.7 Discussion of Skin Manifestations in Response to Brief Intermittent Pressure-Loading and Skin Microclimate in Healthy Adults

This section explores and interprets the results of the linear mixed models (LMM) analyses for Study III, and outlines how these results address the research hypotheses. As previously outlined, Study III of this research program was conducted with the aim of exploring the impact of skin microclimate on clinical manifestations often attributed to PI development in healthy adults. Specifically, the study sought to examine changes in measures of erythema, stratum corneum hydration and skin temperature in response to the application of thermal and moisture stimuli at the skin-support surface interface. A series of research hypotheses were generated pertaining to the anticipated skin changes and differences that would be observed in the sample.

As outlined in Chapter 7 of this thesis, LMM analyses reported changes for skin parameters by timepoint (i.e., baseline, interim and end measures) and differences by condition (i.e., differences between the control versus heat intervention, and between the control versus moisture intervention) at each of the anatomical testing sites. The data estimates generated by the LMMs provided an indication of the magnitude of change within interim timepoints of data collection for the control versus intervention conditions. For

organisational purposes, the discussion concerning skin manifestations in response to brief intermittent pressure off-loading and skin microclimate is presented separately for heat-induced skin manifestations (Section 9.7.1) and moisture-induced skin manifestations (Section 9.7.2).

9.7.1 Skin Manifestations in Response to Heat

9.7.1.1 Skin Colour (Erythema) Changes in Response to Heat by Timepoint

It can be inferred from the LMM findings that the application of a thermal stimulus over one hour at the posterior aspect of three important PI predilection areas (the elbows, heels and sacrum) of semi-reclined healthy adults was sufficient to have evidenced some significant changes ($\alpha \leq .025$) in skin colour (erythema) over time. These changes in erythema by timepoint emerged at both the elbows and heels among participants, suggesting that over a one hour period of lying semi-recumbent on a standard hospital bed, notable changes in skin redness may appear at the extremities. When taking into the sacrum, erythema increased exponentially at most 10 minute intervals, displaying a much more rapid and notable magnitude of change compared to the elbows and heels. These trends are also reflected by the descriptive data (See Table 7.3 in Chapter 7), where endpoint erythema measures tended to be higher when compared to baseline measures.

The observed increases in sacral erythema, over time, among participants may be attributed to the initiation of thermoregulatory processes in response to skin warming. As previously outlined in Section 6.10.7.2 of Chapter 6, participants lay on a heated blanket that was set to the desired heat-generating level of 30°C for the one hour period of data collection. Over time, 'heat trapping' may have occurred at the skin-support surface interface (Webb et al., 2015; Werner et al., 1992). This gradual accumulation of heat would have triggered vasodilation of the underlying blood vessels, raising cutaneous perfusion (Brienza & Geyer 2005; Johnson et al., 1986). Increased cutaneous perfusion—mediated by a local vasodilatation in response to sustained heat—enhances heat dissipation from the skin surface, and is an essential part of the heat-defence response (Almeida et al., 2006). Ootsuka et al. (2015) further state that warming the skin to 45°C can substantially increase cutaneous perfusion. However, the findings of Ootsuka et al. (2015) are derived from animal-based studies and, as such, their interpretation in an in-vivo context requires some caution.

One common clinical manifestation of increased cutaneous perfusion tends to be reflected as reactive hyperaemia (or a 'red flush') at the skin surface (Vanderwee et al.,

2007). In the context of this research, it is likely that this red flush was captured by the SD202Skin Diagnostic Mexameter and registered as erythema. It is important to reinforce that reactive hyperaemia is considered a normal reaction to pressure and differs from an abnormal response of non-blanching hyperaemia (Del Bene, 1990; Vanderwee et al., 2007). Thus, it can be argued that in the current research, erythema—as registered post-pressure relief—was reactionary in nature. Furthermore, nil participants experienced non-blanching hyperaemia upon tactile assessment at the interim off-loading intervals. These results were expected given that the sample comprised healthy individuals without a predisposition to skin damage. Furthermore, the stipulated immobilisation interval did not exceed the reported PI skin damage threshold of two hours (Black et al., 2011; Defloor et al., 2005; Lyder, 2003; Peterson et al., 2013; Reddy et al., 2006; Vanderwee et al., 2007). However, whether the reported elevations in sacral erythema are clinically pertinent when transferred to vulnerable patient cohorts requires further investigation. Future studies are needed to explore the clinical relevance of incremental elevations in erythema at the sacrum in both healthy and non-healthy patient cohorts.

An important observation of this study relates to the considerable variation in hues of erythema detected at the anatomical testing sites among individual participants. Measures of erythema, on average, appeared more marked at the sacral region than at the elbows and heels. This observation appears logical when considering that the sacrum, anatomically, has a wider surface area and more prominent vasculature (blood supply) than the extremities (Moore & Dalley, 2014; Scanlon & Sanders, 2010). In this research, skin warming at the sacrum would have initiated local vasodilatory processes; that is, widening of the underlying sacral blood vessels connecting to the dermal capillaries. These events, in turn, may have enhanced cutaneous blood flow, resulting in a 'red flush' at the skin surface. As previously outlined, it can be suggested that this red flush was registered as erythematous discoloration by the SD202 Skin Diagnostic Mexameter in some participants.

In addition to reporting changes for erythema by timepoint, the results of the LMM analyses also reported differences in erythema by condition, over the course of one hour, in semi-recumbent participants. These findings are discussed below.

9.7.1.2 Skin Colour (Erythema) Changes in Response to Heat by Condition

It was hypothesised that erythema would increase among participants in the heat intervention at the elbows (Alterative Hypothesis One), heels (Alterative Hypothesis Two)

and sacrum (Alterative Hypothesis Three) more than would be observed in participants in the control. Results indicated that the introduction of heat to semi-recumbent participants had marginal effect on erythema at the elbows and heels and, as such, Alternate Hypotheses One and Two were not supported ($p > .025$). Conversely, the introduction of heat induced greater changes in erythema at the sacrum of semi-recumbent participants, supporting Alternative Hypothesis Three ($p \leq .025$).

It can be inferred from the above findings that heated sacral skin of healthy semi-reclined individuals, when compared to unheated sacral skin, displayed significant differences in erythema over the one hour. In other words, the introduction of heat to the sacral skin had a notable and direct impact on skin colour. The same finding was not evident at the elbows or heels, suggesting that the introduction of heat, over one hour, at the elbows and heels of semi-reclined healthy adults does not impact erythema. As outlined in the previous section, such discrepancies in the anatomical distribution of erythema may have been attributed to more prominent vasculature underlying the skin at the trunk of the body as opposed to the extremities (Guyton, 2005).

As outlined in Chapter 1 of this thesis, the most commonly-recognised indicator of possible existing or impending skin and soft tissue damage is erythema; however, erythema may be a normal reaction to pressure (manifested as reactive hyperaemia), or an abnormal reaction to pressure (manifested as non-blanching hyperaemia) (Vanderwee et al., 2007). Non-blanching hyperaemia is thought to represent an inflammatory response, and inflammation has been reported as the earliest sign of impending ulceration (Bates-Jensen, 2007; Schubert & Fagrell, 1989; Sprigle, Linden & Riodan, 2003; Wong et al., 2011). To date, the presence of non-blanching hyperaemia is considered a traditional marker of initial stages of ischemic tissue damage preceding Stage I PI (Bergstrand et al., 2010). In the context of the present research, it can be argued that the registered hues of erythema—post-pressure relief—were reactionary in nature. Thus, the erythematous hues detected at participants' skin surface should not be immediately perceived as post-inflammatory erythema, but rather as a normal physiological response to heating concurrent to the application of brief consecutive pressure. Clinically, however, the results do highlight the importance of managing heat at the sacral skin-support surface interface of semi-recumbent healthy adults.

The guideline recommendations for the *Prevention and Treatment of Pressure Ulcers Clinical Practice* (EPUAP-NPUAP-PPPIA, 2019) advise that clinicians attend to regular visual inspection for erythematous discolouration in individuals at risk of PI development. Based on the reported elevations in sacral erythema occurring over the one hour immobilisation period among participants in this research, there may be clinical benefit of attending to an hourly biophysical assessment of erythema at heated sacral skin. However, it is challenging to interpret the elevations in sacral erythema in the context of merely one hour; that is, it is unknown whether erythema would remain stable, progress or perhaps reverse if investigated beyond the one hour immobilisation interval.

9.7.1.3 Stratum Corneum Hydration Changes in Response to Heat by Timepoint

Upon inspection of the LMMs, aside from some occasional inconsistent time effects in the heat intervention, there were no major changes in stratum corneum hydration by timepoint for the elbows or heels over the one hour period. Thus, the influence of time was not apparent for measures of stratum corneum hydration at the elbows and heels. For the sacrum, stratum corneum hydration appeared to change readily at 10 minute intervals on exposure to heat; with the exception of *T50* (p=.055), stratum corneum hydration changed significantly at all timepoints ($\alpha \leq .025$), suggesting change in stratum corneum hydration measures from the initial measures to the last measure at the sacral region of the body.

In addition to reporting changes for stratum corneum hydration by timepoint, the results of the LMM analyses also reported differences in stratum corneum hydration by condition, over the course of one hour, in semi-recumbent participants. These findings are discussed below.

9.7.1.4 Stratum Corneum Hydration Changes in Response to Heat by Condition

It was hypothesised that in the heat intervention—when compared to the control—there would be no increase in stratum corneum hydration at the elbows (Null Hypothesis Eight), heels (Null hypothesis Nine) and sacrum (Null Hypothesis Four). As no differences between the heat intervention and control were detected at the elbows, heels and sacrum, these Null Hypotheses failed to be rejected ($p > .025$). As such, it can be concluded the introduction of heat over one hour to the elbows, heels and sacrum of semi-recumbent participants was not sufficient to have directly or consistently altered stratum corneum hydration. This implies that attending to hourly measurements of stratum corneum hydration at the elbows, heels and sacrum may be of limited clinical benefit in healthy adults.

Changes in stratum corneum hydration emerged by timepoint in response to the application of heat at the sacrum, however, when considering differences by condition (i.e., differences between the control versus heat intervention) at the sacrum, the introduction of heat did not impact hydration measures.

9.7.1.5 Skin Temperature Changes in Response to Heat by Timepoint

Significant changes ($\alpha \leq .025$) in skin temperature were observed at the 10 minute intervals over the one hour period in the heat intervention, for all anatomical testing sites, among the study participants. These findings translate to evident increases in skin temperature, registered at every 10 minute interim interval, in response to the application of heat to the posterior aspect of the body of healthy semi-recumbent participants. This influence of time was apparent from baseline to end, reflected by consistent and uniform elevations in skin temperature over the one hour. It can, therefore, be suggested that an hourly skin temperature monitoring regime at the bony prominences of the elbows, heels and sacrum in healthy adults lying in a semi-recumbent position may identify clinically important cutaneous (thermal) changes that may be linked to important clinical manifestations.

The results of the LMM analyses for skin temperature changes by timepoint are supported by the descriptive data for skin temperature changes between baseline and endpoints (see Table 7.7 in Chapter 5). Upon inspection of the dataset, a positive linear relationship between the variables of skin temperature and time was evident. This upward trajectory of skin temperature data was most apparent at the sacrum. Of particular note is the maximum skin temperature score recorded at 42.8°C at the sacrum. This maximum score, when compared to the minimum baseline skin temperature score of 26.2°C at the sacrum indicates a considerable increase in sacral skin temperature in response to heat applied over the one hour period of data collection. When considering mean differences in skin temperature scores between baseline and end, on average, the skin at the sacrum was 3.0°C higher in the heat intervention when compared to the control. To illustrate, for unheated skin, the mean sacral skin temperature value at baseline and end was recorded at 32.3°C and 35.0°C, respectively, denotating a mean difference of 2.7°C. For heated skin, the mean sacral skin temperature value at baseline and end was recorded at 32.7°C and 38.4°C, respectively, indicating a mean difference of 5.7°C.

The mean difference of 2.7°C for skin temperature between baseline and end of unheated skin is a particularly salient finding when considered against a study by Sae-Sia et

al. (2005). Sae-Sia et al. (2005), in their study of 17 hospitalised neurologically-impaired patients positioned supine and then laterally, found that mean sacral skin temperature in patients who developed a PI was higher (p=.001) when compared with those who did not develop a PI. Regardless of the resting position, the mean sacral skin temperature in patients who developed a PI was 37.2°C, compared to 36.0°C in those who did not develop a PI. A key message of Sae-Sia et al.'s (2005) study is that sacral skin temperature had increased by 1.2°C in 24 to 96 hours before a PI had developed. This finding may hold particular clinical relevance when considered against the current study data, which evidenced a mean skin temperature difference of 2.7°C between baseline and end over the one hour loading period with brief interruptions. However, it is important to reinforce that in the current study, skin temperature changes were observed over a considerably shorter loading interval in comparison to Sae-Sia et al.'s (2005) study, restricting data comparisons. It is unknown if skin temperature would have remained stable or continued to increase if investigated beyond the one hour limit of data collection.

The reported elevations in skin temperature in response to the application of heat at the pressure-prone areas of the body may hold clinical relevance in view of the literature indicating associations between increased skin temperature and early onset skin damage (Kottner et al., 2015; Patel et al., 1999; Sae-Sia et al., 2005; Yusuf et al., 2015). The literature reviewed in Chapter 3 of this thesis postulated that an elevation in skin temperature precipitates skin and soft tissue damage (Gefen et al., 2011; Jan et al., 2012; Yusuf et al., 2015; Zeevi et al., 2018) and is positively correlated with tissue inflammation (Gefen et al., 2011). Indeed, sustained elevations in skin temperature have been found to contribute to a reduction of the cohesive strength of the stratum corneum (Wu et al., 2006). However, to the researchers' best knowledge, there are currently no standardised skin and soft tissue damage thresholds for skin temperature at PI predilection areas of the body. In the absence of these thresholds, it is unclear if the exponential rise in skin temperature—as registered at the anatomical testing sites in the sample—can be regarded as clinically meaningful. As such, despite statistically significant skin temperature changes emerging over one hour, it cannot be inferred if these changes translate into *clinically significant* changes. Further research is required to examine if skin temperature could be used as a reliable predictor of skin and soft tissue damage associated with PI development.

In addition to reporting changes for skin temperature by timepoint, the results of the LMM analyses also reported differences in skin temperature by condition, over the course of one hour, in semi-recumbent participants. These findings are discussed below.

9.7.1.6 Skin Temperature Changes in Response to Heat by Condition

It was hypothesised that skin temperature would increase among participants in the heat intervention at the elbows (Alterative Hypothesis Five), heels (Alterative Hypothesis Six), and sacrum (Alterative Hypothesis Seven) more than it would increase among participants in the control. Given that significant skin temperature differences between the heat intervention and control were detected at the elbows, heels and sacrum, the above hypotheses were supported in relation to all anatomical locations ($p \leq .025$). Thus, it can be concluded that incremental increases in skin temperature at the elbows, heels and sacrum of semi-recumbent participants over time were related to the application of a thermal stimulus, supporting Alternative Hypotheses Five, Six and Seven of this research.

It can be extrapolated from the above findings that warming of the skin over one hour can significantly impact skin temperature ($p < .001$) at the elbows, heels and sacrum of participants lying in a semi-recumbent position. In view of the above findings and the one hour limit of pressure-loading in the current study, it is possible that compounding effects over time may exist. It can also be inferred that beyond a one hour period, a person could be approaching a skin temperature threshold that may place vulnerable skin at risk of damage. Nevertheless, a larger sample size and an extended skin monitoring duration is required to fully examine the veracity of this statement.

Another important observation of this study is that skin temperature scores returned to near baseline following the fifteen minute washout period that was used as a pragmatic interval between the control and heat intervention. Thus, the findings imply that a fifteen minute pressure off-loading regime could be an effective strategy to promote skin cooling of previously-loaded anatomical regions. However, it is unknown whether this relatively rapid skin recovery rate would have been observed if the study were to be replicated with a sample of individuals susceptible to PI development.

The detection of significant thermal changes—as captured using biophysical instruments at participants' skin surface—presents an important consideration with regard to current clinical methods of skin temperature appraisal in PI risk assessment. As outlined in

Chapter 4 of this thesis, the NPUAP (2019) recommends that clinicians use touch to detect localised elevations in skin warmth over pressure-prone areas of the body. Although localised areas of elevated skin warmth may be felt merely by the touch of the hand, this technique is highly interpretive and only as sensitive as the hand of the assessor (Pompei, 1996). In other words, skin temperature is relative to the assessor; a cold hand will indicate a warm skin surface and a warm hand will indicate a cooler skin surface (Levine at al., 2018; Pompei, 1996). It may, therefore, be challenging to accurately detect and interpret variations in relation to skin temperature using touch alone. Ultimately, skin temperature should be considered as a continuum measured in small units. However, from the perspective of a subjective assessment, skin temperature assessment, at best, serves as a gross measurement. Overall, the findings of the current study have yielded an important recommendation for future related research that will be proposed in Section 9.11 of this chapter.

