Original article

Current perspectives on pressure injuries in persons with dark skin tones from the National Pressure Injury Advisory Panel

ABSTRACT

Background Pressure injury (PI) development is multifactorial. In patients with dark skin tones, identifying impending PIs by visual skin assessment can be especially challenging. The need for improved skin assessment techniques, especially for persons with dark skin tones, continues to increase. Similarly, greater awareness of the need for inclusivity with regard to representation of diverse skin colours/tones in education materials has been apparent in recent years.

Objective To provide current perspectives from the literature surrounding skin assessment and PI development in patients with dark skin tones.

Methods The following elements will be discussed through the lens of skin tone: 1) historical perspectives of PI staging from the National Pressure Injury Advisory Panel, 2) epidemiology of PI, 3) anatomy and physiology of the skin, 3) skin tone assessment and measurement, 4) augmented visual assessment modalities, 5) PI prevention, 6) PI healing, 7) social determinants of health, and 8) gaps in clinician education.

Conclusions This review highlights the gap in our clinical knowledge regarding PIs in patients with dark skin tones. Racial disparities with regard to PI development and healing are especially highlighted among patients with dark skin tones. Skin tone colour assessment must be standardised and quantifiable in clinical education, practice, and research. This work is urgently needed, and support from private and governmental agencies is essential.

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Keywords dark skin tones, health equity, pressure injuries, skin assessment, social determinants of heath

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INTRODUCTION

Racial diversity across the United States has increased over the past 2 decades. The US Census Bureau reports that the overall diversity index has increased from 54.9% in 2010 to 61.1% in 2020 and is projected to continue to escalate over the next decade.[1] Worldwide, people with dark skin tones comprise the majority of the population.[2]

Over the past 20 years, there has been a growing awareness of and interest in the need for skin assessment that is inclusive of all persons regardless of their skin colour.[3-24] The clinical reality is that in the US and worldwide, skin tone demographics have shifted, and awareness of various skin colours has increased, such that the need for accurate skin assessment and diagnosis for all patients has taken on great urgency. Moreover, awareness of the need to include diverse skin colours/tones in education materials (basic professional and ongoing education) for students and clinicians has also increased in recent years.[13]

In this article, the authors outline current perspectives on pressure injuries (PIs) in patients with dark skin tones. The first section addresses the historical efforts of the National Pressure Injury Advisory Panel (NPIAP; formally the NPUAP) to address this disparity and revise staging definitions to be relevant for all skin tones. Additional topics addressed through the lens of skin tone include the epidemiology of PIs, anatomy and physiology of the skin, visual and augmented skin assessment techniques and modalities, PI prevention and healing, social determinants of health (SDOH) considerations, and professional education gaps.

HISTORICAL PERSPECTIVES ON PI STAGING IN PERSONS WITH DARK SKIN TONES

In the mid-1990s, clinicians and NPIAP board members recognised the need for the stage 1 definition to include how these PIs would appear in persons with dark skin tones.[1] They noted that assessments that identify a stage 1 PI, such as "erythema" and "non-blanchable erythema," were not always visible in patients with dark skin tones and, therefore, other indicators of stage 1 PI were needed. A task force was formed to address these issues.[2,4]

The NPIAP Task Force agreed on the following assumptions:[20]

- Intact skin has a variety of colour changes
- Very darkly pigmented skin does not have visible blanching
- The person's race and ethnicity are not predictive of skin pigmentation
- Non-blanchable erythema only reflected one description of change in skin colour seen in early PI; it was not a universal descriptor
- Other objective findings of stage 1 PI could include temperature changes (warmth, coolness, edema, induration).

Table 1 lists the various iterations of the definition over the years.[1,5,9,17,18,20]

In 2005, the NPIAP identified deep tissue PIs (DTPIs). As with stage 1 PIs, DTPIs are difficult to detect in patients with dark skin tones (Figures 1-3).[25] Other NPIAP initiatives that call attention to skin tone diversity include staging diagrams illustrating different skin colours/tones (available on the NPIAP website).

EPIDEMIOLOGY OF PIS IN PERSONS WITH DARK SKIN TONES

A limited number of studies have examined PI rates by race/ethnicity or skin tone in individuals admitted to acute care settings or long-term care facilities or residing in a nursing home. However, in a literature review of PIs in patients with dark skin tones, Gunowa and colleagues[26] report that patients with dark skin tones are more likely to develop higher stage PIs regardless of the type of healthcare setting. The following discussion outlines what is known regarding the prevalence/incidence of PIs among patients with dark skin tones delineated by healthcare setting.