9.7.2 Conclusion

The results of the LMMs pertaining to differences in erythema, stratum corneum hydration and skin temperature in heated versus unheated skin conditions have generated three important observations. First, the introduction of heat over one hour at the elbows, heels and sacrum of semi-recumbent healthy adults may not be sufficient to significantly alter stratum corneum hydration. These findings also suggest limited clinical benefit of attending to hourly biophysical measurements of stratum corneum hydration at warmed skin at the elbows, heels and sacrum. Second, significant changes in erythema may start to emerge over one hour in warmed sacral skin of semi-recumbent healthy adults. Finally, some significant thermal skin manifestations may start to appear over one hour in warmed skin at the elbows, heels and sacrum of semi-recumbent healthy adults. These findings may suggest the need for interventions aimed at managing heat accumulation at the skin-support surface interface or bedbound patients.

The findings of this research also allude to the feasibility of adopting biophysical skin monitoring systems to detect for changes in erythema and skin temperature at pressure-prone areas of the body of patients with a predisposition to PI formation. An hourly biophysical skin assessment regime target at the elbows, heels and sacrum could identify important skin manifestations that may not always be identified visually. However, given the impost of attending to hourly skin measurements (for example, in the form of disruptions to patient sleep patterns or to the provision of routine nursing care), a less frequent or less obtrusive monitoring regime would be required.

9.7.3 Skin Manifestations in Response to Moisture

9.7.3.1 Skin Colour (Erythema) Changes in Response to Moisture by Timepoint

Upon inspection of the LMMs, there were no major changes in skin colour (erythema) over the one hour at the elbows, heels or sacrum in the moisture intervention. Given that the elbows and heels were not exposed to moisture as per the Study III protocol, the above findings were expected. There was nil evidence of major changes in erythema, over time, in response to external moisture at the sacrum. This suggests that hourly skin-loading with brief interruptions does not impact erythema in wet/damp sacral skin.

In addition to reporting changes for erythema by timepoint, the results of the LMM analyses also reported differences in erythema by condition, over the course of one hour, in semi-recumbent participants. These findings are discussed below.

9.7.3.2 Skin Colour (Erythema) Changes in Response to Moisture by Condition

It was hypothesised that in the moisture intervention—when compared to the control—there would be no increase in erythema at the elbows (Null Hypothesis Twelve), heels (Null Hypothesis Thirteen) and sacrum (Null Hypothesis Fourteen). Based on the results of this research, these null hypotheses failed to be rejected (p>025). When considering results for the sacrum, it can be inferred that wet/damp sacral skin does not experience PI-related skin manifestations over a one hour immobilisation period with brief interruptions.

9.7.3.3 Stratum Corneum Hydration Changes in Response to Moisture by Timepoint

There were no notable changes in stratum corneum hydration results over the one hour period at the elbows and heels among the study participants. These results were expected given that the elbows and heels were not exposed to the wetting agent during the data collection. Thus, the results for the elbows and heels served as control parameters. In regard to wet/damp sacral skin, measures of stratum corneum hydration appeared to change readily at each 10 minute interim interval over the one hour. These findings suggest clinical benefit of biophysical monitoring wet/damp sacral skin for changes in stratum corneum hydration as an indicator of possible moisture-associated skin changes. Such a detection system could be particularly relevant in the nursing care of bedbound incontinent and/or diaphoretic patients at risk of skin damage associated with increased moisture. However, further research is needed to establish the thresholds for biophysical measures of stratum corneum that could serve as a trigger for potential moisture-induced skin damage.

In addition to reporting changes for stratum corneum hydration by timepoint, the results of the LMM analyses also reported differences in stratum corneum hydration by condition, over the course of one hour, in semi-recumbent participants. These findings are discussed below.

9.7.3.4 Stratum Corneum Hydration Changes in Response to Moisture by Condition

It was hypothesised that there would be no differences in stratum corneum hydration at the elbows between the moisture intervention and the control at the elbows (Null Hypothesis Fifteen) and heels (Null Hypothesis Sixteen). Based on the results, these hypotheses failed to be rejected ($p > .025$). These results were expected given that the elbows and heels were not exposed to moisture during the data collection. Thus, the results for the elbows and heels served as control parameters.

It was hypothesised that stratum corneum hydration would increase among participants in the moisture intervention at the sacrum (Alterative Hypothesis Ten) more than would be observed in participants in the control. Significant differences between the moisture intervention and control were detected at the sacrum and, as such, Hypothesis Ten was supported ($p \leq .025$).

Based on the above findings, it can be concluded that changes in stratum corneum hydration at the sacrum of semi-recumbent participants were directly related to the presence of moisture at the skin-support surface interface. In other words, direct exposure of sacral skin to moisture over the one hour immobilisation period (with intermittent off-loading) was sufficient to have significantly altered skin status. The trends in data for stratum corneum hydration at the sacrum, as generated by the LMMs, are supported by the descriptive data (See Table 7.6 in Chapter 7). These data, when considered against the SD202 Skin Diagnostic Measurement Interpretation Guide (Barrett-Hill, 2015) (see Figure 6.2 in Chapter 6), indicate that the sacral skin, on average, presented as 'over-saturated' (range: 83-99).

The finding of over-saturation of the stratum corneum upon immersion in moisture may be clinically meaningful when considered in the context of the literature reviewed in Chapter 3. The prolonged over-hydration and/or excessive moisture uptake by the skin has been reported to induce epidermal softening (Harrow & Mayrovitz, 2014; Mojumdar et al., 2017). Epidermal softening has been shown to influence the mechanical properties of the stratum corneum (Schwartz & Gefen, 2020), gradually leading to a reduction of the skin's

tensile strength and compromised barrier function (Falloon et al., 2018; Gray et al., 2002; Ichikawa-Shigeta et al., 2014). Over time, these events substantially raise the skin's susceptibility to friction-induced damage (Yoshimura et al., 2015; Xu et al., 2020). Similarly, other related studies have postulated that continual moisture at the skin surface induces microstructural changes (Wu et al., 2006) and can raise the coefficient of friction (Gerhardt et al., 2008), which can accelerate the progression of skin breakdown.

The association between moisture and superficial skin damage has been previously postulated by Kwong et al. (2009) and Cutting et al. (1999), who reported that prolonged skin moisture can create alterations in the mechanical properties of the epidermis, leading to skin maceration; a well-known pre-curser for PI onset. Grey, Harding and Enich (2006) state that macerated skin has been found to be five times more likely to become ulcerated than dry skin due to its diminished ability to withstand compressive and shearing forces. In addition, a previous related study by Borzdynski, McGuiness and Miller (2016) indicated that higher levels of skin moisture at the sacrum of bedbound geriatric aged-care residents (n=38) were significantly associated with PI risk. Taken together, the above published literature and the present study finding reinforce the importance of nursing interventions targeted at adequate moisture-control at wet/damp sacral skin. The recommendation for clinical practice in relation to this study finding has been proposed in Section 9.10 of this chapter.

As per the Study III protocol, to ensure measurement consistency, skin measurements were taken as soon as the participant lay semi-recumbent on the laboratory bed. In the moisture intervention, this process—whilst ensuring positioning consistency and that the measurement accuracy was not impacted by the angle of the skin-measuring probe—resulted in the brief exposure of the sacrum to the wetting agent immediately prior to baseline measures. Although brief, the impact was absolute. These findings, whilst limiting the capacity for baseline comparisons, have generated an important observation; that is, even the briefest contact of the skin with moisture appears to have resulted in immediate 'soaking' or 'water uptake effect' at the level of the stratum corneum.

The finding of instantaneous skin saturation upon exposure to moisture may hold clinical relevance when extrapolated to patients with urinary and/or faecal incontinence, whose skin may be subjected to extended periods of exposure to moisture originating from urine and/or faeces (Beeckman et al., 2017; Gray et al., 2012). As discussed in Chapter 2 of this thesis, the sustained contact of the skin with moisture can precipitate skin softening and

maceration (Gray et al., 2011; Schnelle et al., 1997), rendering the skin vulnerable to the compounding effects of pressure, shear and friction (Bliss et al., 2015; Langemo et al., 2011). These findings, when considered together with those of the current study, raise an important recommendation for practice that will be presented in Section 9.10 of this chapter.

9.7.3.5 Skin Temperature Changes in Response to Moisture by Timepoint

When considering changes in skin temperature by timepoint in the moisture intervention, significant changes were observed ($\alpha \leq .025$) at the 10 minute intervals over the one hour period at all anatomical testing sites among the study participants. These findings suggest that skin temperature notably increased over time (from baseline to end) over the one hour in participants lying in a semi-recumbent position.

In addition to reporting changes for skin temperature by timepoint, the results of the LMM analyses also reported differences in skin temperature by condition, over the course of one hour, in semi-recumbent participants. These findings are discussed below.

9.7.3.6 Skin Temperature Changes in Response to Moisture by Condition

It was hypothesised that there would be no differences in skin temperature between the moisture intervention and the control at the elbows (Null Hypothesis Seventeen) and heels (Null Hypothesis Eighteen). As no differences were detected at the elbows and heels, Null Hypotheses Seventeen and Eighteen failed to be rejected ($p > .025$). This is an expected result given that the elbows and heels served as control sites and were not exposed to moisture as per the study protocol; thus, consistency between the results was expected.

In contrast to the elbows and heels, it was hypothesised that skin temperature would increase among participants in the moisture intervention at the sacrum (Alterative Hypothesis Eleven) more than it would increase in the control. Given that no significant differences in skin temperature between the moisture intervention and control were detected at the sacrum, Alterative Hypothesis Eleven was not supported ($p > .025$). These findings indicate that the application of moisture at the sacrum did not impact skin temperature. However, it is important to highlight that in this study, the documented timepoint changes with regard to skin temperature in the presence of moisture were limited to one hour. Thus, it is unknown to what extent skin temperature would have been impacted by the presence of moisture, had the duration of loading exceeded the one hour limit.

As previously outlined, in the moisture intervention of Study III, a solution of normal saline was applied to participants' sacral skin to test for the effect of increased moisture on skin manifestations. This solution was maintained at an average room temperature of 22.9˚C (range: 20.6-25.8˚C) which, when compared to the temperature of urine in humans, is considerably lower. The temperature of the urine within the first four minutes of voiding ranges from 32.2 to 37.7°C (Rouf, Bettinger & Fudin, 2018). Thus, it is unclear if skin temperature would have differed had the skin been exposed to a wetting agent bearing a similar temperature to that of human urine. Furthermore, it is unclear if there are implications for using a solution of normal saline, as opposed to a slightly alkaline or acidic solution containing a similar chemical composition to urine, in understanding moisture-associated skin changes. A suggestion, therefore, is that future related protocols consider the use of simulated urine that has been warmed to a urine-consistent temperature.

9.7.4 Conclusion

Overall, the results of the LMMs pertaining to differences in erythema, stratum corneum hydration and skin temperature in wet versus dry skin have generated three important observations: first, no significant changes were evidenced for erythema at wet/damp sacral skin over the one hour in semi-recumbent participants. These findings indicate that the presence of moisture at the sacrum over one hour has no apparent effect on erythema, and suggests limited clinical benefit of attending to hourly biophysical erythema measurement at the wet/damp sacral skin of bedbound healthy adults. Second, stratum corneum hydration appeared to change readily at each 10 minute timepoint over the one hour in wet/damp sacral skin, with notable differences also evident by condition (control versus moisture intervention). It can, therefore, be concluded that differences in stratum corneum hydration at the sacrum of semi-recumbent participants were directly related to the presence of moisture at the skin-support surface interface. Finally, evident changes in skin temperature emerged at each 10 minute timepoint over the one hour at the elbows, heels and sacrum. However, when considering skin temperature differences by condition (control versus moisture intervention), nil significant differences were evident at any of the anatomical testing sites. These findings suggest limited clinical benefit of attending to hourly skin temperature measurement at the wet/damp sacral skin of healthy bedbound adults.

9.8 Pressure Injury Risk Identification using Biophysical Methods

It has been well-established within the literature that a reduction in skin integrity renders the skin susceptible to PI formation (Lustig et al., 2020; Okonkwo et al., 2020;

Schwartz et al., 2018; Shaked & Gefen, 2013; Stekelenburg, Oomens & Bader, 2005). Thus, capturing early signs of skin compromise is of critical importance in initiating interventions aimed at restoring and maintaining patients' skin health. As outlined in Chapter 1 of this thesis, the first and most fundamental step in determining PI risk is the evaluation of skin condition for its vulnerability to superficial breakdown (Defloor, 2005; Moore, 2008; Ousey & Cook, 2011). Clinicians have been traditionally taught to monitor patients' skin overlying the bony prominences for changes in skin colour, moisture and warmth (Lavery et al., 2004; Posada-Moreno e al., 2011). While a reddened skin discolouration—clinically termed erythema—is considered to be a cardinal sign of early stages of possible pressure-induced skin damage (Chacon et al., 2010; Kosiak, 1961; Vanderwee et al., 2007), elevations in skin temperature and skin moisture—adjacent to the skin surface—are inextricably linked to soft tissue deformation and diminished mechanical integrity of the stratum corneum (Baldwin, 2006; Gefen, 2021; Oerlemans et al., 1999; Sprigle et al., 2003; Wilkes et al., 1973; Wu et al., 2006). However, current methods of apprising the aforementioned skin parameters within a PI risk assessment context are fraught with challenges.

To date, clinicians continue to depend on visual skin assessment (VSA) for detecting signs of early or established pressure area damage (de Oliveira et al., 2022). However, the rising incidence of PIs within the clinical setting (NPUAP, 2019) raises the question of whether VSA is precise and reliable. In general, VSA involves using clinical judgment to assess an array of subjective cutaneous indicators of PI risk, and warrants reliance on the assessor's diagnostic accuracy of appraisal (Ousey & Cook, 2011). Clinical judgment, informed by VSA, has at times been found to have poor predictive capacity and inter-rater reliability (Garcia-Fernandez et al., 2014; Kottner & Dassen, 2010), and has been reported to suffer from high inter-examiner variability (Pancorbo-Hidalgo et al., 2006). Due to the skill dependency of VSA, the correct identification of a Stage I PI was observed to be as low as 60% in a diverse group of 1452 nurses (Beeckman et al., 2007). Consequently, there is potential for wide discrepancy among assessors due to varied interpretations of superficial skin changes which, in turn, may yield inconsistent or even erroneous findings (Kottner & Balzer, 2010). It can, therefore, be argued that the assessment findings are only as 'good' as the clinician's interpretation and judgment of risk status.

It should be explicitly highlighted that VSA does not address non-visible cues of tissue damage and, as such, may be unreliable in capturing deep tissue injury; that is, damage to the underlying soft tissue that occurs beneath the skin surface *without* prior visual and

palpable cues (Okonkwo et al., 2020). Consequently, possible 'latency' between the initial onset of tissue damage and the subsequent manifestation of a visible injury at the skin surface can often hinder the application of timely interventions. It can, therefore, be argued that VSA only captures something that has 'already happened', for example, tissue ischemia manifested as non-blanching erythema. Thus, there is a clear need for adopting an objective assessment method with the capacity to detect skin changes *before* visible signs of skin damage occur. The early detection of clinically concerning skin changes, while any potential damage is still microscope and reversible, could be followed by swift implementation of PI risk mitigating anatomy-specific strategies.

A review of the literature in Chapter 4 identified that there are non-invasive biophysical skin-analysis instruments with the capacity to provide a numerical and objective appraisal of key skin parameters (including erythema, skin hydration and skin temperature) associated with PI risk. An objective method of skin appraisal—used alongside clinical judgement—to identify PI risk has been advocated by some studies (Andersen & Karlsmark, 2008; Lin et al., 2021; Yoshimura et al., 2015). There is also some limited in-vivo evidence to support the validity and feasibility of some biophysical instruments in detecting skin changes (Bates-Jensen et al., 2007; Bates-Jensen et al., 2012; Borzdynski, McGuiness & Miller, 2016; Denzinger et al., 2020; Huimin et al., 2017; Lindgren et al., 2004). For example, Borzdynski, Miller, and McGuiness (2016) investigated the validity of a capacitance instrument; the SD202 Corneometer (Courage+Khazaka Electronic GmbH) in its assessment of skin moisture at pressure-prone areas of geriatric aged-care residents (n=38). The validity of the SD202 Skin Diagnostic Corneometer was demonstrated by its ability to establish statistically significant correlations ($p<0.05$) between skin moisture and risk of sacral and ischial PIs. Additionally, the study established the validity of the SD202 Skin Diagnostic Mexameter (Courage+Khazaka Electronic GmbH) in its ability to detect skin colour changes at pressure-prone areas; statistically significant associations ($p<0.01$) between erythema and risk of sacral PIs emerged for the sample (Borzdynski, McGuiness & Miller, 2016). The findings of this study also indicated that the measures afforded by instrumental evaluation in the assessment of the subtle red hues displayed in erythematous skin may provide an advantage over traditional clinician assessment.

Although beyond the scope of this research, it is worth to highlight that the SD202 Skin Diagnostic Mexameter could be of particular advantage in capturing signs of early pressure-induced skin damage in patients with deeply-pigmented skin (i.e., with greater

concentrations of melanin). As identified in Chapter 4 of this thesis, deep skin pigmentation can mask erythematous discolouration which, in turn, may mean that Stage I PIs are more likely to go undetected and deteriorate to full thickness injury (Bennett, 1995; Bethall, 2005; Sprigle et al., 2003). The SD202 Skin Diagnostic Mexameter has capacity to measure erythema irrespective of skin tone (Enviroderm, 2019) and, thus, may remove the challenges inherent to the identification of erythema in deeply-pigmented skin. In this respect, the effectiveness of biophysical identification of skin colour changes may exceed that of clinical judgment alone. However, future studies are required to confirm or refute this suggestion.

Overall, the findings of the current research add new evidence to support the utility of biophysical instruments in the assessment of skin parameters without altering skin status. As such, this research advocates for the integration of biophysical skin-analysis instruments as a feasible modality in the daily or multi-daily surveillance of PI risk. Specifically, infrared skin temperature scanners (such as the Exergen DermaTemp) (Exergen Corporation, Watertown, Massachusetts, USA) can be used to monitor heat patterns at pressure-prone areas, while the SD202 Skin Diagnostic Corneometer and Mexameter (Courage+Khazaka Electronic GmbH) can be used to evaluate skin hydration and skin erythema, respectively.

Depending on the clinical setting and patient acuity, the frequency and/or schedule of skin parameter monitoring may vary. For example, skin parameter measurement may be attended to at multiple intervals during the day in an intensive care unit, as opposed to daily measurement within a residential aged-care facility. In the case of limited skin changes, less than weekly assessment using a biophysical instrument may be sufficient to appraise skin status, unless a clinical change in patient condition warrants more frequent assessment. The non-invasive nature of these instruments and relative ease of use may also be of benefit to novice clinicians. Nevertheless, it must be explicitly acknowledged that despite their advantages, biophysical instruments cannot entirely compensate for the clinician's visual judgement and skills applied in PI risk evaluation.

Although the results of this research are preliminary, they support the possibility of moving beyond a purely subjective PI risk assessment approach to an objective one. As previously discussed, current methods of PI risk appraisal used in practice are deeply reliant on clinical judgment, and may fail to accurately identify cardinal signs of skin damage until they visibly manifest at the skin surface. The incorporation of commercially available biophysical instruments within clinical practice may facilitate earlier detection of important

skin changes associated with impending PI. A such, there is opportunity for biophysical instruments to be considered as a form of 'bedside technologies' that may help prompt timely preventive actions. To date, the long-term clinical efficacy of biophysical skin-analysis instruments specifically in PI risk assessment is yet to be tested. As such, the notion of instrumental skin evaluation—as a future novel approach to PI prevention—represents a fertile area for further research that will discussed in Section 9.11.2 of this chapter.

9.9 Strengths and Limitations

The studies undertaken as part of this research program have strengths and limitations. A key strength included an in-vivo approach to studying cutaneous skin manifestations in response to pressure-loading. During these simulations, participants were instructed to lay on a standard hospital-grade bed mattress in supine and semi-recumbent positions; two common resting positions adopted in the care of hospitalised bedbound patients (Burk & Grap, 2018), therefore, helping to optimise the generalisability of the findings to clinical settings. An additional strength of the study was that data were not missing at any timepoint, which helped to ensure completeness of the information contained within the dataset. Finally, the biophysical instruments used in this research were explored in previous validation studies (Borzdynski, Miller & McGuiness, 2016; Huimin et al., 2017; Rowledge et al., 2016), which conveys accuracy of the present study results and has strengthened its measurement validity (Polit & Beck, 2009; Whitehead & Schneider, 2013). The results of these previous validation studies have also enabled the consideration of the biophysical instruments in future research initiatives (Ahmed & Ishtiaq, 2021).

The preliminary phase of this research (Studies I and II) allowed the research team to address a previously unaddressed area of the literature (which also represented the methodological issue in the preliminary phase of this research) of whether brief pressure off-loading (interruptions) to enable skin parameter measurement altered skin parameter scores at pressure-prone areas of the body. The results indicated that brief off-loading did not alter skin status. These findings strengthened the internal validity of Study III, and further provide confidence to other researchers wishing to implement similar protocols in their own programs of research.

Although this research has made significant contributions to the body knowledge regarding PI-related skin manifestations in semi-reclined healthy adults, there are a number of limitations that must be acknowledged. One key limitation of this research relates to the

participant population. In the current research, the recruited participants were a relatively homogenous sample of predominantly young and ambulant healthy individuals with no existing skin conditions. These sample characteristics are less representative of populations with a predisposition to PI development. The study results cannot be generalisable, for example, to populations such as the bedridden elderly with existing co-morbidities and compromised skin barrier function. Furthermore, more females were recruited into this research than males (68.3% of participants were female in Studies I and II, and 70.8% of participants were female in Study III), which results in an imbalanced representation of individuals with male gender. As such, it can be argued that this study contributes with knowledge based only on a healthy and predominantly female population with minimal risk of acquiring PI. These limitations need to be considered when interpreting the study findings.

Another limitation relates to the ambiguity regarding the interpretation of baseline stratum corneum hydration measures gathered in the moisture intervention of Study III. It is unclear whether the baseline results imply 'instant saturation' of the stratum corneum upon immediate exposure to moisture and allude to an immediate water uptake/imbibition effect. Furthermore, the reported maximum baseline and interim stratum corneum hydration scores may have created a 'ceiling effect' where further change was limited, thereby, restricting the ability of the SD202 Skin Diagnostic Corneometer to accurately measure the intended outcome. There is also possibility that the SD202 Diagnostic Corneometer sensor, rather than measuring the moisture 'within' the stratum corneum, may have registered the wetting agent (normal saline) contiguous to the skin surface. Future studies are required to confirm or refute these speculations.

Although this research enabled the establishment of a descriptive library of skin parameter scores gathered at pressure-prone areas of healthy adults, it is limited by a lack of benchmark data against which these gathered scores could be interpreted. In the absence of benchmark data, it is challenging to determine if the reported data are significant from a clinical perspective. Furthermore, thresholds for biophysical measurements of erythema, stratum corneum hydration and skin temperature that would precede early stages of skin damage and/or compromise are unknown. Consequently, it cannot be inferred if the reported differences in skin parameter scores are clinically meaningful and, thus, could serve as reliable predictors of early skin damage.

Finally, this research was limited by the one hour immobilisation interval restriction that was used to examine skin changes across the anatomical testing sites. As such, the study results relate only to changes examined over the one hour period, which precludes generalising the findings beyond this timeframe. Thus, it can be argued that the one hour period of data collection provided only a 'snapshot' of skin changes, and may be insufficient to propose a recommendation for clinical practice. Monitoring skin changes over an extended period of time would have enabled the research team to capture longer-term skin changes. However, it is important to acknowledge that this immobilisation interval was necessary to ensure participant safety. Yet, despite the one hour limit of data collection to examine skin changes, the study was nonetheless able to identify some significant skin changes occurring over this relatively brief period of time. Establishing if these changes can be considered as clinically meaningful, however, requires further investigation. Furthermore, investigating skin changes beyond one hour is pivotal if generalisability to bedbound populations is to be achieved, given that these groups of patients are often subjected to extended immobilisation intervals.

Overall, the study limitations have provided an opportunity for future research and refinement of future related protocols. The recommendations for future research have been proposed in Section 9.10.2 of this chapter.

9.10 Recommendations

Section 9.7 of this chapter outlined the significance of the results gathered in this research in terms of their clinical relevance and contribution to the existing evidence base. Based on the study findings in regard to skin parameter differences occurring over one hour in supine and semi-reclined healthy adults, modifiable considerations for future clinical practice can now be discussed.

9.10.1 Recommendations for Clinical Practice

9.10.1.1 Responding to Anatomical Variability in Skin Condition

It is a recommendation that clinicians attend to individualised regional inspection of skin parameters in semi-recumbent healthy adults. This research identified wide variability in skin parameter scores at investigated anatomical regions. As previously outlined, this research examined baseline and end measures of skin manifestations (erythema, stratum corneum hydration and skin temperature) occurring at the bony prominences of the body (the elbows, heels and sacrum) over one hour. Skin manifestations were assessed using

commercially available biophysical skin analysis instruments. While the results for erythema and stratum corneum hydration displayed an oscillatory behaviour with no discernible pattern over the one hour period of data collection, the same finding was not evident for skin temperature results. Distinct differences in baseline and end scores of skin temperature emerged among participants. Notable differences in skin temperature were also apparent between the trunk and extremities; higher skin temperature scores, on average, were recorded at the sacrum than at the elbows and heels. As such, assessable anatomical regions may need to be better defined in contemporary PI risk assessment scales. Furthermore, a large variance in site-specific minimum and maximum scores emerged for all gathered skin parameters. To strengthen the study inferences, the study protocols need to be replicated with larger and more diverse samples.

It is recommended that clinicians attend to individualised regional inspection of skin parameters in semi-recumbent healthy adults. This research identified wide variability in baseline and endpoint skin parameter scores at the investigated anatomical sites in response to pressure-loading and extrinsic factors. Notable differences in skin temperature were also apparent between the trunk and extremities; higher skin temperature scores, on average, were recorded at the sacrum than at the elbows and heels. Overall, these findings—when considered in a clinical practice context—allude to the need for a nursing assessment that can accommodate skin parameter assessment anatomically.

9.10.1.2 Monitoring Erythema at the Sacral Skin

It is a recommendation that clinicians incorporate additional biophysical monitoring of erythema at warmed sacral skin of healthy semi-recumbent individuals. As previously outlined in Section 9.7.1 of this chapter, the results of the LMMs analyses (Study III) indicated that heated sacral skin, when compared to unheated sacral skin, displayed significant differences (p=.012) in erythema over a relatively brief period of time (one hour) in a semi-recumbent participants. The same finding was not evident at the elbows or heels, suggesting that the introduction of heat, over one hour, at the extremities of semi-reclined participants does not impact skin colour. These findings imply that clinicians may need to exert particular vigilance when monitoring for skin colour changes at the sacrum of febrile patients in whom fever may cause elevations in core body temperature and promote skin warming. However, there is a need to first examine methods of attending to biophysical skin colour assessment without impacting provision of routine nursing care.

9.10.1.3 Monitoring Skin Temperature at the Elbows, Heels and Sacrum

It is a recommendation that clinicians incorporate additional biophysical monitoring of skin temperature of warmed skin at the elbows, heels and sacrum of healthy semi-recumbent individuals. As previously outlined in Section 9.7.1 of this chapter, the results of the LMMs analyses (Study III) indicated that heated skin at the elbows, heels and sacrum, when compared to unheated skin at the same locations, displayed significant differences ($p<.001$) in skin temperature in healthy individuals lying in a semi-recumbent position over one hour. It can be extrapolated from these findings that warming of the skin over one hour has a direct impact on skin temperature. This observation is particularly salient when considered against the long-standing literature that highlights a correlation between sustained elevations in skin temperature and the aetiological processes that give rise to PI development.

It has been postulated within the literature that sustained elevations in skin temperature may influence the functional and mechanical properties of the skin. Elevations in skin temperature in loaded skin have been associated with increased cutaneous stiffness and metabolic demand, as well as a decrease of dermo-epidermal adhesion (Hatje et al., 2015). These processes, in turn, increase the risk of deformation injury (Hatje et al., 2015; Kokate et al., 1995; Patel et al., 1999) by lowering the damage thresholds for the skin and underlying soft tissues (Gefen et al., 2011; Jan et al., 2012; Yusuf et al., 2015; Zeevi et al., 2018). Thus, localised areas of pronounced skin warmth may serve as an indicator of incipient skin damage with the propensity to progress toward a PI. However, the specific thresholds at which skin temperature can be regarded as a reliable indicator of impending skin damage are yet to be elucidated.

It should be noted that the observed elevations in skin temperature over the one hour period of data collection should not be immediately perceived as a threat. That is, it cannot be assumed that underlying skin and soft tissue damage occurs upon brief exposure of the skin to topical heat. Nevertheless, in the absence of published skin temperature thresholds (i.e., thresholds that would create conditions conducive to skin damage in both healthy and non-healthy skin), it is unknown if the reported skin temperature elevations occurring over one hour can be considered clinically meaningful if extrapolated to at risk populations.

9.10.1.4 Monitoring and Responding to Skin Wetness at the Sacrum

A recommendation derived from the results of this research is additional monitoring of sacral skin that is moist. As previously outlined in Section 9.7.3 of this chapter, the results

of the LMMs analyses (Study III) indicated that wet sacral skin, when compared to dry sacral skin, displayed significant differences ($p<.001$) in stratum corneum hydration, over one hour, in healthy semi-reclined individuals. Descriptive data further indicated maximum readings (range: 97-99 units) for stratum corneum hydration at each timepoint increment, translating to 'over-hydrated' skin as per the SD202 Skin Diagnostic Interpretation Guide (reference range: 83-99 units) (Barrett-Hill, 2015).

A review of the literature in Chapter 3 of this thesis identified that over-hydration of the skin has been postulated to be a contributing factor to PI development (Clark et al., 2010; Gray et al., 2011; McCreath et al., 2006; Schnelle et al., 2013). The prolonged over-hydration of the stratum corneum induces the disruption to the skin's acid mantle (Clark et al., 2010) which, in turn, alters the skin's resilience to frictional forces (Gray et al., 2011), making it more friable and susceptible to mechanical injury (Bates-Jensen et al., 2007; McCreath et al., 2006; Schnelle et al., 2013). In-vivo research by Warner et al. (2003) demonstrated that extended exposure of as little as four hours of the skin to moisture can lead to extensive disruption of the intercellular lipid lamellae in the stratum corneum, heightening the skin's vulnerability to the mechanical forces of shear and friction.

In this research, the direct contact of the sacral skin with moisture appeared to have initiated an immediate skin response among participants. A possible suggestion for clinical practice, in relation to this finding, is the integration of a monitoring system into bedlinen that could act as a trigger for 'skin wetness' in bedbound patients. It is important to acknowledge that the new evidence generated by this research—by demonstrating the immediacy and complete saturation of the skin upon exposure to moisture—provides emphasis to the importance of the above recommendation and the need for its widespread implementation within clinical practice. Skin monitoring systems could have particular applications in the detection of skin changes in patients presenting with urinary and/or faecal incontinence, whose skin may be at risk of continual exposure to moisture. In this respect, a skin moisture detection system could act as a trigger to instigate immediate nursing responses, such as the application of s skin barrier product, aimed at counteracting excess moisture at the target site (Brennan et al., 2017).

9.10.1.5 Monitoring and Responding to Skin Dryness at the Elbows and Heels

A recommendation based on the results of this research is a nurse-initiated skin care regime that restores stratum corneum hydration at the elbows and lower limbs. In this

research, the mean stratum corneum hydration scores were lowest at the elbows (range: 8.1-9.6 units) and heels (range: 8.0-9.3 units). When considered against the SD202 Skin Diagnostic Measurement Interpretation Guide (Barrett-Hill, 2015), these ranges correspond to 'very dry skin' (reference range: 0-23 units). As discussed earlier in this chapter, a large volume of in-vivo and in-vitro dermatological literature indicates that dry skin is a relevant factor for PI development (Draelos et al., 2020; Elias & Menon, 1991; Rawlings et al., 2004). Studies report that skin dryness diminishes the skin's tensile strength (Rawlings et al., 1994), making it more prone to the development of cracking or fissures that permits invasion (via permeation) of micro-organisms at the skin surface (Elias & Menon, 1991). Similarly, a key study by Proksch and Lachapelle (2005) indicated that chronic skin dryness results in impaired skin barrier function and leads to a reduction of elastic properties. A reduction in skin elastin predisposes the skin to the adverse effects of shear which, in vulnerable skin, can contribute to epidermal blisters and superficial ulceration (Grey et al., 2006).

Lechner et al. (2017), in their multi-centre study, investigated the association between dry skin and PIs at the sacrum and heels of nursing-home residents (n=3,837) with a mean age of 76.1 years (*SD*=15.5). The findings indicated that 71.9% of residents with heel PIs had dry skin, which is comparable to a similar study by Lichterfeld et al. (2016). In both these studies, the severity of skin dryness was also higher in residents with existing PIs. Approximately half of the residents with dry skin but without PI exhibited mild forms of dryness at the heels. Overall, the results of Lechner et al. (2017) indicate that dry heel skin may be considered a risk factor for PI development. The same finding, however, was not reported for dry skin at the sacrum; it was documented that 46.2% of residents with sacral PIs had dry skin. Furthermore, based on multi-level logistic regressions predicting PIs at the sacrum, Lechner et al. (2017) found that there was no association between skin dryness and sacral PIs [$p<.001$, 95% CI (0.62-2.0)]. Thus, it was concluded that skin dryness may be less of a factor in the formation of sacral PIs.

The application of a daily or multi-daily ceramide-containing moisturising cream and/or emollient to dry skin at the elbows and heels may help to increase stratum corneum water content and reduce trans-epidermal water loss (Draelos et al., 2020). Ceramides are one of the most important lipids that provide the waterproof characteristics of the skin barrier, and may aid in the establishment and/or maintenance of the water-retention capacity of the stratum corneum (Danby et al., 2020; Draelos et al., 2020). Thus, ceramide-containing moisturising creams have the ability to restore the capacity of intercellular lipid bilayers to

absorb, retain and redistribute water content. In their recent study of 53 healthy women, Draelos et al. (2020) reported the clinical benefit of applying ceramide-containing moisturising cream to dry skin at the lower limbs over three days. The results indicated a marked increase in stratum corneum intercellular lipid levels among the study participants.

9.10.2 Recommendations for Future Research

Thorough consideration of the study results has led to the development of recommendations for subsequent research. These future research directions have been proposed with consideration to the study limitations outlined previously in this chapter.

9.10.2.1 Exploration Repositioning Frequency on Skin Condition

The findings generated by this study may inform future research examining the frequency of repositioning schedules for bedbound patients. Repositioning is critical in restoring blood volume to previously-compressed anatomical regions and, thus, forms an integral component of PI prevention. The current guidelines recommend that bedbound patients be repositioned every two hours (EPUAP-NPUAP-PPPIA, 2019). However, the results of the present study, collected over an hourly interval, indicate that there may be potentially clinically significant skin changes occurring earlier than the suggested two hour threshold for skin damage. Although the current practice of repositioning high-risk patients every two hours is not without benefit, its origin is somewhat arbitrary. That is, the two hour repositioning recommendation is based primarily on 'expert opinion', with few epidemiological studies and inconclusive evidence that repositioning at this frequency is effective in preventing PI development (Rich et al., 2011). Despite the dearth of evidence, the two hour repositioning schedule has become firmly established as the standard of care. It is, therefore, recommended that future research initiatives explore skin changes manifested beyond a one hour pressure-loading timeframe in diverse patient cohorts.

9.10.2.2 Exploration of Pressure Injury Risk Assessment and Interventions Customised by Anatomical Location

It is recommended that future related studies explore individualised PI risk assessment regimes customised by anatomical location. This recommendation stems from the wide variability in skin parameter scores noted at the investigated anatomical testing sites (sacrum, elbows and heels) in response to pressure-loading and extrinsic factors. Current and well-recognised standardised PI risk assessment scales are not customised by anatomical location. Rather, an assessment of skin condition guided by these scales tends to be broad

and reliant on assessor training (Bryant, Moore & Iyer, 2021). As such, accessible anatomical regions may need to be better defined in contemporary PI risk assessment scales. It is also recommended that related investigations examine the distributional characteristics of skin parameters over anatomical locations that were not the focus of the current research. Skin assessment could be considered at regions susceptible to PI formation in the supine and semi-recumbent positions (such as the occiput, scapulae, gluteal cleft and ischial tuberosities), and lateral positions (such as the iliac crest, greater trochanters and malleoli).

Future related studies may additionally explore the utility of biophysical technologies in capturing site-specific skin changes in an attempt to prompt the provision of anatomy-targeted care. It can be inferred from this research that distinct interventions aimed at PI prevention may vary by anatomical location based on the condition of the skin. For example, while under-hydrated limbs may benefit from the application of topical agents such as a moisturising cream and/or emollient, over-saturated skin at the pelvic region may benefit from the application of a barrier product. Similarly, while cooler extremities may benefit from warming, intermittent skin cooling may be advised at warmed skin at the trunk of the body to optimise the local skin microclimate (Jan et al., 2002; Jan et al., 2013; Lachenbruch, 2005; Patel et al., 1999).

In this research, skin temperature scores returned to near baseline following the fifteen minute washout period that was used as a pragmatic interval between the control and heat intervention. Thus, the findings imply that a fifteen minute off-loading regime could be an effective strategy to promote skin cooling of previously-loaded pressure-prone areas of the body. However, it is unknown whether such a relatively rapid recovery rate would have been observed if the study were to be replicated with cohorts susceptible to PI development. The washout interval data in the current study has not been examined in this thesis; however, the contribution of these data to nursing care may be an important area of consideration in future related studies. The data yielded could build on the body of evidence in relation to skin cooling as a method of optimising and/or modifying the local skin microclimate.

9.10.2.3 Exploration of Longer-Term Skin Changes in At Risk Populations

It is recommended that future related research considers the longitudinal evaluation of cutaneous skin changes in vulnerable patient cohorts. As outlined in Chapter 2 of this thesis, PIs are most prevalent among the bedridden elderly (Khor et al., 2014) due to the increased susceptibility of aged skin to the effects of prolonged mechanical loading (Moda et

al., 2021). Thus, investigations comprising a sample of older adults and in clearly defined clinical populations are warranted. To further enhance the generalisability and robustness of results, the inclusion of multiple study sites (such as acute medical/surgical, post-operative and residential aged-care) is recommended. Future research initiatives could consider recruitment of individuals in whom the skin tends to be subjected to compressive forces for extended durations of time, such as long-term intubated patients in the intensive care unit, in whom PIs represent a common complication of immobility (Anderson, 2007; He et al., 2016). Studies could further recruit individuals living in the community with limited functional capacity and diminished ability to engage in effective pressure off-loading.

As previously outlined, this research was limited by an inability to examine skin changes and compounding effects of pressure-loading beyond a one hour immobilisation interval. This individual period of participant immobilisation was restricted to one hour as an initial excursion to ensure participant safety with consideration to the *Prevention and Treatment of Pressure Ulcers Clinical Practice Guidelines* (EPUAP-NPUAP-PPPIA, 2019). As such, this immobilisation interval precluded the examination of skin parameter changes exceeding one hour. It is possible that compounding effects beyond the one hour may have existed. Thus, the incorporation of an extended skin monitoring duration (for example, extending to weeks or months) could enable exploration of longer-term skin changes. An extended monitoring timeframe is particularly important when examining chronic moisture-related skin changes in incontinent patients, in whom the exposure of the skin to moisture often continues throughout the day or night (Falloon et al., 2018; Gray et al., 2002; Ichikawa-Shigeta et al., 2014). As such. future studies may consider intermittent daily and overnight monitoring of skin changes in incontinent patient cohorts. However, the imposition of intermittent measurements, placed on both the patient and clinician, warrants consideration.

9.10.2.4 Exploration of Conceptual Differences in Skin Moisture

A research-orientated recommendation is to examine and clearly define the conceptual differences regarding skin moisture that are represented by PI risk assessment scales. As outlined in Chapter 4 of this thesis, a number of PI risk assessment scales have been developed for the formal assessment of PI risk factors, and are strongly advocated for clinical use by the *Prevention and Treatment of Pressure Ulcers Clinical Practice Guidelines* (EPUAP-NPUAP-PPPIA, 2019). Three commonly-used standardised PI risk assessment scales include the Braden Scale (Bergstrom & Braden, 1987), Waterlow Scale (Waterlow, 1985) and Norton Scale (Norton, 1987). All three scales feature the parameter of skin

moisture as a sub-scale assessment component that is reliant on a broader interpretation of moisture contiguous to the skin surface. Often, descriptors such as 'boggy', 'damp' or 'wet' are used. It is recommended that future studies compare the Braden, Waterlow and Norton scales in terms of their classification of skin moisture; the understanding emerging from these studies could be applicable on a conceptual level. Further comparisons could also be made with reference to objective measures of skin moisture—as measured using the SD202 Skin Diagnostic Corneometer—within the deeper skin layers. The respective data analyses could enable the preliminary exploration of which concepts of skin moisture are relevant to skin changes preceding PI development.

It is recommended that future studies re-examine whether the briefest contact of the skin with moisture leads to the immediate saturation of the stratum corneum. To reinforce, in this research, the sacral skin was exposed to a wetting agent (a solution of normal saline) over the course of one hour. Upon baseline measurement, over-saturation of the stratum corneum was immediately apparent, reflected by maximum stratum corneum hydration scores of 97-99 units registered by the SD202 Skin Diagnostic Corneometer. However, it is unclear if these baseline results represent the intended outcome (and are a matter of clinical relevance) or the wetting agent contiguous to the skin surface. Establishing this knowledge is essential to confirm the sensitivity of SD202 Skin Diagnostic Corneometer in the appraisal of stratum corneum hydration in wet/damp skin. Future studies may investigate moisture transfer rates at the level of the stratum corneum. The next logical step would be to determine the rate of the re-establishment of the natural skin condition following brief and longer-term exposure to external moisture. To enable comparisons, data could be extracted from both healthy and non-healthy skin with inherent stratum corneum impairments.

9.10.2.5 Exploration of Skin Changes in Darkly-Pigmented Skin

As part of baseline data collection of this research, the Fitzpatrick Skin Type Classification (Fitzpatrick, 1988) was used to classify participants into categories reflective of their calculated skin type. Due to the preponderance of participants with 'fair' and 'pale skin' (Fitzpatrick Skin Types I and II), the study results are based largely on participants with lightly-pigmented skin. None of the participants had 'dark brown to black skin' (Fitzpatrick Skin Type VI). Only 17.1% of participants in Studies I and II and 12.4% of participants in Study III had 'brown skin' (Fitzpatrick Skin Type V). It is recommended that future related studies recruit a greater number of individuals with darkly-pigmented skin. The results of

these future studies could also confirm the sensitivity of the SD202 Skin Diagnostic Mexameter in capturing erythema data irrespective of the level of pigmentation.

9.10.2.6 Exploration of Biophysical Instruments as Innovative Tools in Pressure Injury Risk Assessment

There is an opportunity to integrate a biophysical skin assessment approach, as an adjunct to clinical judgment, within clinical practice. As previously outlined, the prevention of PIs is an important nursing priority that requires swift recognition of changes in skin colour and skin temperature localised over the bony prominences of the body (NPUAP, 2019). However, underpinning these actions is a reliance on the assessor's diagnostic accuracy of skin appraisal itself, which is deeply subjective and may be limited. As such, the initiation of PI prevention measures may be hindered by the fact that skin appraisal may not always yield reliable and accurate results. In view of these limitations, there is an opportunity to augment appraisal accuracy with objective techniques. Biophysical instruments have the capacity to provide 'real-time' monitoring of skin condition and yield clinically relevant data—that may not always be quantified visually—at the skin-support surface interface. In terms of the practical application, biophysical monitoring systems could enable early detection of cutaneous alterations, generating a nursing 'alert' to prompt the initiation of risk-mitigating interventions. However, future studies are needed to strengthen the emerging evidence relating to the diagnostic utility of biophysical instruments.

Overall, this research has evidenced some significant cutaneous manifestations, occurring over one hour, at common pressure-prone areas of the body of semi-recumbent healthy adults. As previously outlined in this thesis, the current evidence-base alludes to a correlation between skin colour, skin moisture, skin temperature and early stages of skin damage. Thus, the clinician's awareness of any alterations to the aforementioned parameters forms a fundamental step in the implementation of strategies aimed at the restoration and/or maintenance of skin condition. At present, an appraisal of skin condition tends to be largely subjective. Furthermore, there often tends to be 'latency' between the initial onset of PI-associated skin damage and the subsequent manifestations of visible skin and tissue damage which, in turn, can hinder the application of timely interventions. A biophysical skin appraisal approach could enable earlier detection and confirmation of skin and soft tissue damage associated with early onset PI development. To date, however, the long-term clinical efficacy of biophysical instruments in a PI risk assessment context is yet to be tested and, as such, represents an area of recommended future research.

9.10.2.7 Establishing Biophysical Thresholds for Skin Damage at Pressure-Prone Areas

The incorporation of a biophysical skin monitoring regime has the potential to serve as a detection system of clinically relevant skin changes occurring at the pressure-prone areas of the body. This detection system could trigger nursing actions designed to mitigate the risk of PI formation. However, there is first a need to translate biophysical skin measures into clinically meaningful indicators of impending PI development. Studies are specifically needed to determine clinically concerning thresholds of erythema, stratum corneum hydration and skin temperature in healthy adults, and to establish the independent contribution of biophysical skin measures to superficial skin damage. Furthermore, establishment of site-specific thresholds are required to target PI prevention strategies by anatomical region. This initiation of anatomy-specific interventions *before* any damage threshold is breached is crucial in preventing the progression of further damage.

Future studies may seek to investigate the correlation between skin temperature data (gathered using the infrared technique) and inflammatory markers associated with PI development. As previously outlined in Chapter 4 of this thesis, there is evidence to suggest that raised skin temperature may act an important clinical indicator of tissue inflammation associated with imminent PI (Kokate et al., 1995; Soares et al., 2019), with inflammatory changes reported to occur three to ten days *before* visual signs of skin breakdown (Bach et al., 2015; Herrman et al., 1999; Oerlemans et al., 1999). Local inflammation is predominantly mediated by pro-inflammatory cytokines, such as interleukin-1β, tumour necrosis factor alpha (TNF- α), and interleukin-6 (Ren et al., 2009). These factors play an important role in activating inflammatory cells such as neutrophils, macrophages and monocytes at the affected area (Ansar & Ghosh, 2016). Given the multi-faceted nature of the current study findings, there is scope for multi-disciplinary research to continue to examine the clinical applications of infrared technologies in their assessment of inflammatory skin changes associated with PI development.

In conjunction with skin temperature, sub-epidermal moisture has been reported to predict inflammatory tissue changes associated with the initial stages of PI formation (Bates-Jensen et al., 2008; Bates-Jensen et al., 2009; Okonkwo et al., 2020; O'Brien et al., 2018). Commercial systems sensitive to tissue capacitance and permittivity have gained attention for their ability to non-invasively detect sub-epidermal moisture within the deeper tissue layers (Mayrovitz et al., 2009). As such, future related research may employ capacitance devices to gather sub-epidermal moisture data over pressure-prone areas of individuals at risk of PI

development. The data gathered could be subsequently examined for their ability to inform incipient inflammatory-mediated skin damage with the propensity to progress towards a PI.

9.11 Conclusion

Overall, this thesis has provided a unique exploration of skin manifestations attributed to changes in the local skin microclimate (enabled through the application of thermal and moisture stimuli at the skin-support surface interface) of healthy adult volunteers. Skin manifestations were examined over time (one hour) in conditions simulating immobilisation with brief interruptions. Although this research is limited by the use of a discrete cohort of healthy participants with negligible risk of PI development, the findings have highlighted distinct skin responses in response to disruptions in the local skin microclimate. The insights gained from this research are anticipated to contribute to the emerging evidence relating to the interactions between skin colour and skin microclimate properties at the skin-support surface interface.

As a relatively new area of enquiry, the results of this research require replication using larger sample sizes and in clinical populations. Extended skin monitoring durations are also required to comprehensively appraise and evaluate the individual contribution of the investigated biophysical skin properties to superficial skin changes. The establishment of site-specific damage thresholds for biophysical measures of erythema, stratum corneum hydration and skin temperature is also needed in order to expand on the results of the current study. Establishing these thresholds could enable the provision of timely preventative actions before skin damage becomes visually apparent at the skin surface. Finally, the results of this thesis contribute to the advancement of scientific knowledge informing the utility of biophysical skin assessment in optimising current approaches used in PI risk identification.

9.12 Chapter Summary

This chapter has discussed the results of this thesis in detail, with their clinical implications considered in conjunction with the existing literature. Conclusions have been drawn with reference to the research aims and objectives, and the significance of the generated findings has been discussed. The strengths and limitations of this research have been acknowledged, recommendations for clinical practice have been proposed, and future research directions have been outlined. The next chapter will provide a final conclusion of this thesis and its contribution to the body of scientific knowledge regarding cutaneous manifestations in response to changes in the skin microclimate.

CHAPTER 10
Conclusion

This final chapter brings the thesis to its conclusion. The chapter reiterates the study aims, provides an overview of the key study findings, and reinforces key recommendations for practice and future research. The purpose of this research was to explore possible skin manifestations attributed to local pressure and the skin microclimate. Biophysical measurements of erythema, melanin, stratum corneum hydration and skin temperature were evaluated in response to local intermittent pressure-loading over one hour during controlled manipulation of heat and moisture at the skin-support surface interface of healthy adult volunteers. In doing so, this thesis contributes to the existing knowledge base pertaining to biophysical properties of erythema, melanin, stratum corneum hydration and skin temperature changes in heated versus unheated skin, and wet versus dry skin over one hour of immobilisation with periodic off-loading.

This thesis commenced with the broad research question of *how do erythema, melanin, stratum corneum hydration and skin temperature measures—when assessed quantitatively at the skin overlying bony prominences of the body—change over time in response to local pressure and the controlled manipulation of skin microclimate at the skin-support surface interface*?

To address this question, a program of research was undertaken that involved two phases and three related studies. Studies I and II comprised the preliminary phase of this research and piloted many of the procedural and logistical aspects that would determine the progression of the experimental phase (Study III) of this research. All studies involved between- and within-subject comparisons with repeated measures, yielding both descriptive and inferential data. In effect, this research advanced from purely descriptive-correlational to quasi-experimental.

The aim of Studies I and II was to assess for skin parameter changes following brief consecutive off-loading over one hour at pressure-prone areas of the body (the sacrum, elbows and heels) among healthy adults lying supine and then semi-recumbent. Studies I and II sought to answer the following research questions:

1. How do erythema, melanin, stratum corneum hydration and skin temperature measures—when assessed at the skin overlying bony prominences of the body—change over time in response to local pressure?
2. Is there agreement between uninterrupted and interrupted methods when assessing erythema, melanin, stratum corneum hydration and skin temperature over time? That is, are the results from skin parameter measurement altered or relatively unchanged during brief off-loading of bony prominences?

Studies I and II were conducted due to the limited in-vivo literature on skin changes occurring during hourly immobilisation protocols with brief interruptions in healthy human participants. The preliminary phase of this research program was the first to assess for skin parameter differences between two methods (interrupted and uninterrupted) with respect to timepoints (every ten minutes) over one hour. This information was required to address the aforementioned research questions, and to enable consideration if momentarily shifting pressure to enable measurements altered skin parameter readings as a by-product of the data collection method.

Studies I and II resulted in the establishment of a descriptive library of erythema, melanin, stratum corneum hydration and skin temperature scores, which allowed the research team to gauge the range and variability of these skin parameters for the participant sample. Overall, it can be inferred from the Studies I and II data that brief off-loading of pressure-prone areas over a period of one hour in healthy adults does not appear to impact skin parameter outcomes. Thus, when translating these findings into clinical practice, implementing a brief off-loading regime every ten minutes for healthy patients lying continuously in the supine and semi-recumbent positions would not appear to clinically alter PI risk derived from the indirect effects of skin microclimate. However, considering that endpoint measures tended to be higher than baseline measures for all skin parameters, it is possible that beyond a one hour period, a person may be approaching the threshold of skin changes that may place vulnerable skin at risk of initial stages of skin damage. Negligible variation in skin parameter measurements between the supine and semi-recumbent positions in Studies I and II (as was determined through statistical analysis) led the research team to consolidate its focus on the semi-recumbent position only in Study III.

In essence, Studies I and II informed the protocol and procedural aspects of the proceeding quasi-experimental study (Study III). The aim of Study III was to examine the

skin microclimate effects on clinical manifestations often attributed to PI development in healthy adults. Specifically, Study III sought to assess for changes in timepoints and differences in measures of erythema, stratum corneum hydration and skin temperature in response to heat derived from a heated blanket, and moisture in the form of normal saline when applied at the skin-support surface interface of healthy adults lying in the semi-recumbent position, over time (one hour), with periodic off-loading to record the data. The hypotheses of Study III were informed by the literature and the preliminary research phase.

Study III sought to answer the following research question: *How do erythema, stratum corneum hydration and skin temperature measures—when assessed quantitatively at the skin overlying bony prominences of the body—change over time in response to local pressure and the controlled manipulation of skin microclimate at the skin-support surface interface?*

Although Study III did not examine tissue damage or superficial skin injury, it did demonstrate some significant skin changes occurring over a one hour period of immobilisation. Three key findings of Study III were noted: first, statistically significantly differences for erythema (p=.012) emerged between the control and heat intervention for the sacrum. Second, statistically significant differences were evident for skin temperature between the control and heat intervention for the elbows (p<.001), heels (p=.006) and sacrum (p<.001). Third, statistically significant differences for stratum corneum hydration (p<.001) emerged between the control and moisture intervention for the sacrum.

No statistically significant differences emerged for erythema and stratum corneum hydration between the control and heat intervention for the elbows and heels. These findings suggest that while warming the skin at the heels and elbows does not appear to impact on erythema and stratum corneum hydration over one hour in semi-recumbent healthy adults, some early changes in erythema and skin temperature can occur following warming the skin at the sacral region. Furthermore, the application of moisture to the sacral skin over one hour appears to directly impact stratum corneum hydration. Further research is needed to determine the clinical significance and implications of this finding.

The findings of this thesis need to be considered in the context of the several limitations. First, the one hour immobilisation interval used to evaluate skin changes restricted the ability to capture skin changes beyond this timeframe. Second, the sample of

predominately young, healthy participants with no skin compromise limits the generalisation of the findings to at-risk populations such as the bedridden elderly with increased skin fragility and a predisposition to PI. Furthermore, it was beyond the scope of this research to examine connections between superficial skin changes and tissue damage. As such, this research can only contribute knowledge based on skin changes occurring in healthy intact skin with negligible risk of PI. Finally, in view of a lack of benchmark data for biophysical measures of erythema, stratum corneum hydration and skin temperature at pressure-prone areas in healthy adults, it is challenging to establish the significance of the gathered data from a clinical perspective. Further investigation is required to determine benchmarks for investigated biophysical skin measures.

Despite the study limitations, this research can be regarded as a body of evidence that has established the necessary groundwork and provided a framework to better understand the construct of the skin microclimate and superficial skin changes. Taken together, the findings generated by this research have made an important contribution to the current understanding of various skin manifestations (captured using biophysical technologies) attributed to local pressure and the skin microclimate. Clinicians may consider incorporating additional monitoring of skin colour and temperature at pressure-prone regions of febrile patients. More frequent skin-monitoring regimes are also recommended in wet/damp skin.

The results of this study indicate that significant thermal manifestations ($p<.001$) and erythematous hues ($p=.012$) in heated skin may start to be evidenced in as little as one hour at common pressure-prone areas of healthy semi-recumbent adults. These results—when extrapolated to populations with a predisposition PI development—may question the standard two hour re-positioning regime adopted within clinical settings. An increased repositioning frequency may be warranted in semi-recumbent patients with warmed skin to permit the identification of important skin changes. However, it is difficult to fully interpret the clinical relevance of the present study findings in the absence of clinically significant thresholds for erythema and skin temperature over pressure-prone areas.

The large variability in skin parameter measures by anatomical testing site and between participants alludes to the importance of an individualised regional assessment of skin condition in supine and semi-reclined bedbound patients. A key message of this thesis is that distinctive protocols in regard to skin care by anatomical region may be required for individual patients at risk of PI development. While the application of a daily or multi-daily

moisturiser/emollient may be warranted for dry/under-hydrated skin at the elbows and heels, a barrier product may be advised for wet/over-hydrated sacral skin. Finally, sacral skin may benefit from local cooling as a method of microclimate optimisation.

The preliminary evidence generated by this research provides a strong foundation for future related research exploring clinical and research applications of biophysical instruments in the digital quantification of skin parameters associated with PI. The findings of this research support biophysical technologies in capturing notable thermal manifestations and changes in skin hydration at pressure-prone areas of healthy adults. The integration of biophysical skin assessment techniques within clinical nursing practice could serve as an innovative strategy to complement visual skin assessment (VSA) by facilitating earlier detection of skin changes and informing anatomy-specific interventions. However, without further studies, it is difficult to reach a general conclusion concerning the long-term feasibility of biophysical instruments in a PI risk assessment context.

The results of this research provide a basis for future hypothesis testing in related work, for example, exploring the associations between biophysical skin properties and superficial skin damage. This foundational work may be instrumental to the potential refinement of current PI risk assessment processes. Thus, there is scope to conduct studies with the aim of exploring the clinical reliability of cutaneous properties in predicting superficial skin damage. The findings of these studies may, in turn, advance knowledge and strengthen the evidence regarding innovative biophysical instruments as potential novel diagnostic tools applied in PI risk assessment. In essence, future studies should consider the development and validation of an approach to PI risk assessment that would harness the strengths of bimodal skin appraisal; both clinician (visual) and instrumental (objective).

To further build on the findings of the present research and for generalisability to be achieved, refinement of the study sample is required in future related protocols. Specifically, future investigations should aim to capture a broader sample of participants with regard to age and risk status. Broadening the study sample characteristics, combined with a longer-term skin monitoring timeframe, would enhance an understanding of longitudinal microclimate and skin colour skin changes beyond a sample of healthy young adults. Furthermore, there is a need to strengthen the inferences drawn from this study with regard to moisture-associated skin changes. Future research is required to clarify the concept of skin moisture and determine valid and reliable ways of measuring and differentiating between

moisture contiguous to the skin surface, and moisture within the deeper skin layers. Future studies may also examine the rate at which the skin—upon brief contact with topical moisture—becomes saturated or how rapidly water uptake occurs at the level of the stratum corneum.

It is well-established within the literature that PIs are debilitating, painful and costly encumbrances to those affected, with the propensity to become life-threatening injuries. To date, these wounds remain a persistent problem and adverse event, predominantly affecting the bedbound elderly population. The ever-increasing PI prevalence rates and their devastating outcomes emphasises the need to enhance PI risk assessment and prevention efforts. Clinically, the maintenance of skin integrity—via optimisation of the skin microclimate—in vulnerable populations is pivotal to PI prevention efforts. Thus, the early recognition of clinically concerning skin colour and skin microclimate changes occurring over pressure-prone areas of the body is critical for enabling timely interventions to protect tissue viability.

This research focused on the interactions that occur between the skin and support-surface in healthy adults, and investigated their influence on manifestations often attributed to PI formation. The clinical significance of these findings may have relevance in evaluating the correlations between skin colour (melanin and erythema) and skin microclimate (skin moisture/stratum corneum hydration and skin temperature) and pressure-loading on skin and tissue health. The findings of this research further contribute preliminary evidence to future studies exploring the clinical efficacy of biophysical measures of skin colour and skin microclimate properties as clinical indictors of impending pressure ulceration. These innovations are promising and urgently needed given that the incidence and prevalence of PIs remain unacceptably high across all healthcare settings. Overall, these thesis investigations contribute to new paradigms for understanding skin microclimate changes enabled through quantitative appraisal.

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Appendices

APPENDIX A: ETHICS APPROVAL (STUDIES I & II)

From: humanethics@latrobe.edu.au
To: William McGuiness
Cc: CAROLINE JACQUELINE BORZDYNSKI; Charne Miller
Subject: HEC18467 - New Application - Approved
Date: Thursday, 6 December 2018 2:09:19 PM

** This is an automatically generated email, please do not reply. Contact details are listed below.**

Dear William McGuiness,

The following project has been assessed as complying with the National Statement on Ethical Conduct in Human Research. I am pleased to advise that your project has been granted ethics approval and you may commence the study.

Application ID: HEC18467
Application Status/Committee: University Human Ethics Committee

Project Title: Skin manifestations in response to pressure and local microclimate in healthy adults Title on PICF: Skin changes in response to pressure in healthy adults

Chief Investigator: William McGuiness

Other Investigators: Ms Caroline Jacqueline Borzdynski, Charne Miller

Date of Approval: 06/12/2018
Date of Ethics Approval Expiry: 06/12/2023

The following standard conditions apply to your project:

- Limit of Approval. Approval is limited strictly to the research proposal as submitted in your application.

- Variation to Project. Any subsequent variations or modifications you wish to make to your project must be formally notified for approval in advance of these modifications being introduced into the project.

- Adverse Events. If any unforeseen or adverse events occur the Chief Investigator must notify the UHEC immediately. Any complaints about the project received by the researchers must also be referred immediately to the UHEC.

- Withdrawal of Project. If you decide to discontinue your research before its planned completion, you must inform the relevant committee and complete a Final Report form.

- Monitoring. All projects are subject to monitoring at any time by the University Human Ethics Committee.

- Annual Progress Reports. If your project continues for more than 12 months, you are required to submit a Progress Report annually, on or just prior to 12 February. The form is available on the Research Office website. Failure to submit a Progress Report will mean approval for this project will lapse.

- Auditing. An audit of the project may be conducted by members of the UHEC.

- Final Report. A Final Report (see above address) is required within six months of the completion of the project.

You may log in to ResearchMaster (https://rmenet.latrobe.edu.au) to view your application.

Should you require any further information, please contact the Human Research Ethics Team on:
T: +61 3 9479 1443 | E: humanethics@latrobe.edu.au.

Warm regards,
Human Research Ethics Team
Ethics, Integrity & Biosafety, Research Office

APPENDIX B: ETHICS APPROVAL (STUDY III)

Research Office

From	University Human Research Ethics Committee
HEC Number	HEC20300
Project title	Skin Manifestations in Response to Local Pressure and Microclimate in Healthy Adults
Principal Investigator	William McGuiness
Co-Investigators	Caroline Borzdynski, Charne Miller
Approval Period	24th August 2020 – 24th August 2025
Date	24 August 2020

I am pleased to advise you that the University Human Research Ethics Committee has granted ethical approval of the project listed above, subject to the following conditions being met:

Conditions of Approval – All projects

- This research project was approved during COVID-19 restrictions. The conduct of the research during this period should reflect any changes in relation to COVID-19 mandates in the relevant jurisdictions. To accommodate these mandates a modification request must be submitted for any changes prior to their implementation.
- Approval is limited to the research project and associated documents as outlined in this ethics approval letter.
- The Principal Investigator will immediately report anything that might warrant review of ethical approval of the project.
- **Modifications to an Approved Project:** Any changes to the project application, project description/protocol and/or other project documents must be submitted for review and approval in accordance with the instructions outlined on the Human Research Ethics website. Modifications can be implemented once written approval has been received.
- **Annual Report:** If your project continues for more than 12 months, you are required to submit an Annual Report by the due date outlined in the annual report reminder. The form is available on the Human Research Ethics website. Failure to submit a Progress Report will mean approval for this project will be suspended and no further research activities can be carried out until the annual report is received.
- **Final Report or Withdrawal of Project:** At the conclusion of your project you must submit a final report within 6 months via the process outlined on the Human Research Ethics website.
- **Safety Reporting:** If a significant safety issue arises from the conduct of the project, it must be reported via the process outlined on the Human Research Ethics website.

- **Monitoring:** All projects are subject to monitoring at any time and will be monitored in accordance with the University's Research Monitoring Policy.

The Human Research Ethics Committee (HREC) Terms of Reference, membership and standard forms are available from http://www.latrobe.edu.au/researchers/research-office/ethics/human-ethics.

Should you require any further information, please contact the Human Research Ethics Team on:
T: +61 3 9479 1443| E: humanethics@latrobe.edu.au.

Warm regards,

David Finlay
Chair, University Human Research Ethics Committee

Skin Changes in Response to Local Pressure and Microclimate in Healthy Adults

The research is being carried out by the following researchers:
Caroline Borzdynski

The research is being carried out in partial fulfilment of a PhD (Nursing) under the supervision of Assoc. Prof William McGuiness and Dr Charne Miller. The following researchers will be conducting the study:

Role	Name	Organisation
Research student	Ms Caroline Borzdynski	La Trobe University
Research funder	This research is supported by in kind support by La Trobe University.	

1. **What is the study about?**
   You are invited to participate in a study about skin changes linked to wounds called pressure injuries (or bedsores). We hope to learn about how skin properties of temperature, colour, and moisture change in healthy adults over one (1) hour of laying down. This study will take place at La Trobe University Bundoora Campus, and the Alfred Centre, Prahran. To compensate you for your time, you will receive a $40 Coles-Myer Gift Card upon completion of this study.

2. **Do I have to participate?**
   Being part of this study is voluntary. If you want to be part of the study, we ask that you read the information below carefully and ask us any questions. If you decide not to participate, this will not affect your relationship with La Trobe University or with any other organisation listed in this document. If you participate in this study, you may be asked if you would be interested in participating in future related studies, which are also voluntary and you do not have to participate.

3. **Who is being asked to participate?**
   You have been asked to participate because you meet the study eligibility criteria, that is, you are an adult over 18 years of age, you are able to provide written informed consent, and you do not have any skin damage at the elbows, heels, or base of the lower back. As a participant of this study, you must **not** have:

   (i) current wounds or skin conditions (e.g., pressure sores, abrasions, rashes, dermatitis, rosacea, eczema, psoriasis, inflammation) at the elbows, heels and/or base of the lower back;

   (ii) tattoos at the elbows, heels, and/or base of the lower back; and

   (iii) conditions/disorders that could affect your ability to lay stable for up to one (1) hour at a time.

4. **What will I be asked to do?**
   You will be asked to attend **two (2)** visits. Each visit is expected to take two-and-a-half (2.5) hours of your time. Please note that the two study visits will need to be scheduled on separate days.

   The study will be conducted at the locations below. You can select the location that is most convenient for you to get to.

   1. La Trobe University, Plenty Rd and Kingsbury Dr, Bundoora VIC 3086
      George Singer (GS) Building; Level 4 Nursing Laboratories; or
   2. The Alfred Centre, 99 Commercial Road, Prahran VIC 3181
      La Trobe University Clinical School; Level 4 Physiotherapy Laboratory.

   If you decide to take part in this study, you will be asked to contact the Research Student on the telephone number listed on **page 5 of 7** of this document to complete a brief telephone screening checklist. The researcher will go over the study details with you and summarise what will happen at each visit. You will also have an opportunity to ask the researcher about any areas of the study that you may want more information or clarification about.

During the study visit, the researcher will use a skin-analysis device called the **SD202 Skin Diagnostic** (Courage & Khazaka Electronics) (Fig 1) and a skin temperature scanner called the **Exergen DermaTemp** (Exergen Cooperation) (Fig 2) to assess your skin at the elbows, heels, and base of the lower back (Fig 3). The SD202 Skin Diagnostic will be used to assess your skin colour and skin moisture, and the Exergen DermaTemp scanner will be used to assess your skin temperature. This will involve gently pressing the device sensors to your skin, which will not cause you any discomfort or pain.

We ask that you please do not apply any skincare products (moisturisers, lotions, creams, emollients, sprays or other solutions) to the skin at the elbows, heels, and/or lower back for at least 24 hours prior to each visit.

Figure 1. Skin-analysis device (SD202 Skin Diagnostic)

Figure 2. Skin temperature scanner (Exergen DermaTemp)

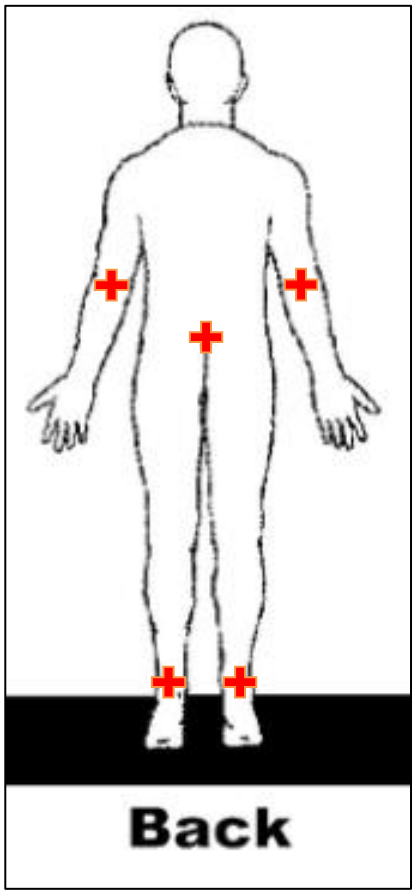

Figure 3. Skin testing sites on the body. The red crosses represent sites of skin measurement.

The next section of this document outlines what will happen during each study visit.

VISIT 1

At the beginning of visit 1, the researcher will collect some basic information from you. This will include your age, gender, and ethnicity. The researcher will also assess your risk of getting a bedsore. This will be followed by an assessment of skin colour and calculation of your body mass index (BMI). After this, you will be provided with a freshly-laundered hospital gown to change into within a private space of the laboratory room. Please note: You will be asked to remove all your underlying clothing from underneath the gown, however, you may leave an upper undergarment on if desired. This is important to ensure assessment results are as accurate as possible.

There are two (2) study conditions that you will complete during each study visit. You may start with either of the two conditions, and this will be determined by the researcher on the day of the first visit. In both conditions, you will be asked to lie down flat on your back on the laboratory bed. A pillow will be used to support your head, and a cotton sheet and blanket will be applied to your body up to your chest. Your arms will be left uncovered. You will be asked to lay flat on your back for one (1) hour while keeping as still as possible.

In the first condition, skin measurements will be taken after 1 hour, while in the second condition, skin measurements will be repeated every 10 minutes over 1 hour whilst you are lying down. At each measurement, the researcher will assess your skin at the elbows, heels and lower back using the devices outlined on Page 2 of 7 of this document. First, you will be asked to briefly lift each arm at a time to enable skin assessment at the elbows. Next, you will be asked to briefly lift each foot at a time for skin assessment at the heels. Finally, you will need to briefly reposition onto your side to enable skin assessment at the lower back.

After you complete the first condition, you will be given a 15 minute break to stand up and walk at a gentle pace around the laboratory room. Every 5 minutes during this time, you will be asked to stand while the researcher re-assesses the skin at the elbows, heels and lower back. After 15 minutes, you will begin the second condition where you will be asked to lie down flat on your back on the laboratory bed for 1 hour, keeping as still as possible. Skin measurements will be taken at the start and end of this condition.

Once you complete both conditions, visit 1 will conclude. You will be provided with privacy upon re-dressing.

VISIT 2

At the start of visit 2, you will be asked to change into a clean standard hospital gown. Once you are wearing the hospital gown, you will be asked to lie down on your back on the laboratory bed. This time, the head of the bed will be adjusted to a 45° angle, which means you will be partially sitting-up in the bed instead of lying flat. A pillow will be used to support your head, and a cotton sheet and blanket will be applied to your body up to your chest. The arms will be left uncovered.

In visit 2, You will complete the same two study conditions as for visit 1. In the first condition, skin measurements will be taken after 1 hour, while in the second condition, skin measurements will be repeated every 10 minutes over 1 hour. At each measurement, the researcher will assess your skin at the elbows, heels and lower back using the devices outlined on Page 2 of 7 of this document. After you complete the first condition, you will be given a 15 minute break to stand up and walk at a gentle pace around the laboratory room. Every 5 minutes during this time, you will be asked to stand while the researcher re-assesses the skin at the elbows, heels and the lower back. You will then begin the second condition where you will be asked to lie down on the same laboratory bed for 1 hour while keeping as still as possible. Once you complete both conditions, visit 2 will conclude. You will be provided with privacy upon re-dressing into your normal attire.

If you would like to participate in this study, the next step will be to contact Ms Caroline Borzdynski (research student) on the telephone number listed on Page 5 of 7 of this document. You will complete a brief telephone screening checklist to determine if you meet the study requirements. You may also use this time to ask the researcher about any areas of the study that you may want more information or clarification about.

5. What are the benefits?

There is no immediate benefit to you participating in this study. However, your participation will help to collect valuable information about skin changes in healthy adults. This information may then be used in future related research. We hope that the results of this research inform the evidence about ways of identifying important skin changes linked to bedsores. Therefore, your participation in this study is highly valued and appreciated.

6. What are the risks?

With any study there are (1) risks we know about, (2) risks we do not know about, and (3) risks we do not expect. If you experience something that you are not sure about, please contact us immediately, so we can discuss the best way to manage your concerns.

The research team does **not** foresee any risk occurring to you by taking part in this study. However, as per the study's safety protocol, the researcher will monitor and assess your skin for any signs of damage. This will involve the researcher gently pressing with their fingertip the skin surface at your elbows, heels and lower back for 2-3 seconds, once every 10 minutes, while you are lying down. If these areas of the skin do not respond as desired, the study will be stopped.

Please note: if you experience discomfort during the visit, please report this to the researcher – the study will be stopped temporarily so that we can remedy this. You'll be given the choice to stop the study if you wish.

7. What will happen to information about me?

We will collect and store information about you in ways that will not reveal who you are. This means you cannot be identified in any report or publication from this study.

You will also be given an individual **Participant Identification Number (PIN)** by the researcher. The PIN will be used to re-identify all data collected for privacy purposes. The PIN will be recorded by the researcher in a log-book, together with your title and full name next to it. Only you and the researcher will know your PIN. The log-book will be stored separately to all other study data in secure storage.

We will keep your information for 7 years after the project is completed. After this time has elapsed, we will destroy all of your data.

We will collect, store and destroy your data in accordance with La Trobe Universities Research Data Management Policy which can be viewed online using the following link: https://policies.latrobe.edu.au/document/view.php?id=106/.

The information you provide is personal information for the purposes of the Privacy and Data Protection Act 2014 (Vic). You have the right to access personal information held about you by the University, the right to request correction and amendment of it, and the right to make a complaint about a breach of the Information Protection Principles as contained in the Information Privacy Act.

8. Will I hear about the results of the study?

You will be asked, in the Consent Form, to indicate if you would like to receive a copy of the results via e-mail or post. We will let you know about the results of the study by e-mailing or posting the group results to you.

9. What if I change my mind?

At any time, you can choose to no longer be part of the study. You can let us know by:

1. Completing the 'Withdrawal of Consent Form' (provided at the end of this document);
2. Calling us;
3. Emailing us.

Your decision to withdraw at any point will **not** affect your relationship with La Trobe University or any other organisation listed. When you withdraw, we will stop asking you for information. Information already collected about you will be used in the study unless you request that it is removed. Furthermore, once the results have been analysed, we can only withdraw information, such as your name and contact details.

10. Who can I contact for questions or if I want more information?

If you would like to speak to us, please use the contact details below:

Name/Organisation	Position	Telephone	Email
Caroline Borzdynski La Trobe University	Research Student		
Assoc. Prof William McGuiness La Trobe University	Chief Investigator	+61 3 9479 6 743	W.McGuiness@latrobe.edu.au
Dr Charne Miller La Trobe University	Co-supervisor	+61 3 9479 5 090	C.Miller@latrobe.edu.au

11. What if I have a complaint?

If you have a complaint about any part of this study, please contact:

Ethics Reference Number	Position	Telephone	Email
HEC18467	Senior Research Ethics Officer	+61 3 9479 1443	humanethics@latrobe.edu.au

Consent Form – Declaration by Participant

I (the participant) have read (or, where appropriate, have had read to me) and understood the participant information statement, and any questions have been answered to my satisfaction. I agree to participate in the study, I know I can withdraw at any time. I agree information provided by me or with my permission during the project may be included in a thesis, presentation and published in journals on the condition that I cannot be identified.

I would like my information collected for this research study to be:
☐ Used for future related studies

☐ I would like to receive a copy of the results via email or post. I have provided my details below and ask that they only be used for this purpose and not stored with my information or for future contact.

Name	Email (optional)	Postal address (optional)

☐ I would like to participate in similar studies in the future, specific to this research. I have provided my details below and ask that they only be used for future contact regarding this research.

Name	Email	Postal address (optional)

Participant Signature
☐ I have received a signed copy of the Participant Information Statement and Consent Form to keep

Participant's printed name	
Participant's signature	
Date	

Declaration by Researcher
☐ I have given a verbal explanation of the study, what it involves, and the risks and I believe the participant has understood;
☐ I am a person qualified to explain the study, the risks and answer questions

Researcher's printed name	
Researcher's signature	
Date	

* All parties must sign and date their own signature

Withdrawal of Consent

I wish to withdraw my consent to participate in this study. I understand withdrawal will not affect my relationship with La Trobe University of any other organisation or professionals listed in the Participant Information Statement. I understand the researchers cannot withdraw my information once it has been analysed, and/or collected as part of a focus group.

I understand my information will be withdrawn as outlined below:

- ✓ Any identifiable information about me will be withdrawn from the study
- ✓ The researchers will withdraw my contact details, so I cannot be contacted by them in the future related studies unless I have given separate consent for my details to be kept in a participant registry.
- ✓ The researchers cannot withdraw my information once it has been analysed, and/or collected as part of a focus group

***if you have consented for your contact details to be included in a participant registry you will need to contact the registry staff directly to withdraw your details.*

I would like my already collected and unanalysed data

☐ Destroyed and not used for any analysis
☐ Used for analysis related to this study
☐ Used for analysis related to future related studies

Participant Signature

Participant's printed name	
Participant's signature	
Date	

Please forward this form to:

CI Name	Assoc. Prof William McGuiness
Email	W.McGuiness@latrobe.edu.au
Phone	+61 3 9479 6743
Postal Address	La Trobe University, Plenty Road & Kingsbury Drive, Bundoora VIC 3086

Skin Changes in Response to Local Pressure and Microclimate in Healthy Adults

The research is being carried out by the following researchers:
Caroline Borzdynski

The research is being carried out in partial fulfilment of a PhD (Nursing) under the supervision of Assoc. Prof William McGuiness and Dr Charne Miller. The following researchers will be conducting the study:

Role	Name	Organisation
Research student	Ms Caroline Borzdynski	La Trobe University
Research funder	This research receives in kind support from La Trobe University.	

1. **What is the study about?**
   You are invited to participate in a study about skin changes linked to wounds called pressure injuries (or bedsores). We hope to learn about how skin properties of temperature, colour, and moisture change in response to heat and moisture over one (1) hour. This study is being conducted at La Trobe University, Bundoora campus. To compensate you for your time, you will receive a $40 Coles-Myer gift card once you complete the study.

2. **Do I have to participate?**
   Participating in this study is voluntary. If you want to be part of the study, we ask that you read this document carefully and ask us any questions. If you decide not to participate, this will not affect your relationship with the researcher or with La Trobe University.

3. **Who is being asked to participate?**
   You have been asked to participate because you meet the study eligibility criteria, that is, you are an adult over 18 years of age, you are able to provide written informed consent, and you do not have any skin damage at the elbows, heels, or base of the lower back. As a participant of this study, you must **not** have:
   - (i) current wounds or skin conditions (e.g., pressure sores, abrasions, rashes, dermatitis, rosacea, eczema, psoriasis, inflammation) at the elbows, heels and/or base of the lower back;
   - (ii) tattoos at the elbows, heels, and/or base of the lower back;
   - (iii) skin sensitivities and/or conditions that you believe could be aggravated or made worse by the skin being in direct contact with heat and moisture for up to one (1) hour at a time; and
   - (iv) conditions/disorders that could affect your ability to lay stable for up to one (1) hour at a time.

4. **What will I be asked to do?**
   If you take part in this study, you will be asked to attend one (1) appointment, which is expected to take four (4) hours of your time. We ask that you please do **not** to apply any skin products (moisturisers, lotions, emollients, sprays or other solutions) to the skin surface of the elbows, heels or lower back for at least 24 hours prior to the appointment.

   This study is taking place at La Trobe University, Plenty Road & Kingsbury Dr, Bundoora VIC 3086.

   The next section of this document outlines what will happen at the study appointment.

   In the first part of this study, the researcher will collect some basic information from you, which will include your age, gender and ethnicity. The researcher will assess your risk of getting a bedsore, followed by an assessment of your skin colour, and calculation of your body mass index (BMI). Next, you will be given a clean hospital gown to change into within a private space of the room. **Please note**: we ask that you remove all underlying clothing underneath the gown, however, you may leave an upper undergarment on if desired. This is important to ensure accuracy of the results.

Once you are wearing the provided hospital gown, you will be asked to lie down on your back on a laboratory bed. The bedhead will be adjusted to a 45° angle and a pillow will be used to support your head. While you are lying down, the researcher will use skin-analysis devices to assess your skin temperature, colour and moisture at your elbows, heels and base of your lower back (Figure 1).

A skin-analysis device called the **SD202 Skin Diagnostic** (Courage & Khazaka Electronics) (Figure 2) will be used to assess skin colour and moisture. This will involve lightly pressing a sensor onto your skin and will **not** cause you any discomfort. Skin temperature will be assessed using a scanner called the **Exergen DermaTemp** (Exergen Cooperation) (Figure 3). This temperature sensor will be held 5mm from your skin and a reading will be taken. All device sensors will be cleaned with hospital-approved disinfectant between use.

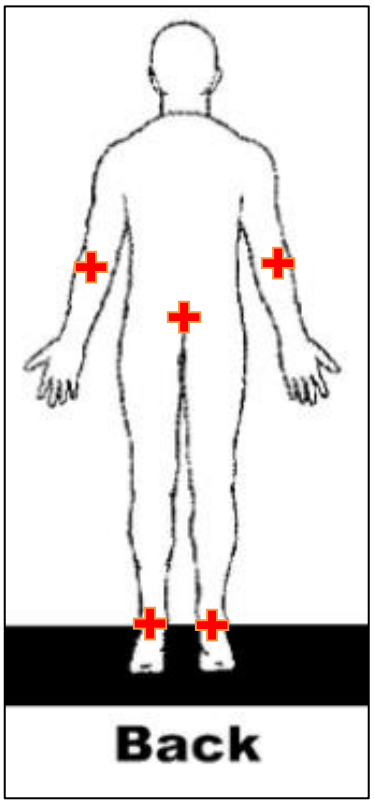

Figure 1. Skin testing sites on the body. The red crosses represent sites of skin measurement.

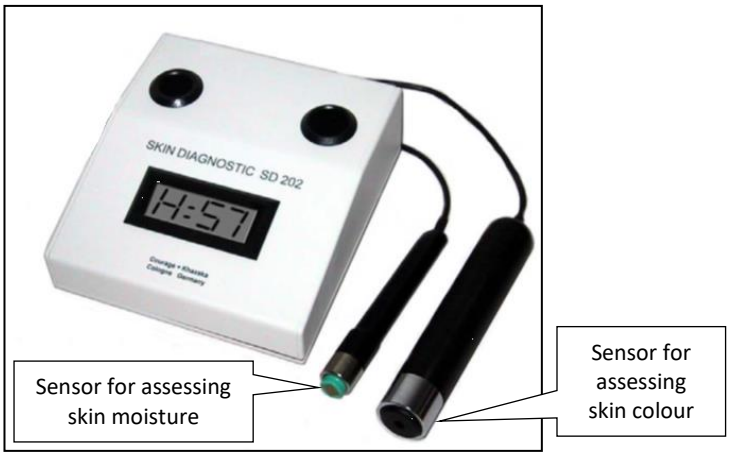

Figure 2. Skin-analysis device (SD202 Skin Diagnostic)

Figure 3. Skin temperature scanner (Exergen DermaTemp)

After your skin is assessed and you are lying down, a cotton bedsheet and blanket will be applied up to your chest, with your arms left uncovered. For the next 60 minutes, you will need to lie in this position while the researcher re-assesses your skin every 10 minutes. You will need to briefly turn to your side to enable skin assessment at the lower back.

Figure 4. Elevation of the bedhead (45° angle)

Please note:
It's important that you keep as still as possible whilst lying down on the laboratory bed once the assessment starts.

If you anticipate that lying down semi-reclined with the bedhead adjusted to a 45° angle (Figure 4) for 1 (one) hour at time may cause you discomfort, please consider your participation in the study carefully. Should you report any discomfort to the researcher during the study, the study will be stopped immediately so that we can remedy this. You'll be given the choice to stop the study if you wish.

After 1 hour, you will be asked to stand up and walk at a gentle pace around the laboratory room for the next 15 minutes. Every 5 minutes during this time, the researcher will ask you to stand while your skin is re-assessed at the elbows, heels, and the lower back. After this, you will complete the next part of the study. You will be asked to lie back down on the laboratory bed, however, this time you will lie directly on a heated blanket set to a comfortable medium temperature of 30°C. After your skin is assessed, a cotton top-sheet and blanket will be used to cover you up to your chest, with your arms exposed. After 10 minutes, the researcher will assess your skin at the elbows, heels and lower back. This process will be repeated every 10 minutes over 1 hour. After 1 hour, you will again be asked to stand up and walk at a gentle pace around the laboratory room for the next 15 minutes. Every 5 minutes during this time, the researcher will ask you to stand while your skin is re-assessed. After this, you will complete the next part of this study, as described below.

In the next part of the study appointment, you will once again lay flat on your back on the laboratory bed. This time, you will be required to position your lower back over an absorbency pad pre-soaked with sterile water. Next, a cotton top-sheet and blanket will be used to cover you up to your chest, with your arms left uncovered. After 10 minutes, the researcher will assess your skin at the elbows, heels and lower back. This assessment will be repeated every 10 minutes over 1 hour.

Once you have completed the study, the appointment will conclude. You will be given time to re-dress within a private space of the laboratory room. A clean towel for you to use at your convenience. You will be thanked for your time and provided with a $40 Coles-Myer Gift Card.

Procedure for booking your study appointment
If you would like to participate in this study, the next step will be to contact Ms Caroline Borzdynski (Research Student) on the telephone number listed on **Page 5 of 7** of this document. You will complete a brief telephone screening checklist to assess if you meet the study conditions. If so, an appointment will be booked. During the telephone screening, you may also ask the researcher about any areas of the study that you may want more information or clarification about.

COVID-19 SCREENING PROCEDURES
At the moment, La Trobe University has implemented restrictions specific to the COVID-19 (Coronavirus) pandemic. This means that during the telephone screening process, the researcher will need to ask you some questions about whether you think you might have symptoms of COVID-19. **It's important that you try answer these questions truthfully or to your best ability. The information you provide us will be kept strictly confidential.**

PLEASE NOTE: If on the day of your appointment you notice that you are feeling unwell, or think you may have symptoms of respiratory infection (fever, cough, shortness of breath, sore throat, fatigue), we ask that you please contact a member of the research team listed on **Page 5 of 7** of this document. We strongly recommend that you immediately seek medical advice/guidance by calling the National COVID-19 Information and Triage Line on 1800 020 080 or COVID-19 call centre on 1800 675 398 should you experience any of these symptoms.

5. What are the benefits?

There is no immediate benefit to you participating in this study. However, your participation will help to collect valuable information about skin changes in healthy adults. This information may then be used in future related research. We hope that the results of this research inform the evidence about ways of identifying important skin changes linked to bedsores. Therefore, your participation in this study is highly valued and appreciated.

6. What are the risks?

With any study there are (1) risks we know about, (2) risks we do not know about, and (3) risks we do not expect. If you experience something that you are not sure about, please contact us immediately, so we can discuss the best way to manage your concerns.

The research team does **not** foresee any risk occurring to you by taking part in this study. However, as per the study's safety protocol, the researcher will monitor and assess your skin for any signs of damage. This will involve the researcher gently pressing with their fingertip the skin surface at your elbows, heels and lower back for 2-3 seconds, once every 10 minutes, while you are lying down. If the skin does not respond as desired, the study will be stopped. If you experience discomfort or feel unwell during the visit, please report this immediately to the researcher; the study will be stopped temporarily so that we can remedy this. You'll be given the choice to stop the study if you wish.

7. What will happen to information about me?

We will collect and store information about you in ways that will not reveal who you are. This means you cannot be identified in any report or publication from this study in accordance with applicable privacy laws. You will also be given a Participant Identification Number (PIN) to re-identify all data collected about you to protect your privacy. The PIN will be recorded by the researcher in a logbook, together with your title and full name next to it. Only you and the researcher will know your PIN. The logbook will be stored separately to all other study data in secure storage at La Trobe University. We will keep your information for 15 years after the research is completed. After this time, we will destroy all data.

The storage, transfer and destruction of your data will be undertaken in accordance with the Research Data Management Policy https://policies.latrobe.edu.au/document/view.php?id=106/.

The personal information you provide us is personal information for the purpose of the Privacy and Data Protection Act 2014 (Vic). Any health information collected about you will be handled in accordance with the Health Records Act 2001 (Vic). Subject to any exceptions in relevant laws, you have the right to access and correct your personal information by contacting the research team.

8. Will I hear about the results of the study?

You will be asked in the Consent Form to indicate if you would like to receive a copy of the results via e-mail or post. We will let you know about the results of the study by e-mailing or posting the group results to you at the study's conclusion.

9. What if I change my mind?

At any time, you can choose to no longer be part of the study. You can let us know by:

1. Completing the Withdrawal of Consent Form (provided on Page 7 of this document);
2. Calling us;
3. Emailing us.

Your decision to withdraw at any point will **not** affect your relationship with La Trobe University or with the researcher.

If you decide to withdraw from the study, we will stop asking you for information. Information already collected about you will be used in the study unless you request that it is removed. Furthermore, once the results have been analysed, we can only withdraw information such as your name and contact details.

10. Who can I contact for questions or if I want more information?

If you would like to speak to us, please use the contact details below:

Name/Organisation	Position	Telephone	Email
Caroline Borzdynski La Trobe University	Research Student		
Assoc. Prof William McGuiness La Trobe University	Chief Investigator	+61 3 9479 6 743	W.McGuiness@latrobe.edu.au
Dr Charne Miller La Trobe University	Co-supervisor	+61 3 9479 5 090	C.Miller@latrobe.edu.au

11. What if I have a complaint?

If you have a complaint about any part of this study, please contact:

Ethics Reference Number	Position	Telephone	Email
HEC20300	Senior Research Ethics Officer	+61 3 9479 1443	humanethics@latrobe.edu.au

Consent Form – Declaration by Participant

I (the participant) have read (or, where appropriate, have had read to me) and understood the participant information statement, and any questions have been answered to my satisfaction. I agree to participate in the study, I know I can withdraw at any time until [four weeks] following the collection of my data. I agree information provided by me or with my permission during the project may be included in a thesis, presentation and published in journals on the condition that I cannot be identified.

I would like my information collected for this research study to be:
☐ Only used for this specific study;
☐ Used for future related studies;
☐ Used for any future studies

☐ I would like to receive a copy of the results via email or post. I have provided my details below and ask that they only be used for this purpose and not stored with my information or for future contact.

Name	Email (optional)	Postal address (optional)

☐ I would like to participate in similar studies in the future, specific to this research. I have provided my details below and ask that they only be used for future contact regarding this research.

Name	Email	Postal address (optional)

Participant Signature
☐ I have received a signed copy of the Participant Information Statement and Consent Form to keep

Participant's printed name	
Participant's signature	
Date	

Declaration by Researcher
☐ I have given a verbal explanation of the study, what it involves, and the risks and I believe the participant has understood;
☐ I am a person qualified to explain the study, the risks and answer questions

Researcher's printed name	
Researcher's signature	
Date	

* All parties must sign and date their own signature

Withdrawal of Consent

I wish to withdraw my consent to participate in this study. I understand withdrawal will not affect my relationship with La Trobe University of any other organisation or professionals listed in the Participant Information Statement. I understand the researchers cannot withdraw my information once it has been analysed, and/or collected as part of a focus group.

I understand my information will be withdrawn as outlined below:

- ✓ Any identifiable information about me will be withdrawn from the study
- ✓ The researchers will withdraw my contact details, so I cannot be contacted by them in the future related studies unless I have given separate consent for my details to be kept in a participant registry.
- ✓ The researchers cannot withdraw my information once it has been analysed, and/or collected as part of a focus group

***if you have consented for your contact details to be included in a participant registry you will need to contact the registry staff directly to withdraw your details.*

I would like my already collected and unanalysed data

☐ Destroyed and not used for any analysis
☐ Used for analysis related to this study
☐ Used for analysis related to future related studies

Participant Signature

Participant's printed name	
Participant's signature	
Date	

Please forward this form to:

CI Name	Assoc. Prof William McGuiness
Email	W.McGuiness@latrobe.edu.au
Phone	+61 3 9479 6743
Postal Address	La Trobe University, Plenty Road & Kingsbury Dr, Bundoora VIC 3086

LA TROBE UNIVERSITY

APPENDIX E: PROSPECTIVE PARTICIPANT TELEPHONE SCREENING CHECKLIST (STUDIES I & II)

Name: ____

✂
- -

SCREENING CHECKLIST

⚐ Complete a checklist for each prospective participant

Study title: **Skin Manifestations in Response to Local Pressure and Microclimate in Healthy Adults**

Screening completed by: ________________________________ Date: ____________ Screening #: _______

[Introduce self and role]

Thank you for calling and your interest in participation. The purpose of this telephone screening is to check if you are eligible to participate in this study, and it should take no longer than 15 minutes to complete. Please know that all the information that you provide during the telephone screening will be kept confidential. Before I ask you some questions, did you have any questions about the study that you would like me to answer?

[ANSWER QUESTIONS]

SCREENING QUESTIONS

What I can do then is ask a few quick questions to ensure that you meet the study eligibility criteria, if that's okay?

[IF YES, PROGRESS TO SCREENING QUESTIONS]

AGE

ASK: [Mr/Mrs/Ms NAME], you are over the age of 18 years – is that correct?

❒ No (ineligible; thank and end call) ❒ Yes (go to next question)

ABILITY TO READ AND COMPLETE/SIGN CONSENT FORM

Do you mind me asking if you have been diagnosed by a medical practitioner with a cognitive impairment, or you suspect a cognitive impairment? ❒ No ❒ Yes

RESEARCHER RECORD: person meets *no cognitive impairment* criterion **Y / N** (If no, thank and end call)

Have you read the Participant Information Statement and Consent Form thoroughly? **Y / N**

Do you/did you require an interpreter to translate any information included in the Participant Information Statement and Consent Form for you? **Y / N**

So, can I clarify that you understand that the study involves:

1. Attending two separate study visits, with a single visit expected to take 2.5 hours of your time **Y / N**
2. Avoiding the application of skin products (moisturisers, lotions, creams, emollients) to the skin surface specifically at the elbows, heels, or lower back for at least 24 hours prior to each visit? **Y / N**

ASK: can you please tell me if you have any current wounds or skin conditions (such as dermatitis, eczema, psoriasis, irritation, inflammation) at the elbows, heels or base of the lower back?

❒ No (go to next question) ❒ Yes (ineligible; thank and end call)

ASK: do you have any tattoos at the elbows, heels, or base of the lower back?

❒ No (go to next question) ❒ Yes (ineligible; thank and end call)

ASK: do you have any conditions/disorders that could affect your ability to lay stable for up to one hour at a time, or that could cause you to experience discomfort whilst lying down?

❒ No (go to next question) ❒ Yes (ineligible; thank and end call)

Thank you, those are all the screening questions I had.

IF ELIGIBLE

It appears likely that you are eligible for the study. If you are still willing to participate, could you please read and sign the Consent Form on Page 6 of the Participant Information Statement, and return it to myself on the first day of your scheduled appointment.

RESEARCHER RECORD: person is willing to sign the Consent Form **Y / N** If no, thank and end call.

As you are aware, this study is being conducted at La Trobe University and the Alfred Centre. Would you like to nominate a preferred location for your visit at either of these locations?

The participant has nominated to attend the visit at:

❒ La Trobe University, Plenty Rd and Kingsbury Dr, Bundoora VIC 3086

❒ The Alfred Centre, La Trobe Allied Health Clinical School, 99 Commercial Rd, Prahran VIC 3181

Reinforce the voluntary nature of the study and re-affirm that the person can choose to withdraw their consent to participate at any time without affecting their relationship with the researcher or La Trobe University. Reiterate that at the start of the session, the person will need to change into a standard back-tie hospital gown within a private area of the laboratory and that the gown needs to be applied over bare skin.

Confirm that the person is fully satisfied with the information and/or explanation provided. If no, reinforce any areas of misunderstanding and/or confusion, and provide clarification as required.

Thanks so much. I'll now e-mail you an appointment vacancy schedule so that you can nominate a preferred day and time to attend the two study visits. Please do not hesitate to contact me should you have any other questions or queries about the study. You may also contact any member of the Research Team listed on Page 5 of 7 of the Participant Information Statement.

ANY OTHER RESEARCHER NOTES:

_____

_____

_____

END.

IF INELIGIBLE

1. Advise of ineligibility
2. Thank for interest in study and time

LA TROBE UNIVERSITY

APPENDIX F: PROSPECTIVE PARTICIPANT TELEPHONE SCREENING CHECKLIST (STUDY III)

Name: ____

✂
- -

SCREENING CHECKLIST

⚐ Complete a checklist for each prospective participant

Study title: **Skin Manifestations in Response to Local Pressure and Microclimate in Healthy Adults**

Screening completed by: ________________________________ Date: ____________ Screening #: _______

[Introduce self and role]

Thank you for calling and your interest in participation. The purpose of this telephone screening is to check if you are eligible to participate in this study, and it should take no longer than 15 minutes to complete. Please know that all the information you provide during the telephone screening will be kept confidential. Before I ask you some questions, did you have any questions about the study that you'd like me to answer?

[ANSWER QUESTIONS]

At the moment, La Trobe University has implemented restrictions specific to COVID-19 (Coronavirus) pandemic. Before I can enrol you in the study, I need to ask you some questions about whether you think you might have COVID-19 symptoms, if that's okay? It's important that you try to answer these questions truthfully or to your best ability. The information you provide us will be kept strictly confidential.

COVID-19 SCREENING QUESTIONS		
Do you have any of the following symptoms?	**Tick Yes or No**	
Fever (temp. >37.8°C) **call COVID-19 line 1800 675 398**	❐ YES	❐ NO
Cough	❐ YES	❐ NO
Shortness of breath	❐ YES	❐ NO
Sore throat	❐ YES	❐ NO
Do you have a diagnosed Respiratory Illness?	❐ YES	❐ NO
Have you returned from overseas in the last 14 days?	❐ YES	❐ NO
In the last 14 days, have you:		
Had known contact with a confirmed case of coronavirus?	❐ YES	❐ NO
Had known contact with a suspected case of coronavirus?	❐ YES	❐ NO

*If the person answers **YES** to **any** of the above questions, advise the person to immediately seek medical advice/guidance by calling the National COVID-19 Information and Triage Line on 1800 020 080 or COVID-19 Call centre on 1800 675 398. Inform the person that they will be able to contact the researcher for re-assessment of study eligibility once the person feeling well and/or symptom-free at the direction of their medical practitioner.*

*If the person answers **NO** to **ALL** of the above questions, go to the next section.*

Next, I will provide you with a brief overview of screening and infection control measures that are to be implemented in this study. These measures are necessary to enable safety of those involved in the study.

On the day of your scheduled appointment and designated time, you will meet with the researcher outside of the Main Entrance of the George Singer Building. Upon your arrival, the researcher will introduce themselves to you and, with your permission, take your temperature using an infrared temperature scanner. This scanner will be held 3-5 cm from your forehead, and a single reading will be taken. If your temperature is between 35.8°C to 37.5°C, you will be directed to the Nursing Laboratory where the study will take place. If your temperature is below 35.8°C or above 37.5°C, this could indicate that you might have an infection and/or fever, and the appointment will need to be re-scheduled to a later date

Please note, La Trobe University has developed COVID-19 guidelines that study participants must follow during all research activities. This means that upon entering (and leaving) the laboratory, you will need to wash your hands with anti-microbial soap at a designated wash basin **OR** sanitise your hands with an hospital-grade alcohol-based hand sanitiser.

Before I continue with the rest of the screening questions, did you have any questions you'd like me to answer?

[ANSWER QUESTIONS]

SCREENING QUESTIONS

What I can do then is ask a few quick questions to ensure that you meet the general study eligibility criteria, if that's okay?

[IF YES, PROGRESS TO SCREENING QUESTIONS]

AGE

ASK: [Mr/Mrs/Ms NAME], you are over the age of 18 years – is that correct?

❒ No (ineligible; thank and end call) ❒ Yes (go to next question)

ABILITY TO READ AND COMPLETE/SIGN CONSENT FORM

Do you mind me asking if you have been diagnosed by a medical practitioner with a cognitive impairment, or you suspect a cognitive impairment? ❒ No ❒ Yes

RESEARCHER RECORD: person meets *no cognitive impairment* criterion **Y / N** (If no, thank and end call)

Have you read the Participant Information Statement and Consent Form thoroughly? **Y / N**

Do you/did you require an interpreter to translate any information included in the Participant Information Statement and Consent Form for you? **Y / N**

So, can I clarify that you understand that the study involves:

1. Attending a single appointment (study visit), which is expected to take 4 hours of your time? **Y / N**
2. Avoiding the application of skin products (moisturisers, lotions, creams, emollients) to the skin surface *specifically* at the elbows, heels, or lower back for at least 24 hours prior to the appointment? **Y / N**

ASK: can you please tell me if you have any current wounds or any skin conditions (such as dermatitis, eczema, psoriasis, irritation, inflammation) at the elbows, heels, or base of the lower back?

❒ No (go to next question) ❒ Yes (ineligible; thank and end call)

ASK: do you believe that you have skin sensitivities and/or conditions that could be aggravated (made worse) when in direct contact with a warming surface for up to one hour at a time?

❒ No (go to next question) ❒ Yes (ineligible; thank and end call)

ASK: do you have any tattoos at the elbows, heels, or base of the lower back?

❒ No (go to next question) ❒ Yes (ineligible; thank and end call)

ASK: do you have any conditions/disorders that could affect your ability to lay stable for up to one hour at a time, or that could cause you to experience discomfort whilst lying down?

❒ No (go to next question) ❒ Yes (ineligible; thank and end call)

Thank you, those are all the screening questions I had.

IF ELIGIBLE

It appears likely that you are eligible for the study. If you are still willing to participate, could you please read and sign the Consent Form on Page 6 of the Participant Information Statement, and return it to myself on the day of your appointment?

RESEARCHER RECORD: person is willing to sign the Consent Form **Y / N** If no, thank and end call.

Reinforce the voluntary nature of study and re-affirm that the person can choose to withdraw their consent to participate at any time without affecting their relationship with the researcher or La Trobe University.

Confirm that the person is fully satisfied with the information and/or explanation provided. If no, reinforce any areas of misunderstanding and/or confusion, and provide clarification as required.

Thanks so much. I'll now e-mail you an appointment vacancy schedule so that you can nominate a preferred day and time to attend the appointment. Please do not hesitate to contact me should you have any questions and/or queries about the study. You may also contact any member of the Research Team listed on Page 5 of 7 of the Participant Information Statement.

Please note, if on the day of your appointment, you notice that you are feeling unwell or think you that may have symptoms of respiratory infection (such as fever, cough, shortness of breath, sore throat, fatigue), we ask that you please let us know before attending the study visit. Your appointment will be re-scheduled to a later date once you are feeling well and/or symptom-free at the direction of your medical practitioner.

ANY OTHER RESEARCHER NOTES:

_____

_____

_____

END.

IF INELIGIBLE

1. Advise of ineligibility
2. Thank for interest in study and time

NORTON PRESSURE INJURY RISK ASSESSMENT SCALE		
Physical condition	Good	4
	Fair	3
	Poor	2
	Severely limited	1
Mental condition	Alert	4
	Apathetic	3
	Confused	2
	Stuporous	1
Activity	Ambulant	4
	Walks with help	3
	Chair-bound	2
	Bedfast	1
Mobility	Full	4
	Slightly impaired	3
	Very limited	2
	Immobile	1
Incontinence	None	4
	Occasional	3
	Usually incontinent	2
	Incontinent	1
Total score (total sum of sub-scores): _____		

Greater than 18	Low Risk of Pressure Injury
Between 14 and 17	Medium Risk of Pressure Injury
Between 10 and 13	High Risk of Pressure Injury
Less than 9	Very High Risk of Pressure Injury

Adapted from:
Norton, D. (1962). *An Investigation of Geriatric Nursing Problems in Hospital*. Edinburgh, UK: Churchill Livingston.

FITZPATRICK SKIN PHOTOTYPE CLASSIFICATION

GENETIC (physical traits)					
Circle score					
Characteristics	**0**	**1**	**2**	**3**	**4**
What is the colour of your eyes?	Light blue, green, grey	Blue, green, grey	Dark blue, green, hazel	Dark brown	Brownish black
What is your natural hair colour?	Red	Blonde	Chestnut, dark blonde	Dark brown	Black
What is the colour of unexposed skin areas?	Pale white	White	Light brown or olive	Moderate brown	Dark brown to black
Do you have freckles on unexposed skin areas?	Many	Several	Few	Rare	None

SENSITIVITY (reaction to sun exposure)					
Circle score					
Exposure	**0**	**1**	**2**	**3**	**4**
What happens to your skin if you stay in the sun for an extended period of time?	Readily burns on exposure, blisters, peels	Moderately burns, blisters, peels	Sometimes burns or peels	Rarely burns	Never burns
Do you tan following sun exposure?	Never	Rarely	Sometimes	Often	Always
How tanned do you get?	Hardly or not at all	Light tan	Medium tan	Dark tan	Very dark
Is your face sensitive to the sun?	Very sensitive	Sensitive	Mildly sensitive	Resistant	Very resistant

INTENTIONAL EXPOSURE (tanning habits)					
Circle score					
Exposure	**0**	**1**	**2**	**3**	**4**
How often do you tan?	Never	Rarely	Sometimes	Often	Always
When did you last expose your skin to the sun or artificial tanning sources (tanning beds)?	More than 3 months ago	In the last 2-3 months	In the last 1-2 months	In the last week	In the last day
Total score (GENETIC + SENSITIVITY + INTENTIONAL EXPOSURE): _____					

Fitzpatrick Skin Phototype Classification

0-6	**Photo Type I**	**Pale White Skin** Example: red hair and freckles
7-13	**Phototype II**	**Fair Skin** Example: fair-skinned Caucasians, Northern Asians
14-20	**Phototype III**	**Fair to Beige Skin** Example: darker Caucasians, some Asians
21-27	**Phototype IV**	**Light Brown Skin** Example: Mediterranean & Middle-Eastern Caucasians, Southern Asians
28-34	**Phototype V**	**Brown Skin** Example: Southern Asians, some Hispanics, some Africans
35-40	**Phototype VI**	**Dark Brown to Black Skin** Example: darker Africans, Indigenous Australians

Adapted from:

Fitzpatrick, T. B. (1988). The validity and practicality of sun reactive skin types I-IV. *Archives of Dermatology, 124*(6), 869-871.

PARTICIPANT DEMOGRAPHIC AND HEALTH INFORMATION (STUDIES I & II)

PIN: _____ **Date**: _____ **Study site**: La Trobe University, Bundoora Campus

DEMOGRAPHIC INFORMATION:

DOB: _____

Current La Trobe University Student: ❐0 Yes ❐1 No

Country of birth: ❐0 Australia ❐1 Other; if Other, please specify: _____

Ethnicity: _____

Gender: ❐0 Female ❐1 Male ❐2 Third gender

HEALTH INFORMATION:

Norton Risk Assessment Scale (NRAS) score:

❐ low risk (≥18)

❐ medium risk (14-17)

❐ high risk (10-13)

❐ very high risk (≤9)

Note: Participants must receive a NRAS score of ≥18 to meet study eligibility criteria.

Height (m): _____ weight (kg): _____

BMI (kg/m^2): _____

❐0 Within normal range (18.5-24.9)

❐1 Underweight (<18.5)

❐2 Overweight (25-29.9)

❐3 Obese (>30)

Fitzpatrick Skin Phototype Classification:

❐1 TYPE I: Pale white skin

❐2 TYPE II: White skin

❐3 TYPE III: Light brown skin

❐4 TYPE IV: Moderate brown skin

❐5 TYPE V: Dark brown skin

❐6 TYPE VI: Dark brown to black skin

History of pressure injury:

❐0 Yes ❐1 No

If Yes, please specify both stage and location:

_____

_____

History of dermatological conditions:

❐0 Yes ❐1 No

If Yes, please specify both type and location:

_____

_____

PARTICIPANT DEMOGRAPHIC AND HEALTH INFORMATION (STUDY III)

PIN: _____ **Date**: _____ **Study site**: La Trobe University, Bundoora Campus

DEMOGRAPHIC INFORMATION:

DOB: _____

Current La Trobe University Student: ❐0 Yes ❐1 No

Country of birth: ❐0 Australia ❐1 Other; if Other, please specify: _____

Ethnicity: _____

Gender: ❐0 Female ❐1 Male ❐2 Third gender

HEALTH INFORMATION:

Norton Risk Assessment Scale (NRAS) score:

❐ low risk (≥18)

❐ medium risk (14-17)

❐ high risk (10-13)

❐ very high risk (≤9)

Note: Participants must receive a NRAS score of ≥18 to meet study eligibility criteria.

Height (m): _____ weight (kg): _____

BMI (kg/m^2): _____

❐0 Within normal range (18.5-24.9)

❐1 Underweight (<18.5)

❐2 Overweight (25-29.9)

❐3 Obese (>30)

Fitzpatrick Skin Phototype Classification:

❐1 TYPE I: Pale white skin

❐2 TYPE II: White skin

❐3 TYPE III: Light brown skin

❐4 TYPE IV: Moderate brown skin

❐5 TYPE V: Dark brown skin

❐6 TYPE VI: Dark brown to black skin

Melanin (baseline):

Elbows: ____ Heels: ____ Sacrum: ____

History of pressure injury:

❐0 Yes ❐1 No

If Yes, please specify both stage and location:

_____

_____

History of dermatological conditions:

❐0 Yes ❐1 No

If Yes, please specify both type and location:

_____

_____

LA TROBE UNIVERSITY

APPENDIX K: PARTICIPANT DATA COLLECTION FORM FOR REPEATED SKIN PARAMETER RESULTS (STUDY I - SUPINE)

PIN: _____ Date: _____ Time (hrs): _____ Room humidity (%):_____ Ambient temp. (°C): _____ Condition first allocated: INT ❐ UNINT ❐

Results for Interrupted Condition									Results for Uninterrupted	
Testing site	**Parameter**	**Measurement Interval [mins]**							**Measurement Interval**	
		$T0_{int\text{-}supine}$	$T10_{int\text{-}supine}$	$T20_{int\text{-}supine}$	$T30_{int\text{-}supine}$	$T40_{int\text{-}supine}$	$T50_{int\text{-}supine}$	$T60_{int\text{-}supine}$	$T0_{unint\text{-}supine}$	$T60_{unint\text{-}supine}$
Right elbow	Temperature (°C)									
	Epidermal hydration									
	Melanin									
	Erythema									
	Blanch test* [✓/✗]									
Left elbow	Temperature (°C)									
	Epidermal hydration									
	Melanin									
	Erythema									
	Blanch test [✓/✗]									
Right heel	Temperature (°C)									
	Epidermal hydration									
	Melanin									
	Erythema									
	Blanch test [✓/✗]									
Left heel	Temperature (°C)									
	Epidermal hydration									
	Melanin									
	Erythema									
	Blanch test [✓/✗]									
Sacrum	Temperature (°C)									
	Epidermal hydration									
	Melanin									
	Erythema									
	Blanch test [✓/✗]									

✓ represents blanching of the skin at a given anatomical testing site

✗ represents non-blanching of the skin at a given anatomical testing site; necessitates immediate application of stopping protocol

LA TROBE UNIVERSITY

APPENDIX K: PARTICIPANT DATA COLLECTION FORM FOR REPEATED SKIN PARAMETER RESULTS (STUDY I - SUPINE)

PIN: _____ Date: _____ Time (hrs): _____ Room humidity (%):_____ Ambient temp. (°C): _____ Condition first allocated: INT ❐ UNINT ❐

Results for Washout Period				
Testing site	**Parameter**	**Measurement interval [mins]**		
		T5	***T10***	***T15***
Right elbow	Temperature (°C)			
	Epidermal hydration			
	Melanin			
	Erythema			
Left elbow	Temperature (°C)			
	Epidermal hydration			
	Melanin			
	Erythema			
Right heel	Temperature (°C)			
	Epidermal hydration			
	Melanin			
	Erythema			
Left heel	Temperature (°C)			
	Epidermal hydration			
	Melanin			
	Erythema			
Sacrum	Temperature (°C)			
	Epidermal hydration			
	Melanin			
	Erythema			

LA TROBE UNIVERSITY

APPENDIX L: PARTICIPANT DATA COLLECTION FORM FOR REPEATED SKIN PARAMETER RESULTS (STUDY 2 - SEMI-RECUMBENT)

PIN: _____ Date: _____ Time (hrs): _____ Room humidity (%):_____ Ambient temp. (°C): _____ Condition allocated in visit 1: INT ❐ UNINT ❐

Results for Interrupted Condition									Results for Uninterrupted	
Testing site	**Parameter**	**Measurement Interval [mins]**							**Measurement Interval**	
		$T0_{int\text{-}semi}$	$T10_{int\text{-}semi}$	$T20_{int\text{-}semi}$	$T30_{int\text{-}semi}$	$T40_{int\text{-}semi}$	$T50_{int\text{-}semi}$	$T60_{int\text{-}semi}$	$T0_{unint\text{-}semi}$	$T60_{unint\text{-}semi}$
Right elbow	Temperature (°C)									
	Epidermal hydration									
	Melanin									
	Erythema									
	Blanch test [✓/✗]									
Left elbow	Temperature (°C)									
	Epidermal hydration									
	Melanin									
	Erythema									
	Blanch test [✓/✗]									
Right heel	Temperature (°C)									
	Epidermal hydration									
	Melanin									
	Erythema									
	Blanch test [✓/✗]									
Left heel	Temperature (°C)									
	Epidermal hydration									
	Melanin									
	Erythema									
	Blanch test [✓/✗]									
Sacrum	Temperature (°C)									
	Epidermal hydration									
	Melanin									
	Erythema									
	Blanch test [✓/✗]									

✓ represents blanching of the skin at a given anatomical testing site

✗ represents non-blanching of the skin at a given anatomical testing site; necessitates immediate application of stopping protocol

LA TROBE UNIVERSITY

APPENDIX L: PARTICIPANT DATA COLLECTION FORM FOR REPEATED SKIN PARAMETER RESULTS (STUDY 2 - SEMI-RECUMBENT)

PIN: _____ Date: _____ Time (hrs): _____ Room humidity (%):_____ Ambient temp. (°C): _____ Condition allocated in visit 1: INT ❐ UNINT ❐

Results for Washout Period				
Testing site	**Parameter**	**Measurement interval [mins]**		
		T5	***T10***	***T15***
Right elbow	Temperature (°C)			
	Epidermal hydration			
	Melanin			
	Erythema			
Left elbow	Temperature (°C)			
	Epidermal hydration			
	Melanin			
	Erythema			
Right heel	Temperature (°C)			
	Epidermal hydration			
	Melanin			
	Erythema			
Left heel	Temperature (°C)			
	Epidermal hydration			
	Melanin			
	Erythema			
Sacrum	Temperature (°C)			
	Epidermal hydration			
	Melanin			
	Erythema			

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APPENDIX M: PARTICIPANT DATA COLLECTION FORM FOR REPEATED SKIN PARAMETER RESULTS (STUDY III)

PIN: _____ Former Participant: Yes ❒ No ❒ Date: _____ Time (hrs): _____ Room humidity (%): _____ Ambient temp. (°C): _____

		Results: **Control**							Results: **Washout**			Results: **Heat Intervention**						Results: **Washout**			Results: **Moisture Intervention**					
Site	Parameter	Measurement interval [mins]							Interval [mins]			Measurement interval [mins]						Interval [mins]			Measurement interval [mins]					
		$T0_c$	$T10_c$	$T20_c$	$T30_c$	$T40_c$	$T50_c$	$T60_c$	$T5_{w1}$	$T10_{w1}$	$T15_{w1}/T0_h$	$T10_h$	$T20_h$	$T30_h$	$T40_h$	$T50_h$	$T60_h$	$T5_{w2}$	$T10_{w2}$	$T15_{w2}/T0_m$	$T10_m$	$T20_m$	$T30_m$	$T40_m$	$T50_m$	$T60_m$
Right elbow	Temperature (°C)																									
	Epidermal hydration																									
	Erythema																									
	Blanch test [✓/✗]																									
Left elbow	Temperature (°C)																									
	Epidermal hydration																									
	Erythema																									
	Blanch test [✓/✗]																									
Right heel	Temperature (°C)																									
	Epidermal hydration																									
	Erythema																									
	Blanch test [✓/✗]																									
Left heel	Temperature (°C)																									
	Epidermal hydration																									
	Erythema																									
	Blanch test [✓/✗]																									
Sacrum	Temperature (°C)																									
	Epidermal hydration																									
	Erythema																									
	Blanch test [✓/✗]																									

✓ represents blanching of the skin at a given anatomical testing site

✗ represents non-blanching of the skin at a given anatomical testing site; necessitates immediate application of stopping protocol