PIs among admissions to nursing homes

Using the Minimum Data Set (MDS) version 2.0, PIs (stage 2, 3 or 4) were present in 15% of older adults admitted to US nursing homes.[27,28] Higher rates of PIs have been reported among Black individuals admitted to nursing homes. Approximately twice as many Black patients (16.6%) admitted to 59 nursing homes in Maryland had a PI compared with White patients (8.4%).[29] In a national chain of nursing homes, Harms et al[28] reported rates of PIs among older adults admitted to nursing homes by race and ethnicity (American Indian and Alaskan Native, Asian or Pacific Islander, Black not Hispanic, Hispanic, White not Hispanic). Black patients admitted to nursing homes had the greatest mean number of PIs per resident at 2.4 (SD, 2.2). The prevalences of stage 2, 3, or 4 PIs were lowest among White patients compared with all other racial and ethnic groups. The prevalence of a stage 1 PI among Black patients at admission was 7%, whereas stage 2 injury prevalence was 20%. Among all racial/ethnic groups, Black patients also had the highest prevalences of the most severe PIs at 7% (stage 3) and 8% (stage 4); White patients had the lowest prevalences at 3% for both stages.

PI prevalence among nursing home residents

Ahn et al[30] found that among individuals residing in all US nursing homes, 8.4% had a PI, and 1.7% had a suspected DTPI.[30] A greater percentage of Black residents (18.2%) compared with White residents (13.8%) had a PI in a set of nursing homes in New York.[31] In a study examining differences in the prevalence of PIs among high-risk residents in US nursing homes over 5 years, PI prevalence decreased for both Black and White residents. However, there was a 5.4% overall unadjusted difference in PI prevalence (higher in Black patients).[32] In a study examining the reporting of PIs by nursing homes using the MDS version 3.0, Chen et al[33] found that the percentage of stage 4 PIs was higher among short-stay Black residents (50.4%) compared with White residents (40.8%).[33] Black race was significantly associated with having a stage 2 to 4 PI (odds ratio [OR], 11.44; 95% CI, 1.44–1.47). Hispanic ethnicity was significantly associated with having a suspected DTPI (OR, 2.63; 95% CI, 1.47–1.58).

PI incidence in nursing homes

Cai et al[31] found that Black residents in New York nursing homes were more likely to develop a PI during their stay than were White residents, controlling for other risk factors (OR, 1.203; $P = .01$).[31] In another study, 7.7% of approximately 90,500 nursing residents developed stage 2 to 4 PIs after they were admitted to a nursing home. Black residents who developed a PI during their nursing home stay did so sooner than did White residents.[34] The disparity in time to development of a PI among Black residents increased over time: The disparity was 3% at 3 months post-admission and grew to 5.8% at 6 months. During a 12-week surveillance in a nursing home in Pennsylvania, a greater percentage of Black residents (47%) developed a stage 2 to 4 PI than White residents (18%).[35] Moreover, no stage 1 PIs were identified in Black residents.

PIs among hospitalised patients

Studies specifically examining PI prevalence and incidence in the acute care setting with a focus on racial distribution or patients with dark skin tones are scarce, with most studies conducted over a decade ago. In a large, multiyear prevalence study conducted between 1989 to 2005, Van Gilder and colleagues[36] found the proportion of stage 1 PIs in patients with dark skin tones to be much lower (13%) than in those with medium (32%) to light (38%) skin tones. This finding may be

Table 1. Npuap/npiap stage 1 pressure injury definitions: historical evolution

Year	Definition
1989	Non-blanchable erythema of intact skin, the heralding lesion of skin ulceration.[4]
1992	Non-blanchable erythema of intact skin; the heralding lesion of skin ulceration. In individuals with darker skin, discolouration of the skin, warmth, edema, induration, and hardness may also be indicators.[1]
1997	A. An observable pressure-related alteration of intact skin whose indicators as compared to an adjacent or opposite area on the body may include changes in skin colour (red, blue, purple tones). Skin temperature (warmth or coolness), skin stiffness, and/or sensation (pain).[1] B. An observable pressure-related alteration of intact skin whose indicators as compared to an adjacent or opposite area on the body may include changes in one or more of the following parameters: skin temperature (warmth or coolness), tissue consistency (firm or boggy feel), sensation (pain, itching) and or a defined area of persistent redness in lightly pigmented skin colour whereas in darker skin tones, the ulcer may appear with persistent red, blue or purple hues.[1]
2001	A stage I pressure ulcer is an observable pressure-related alteration of intact skin whose indicators as compared to the adjacent or opposite area on the body may include changes in one or more of the following: skin temperature (warmth or coolness), tissue consistency (firm or boggy feel) and/or sensation (pain, itching). The ulcer appears as a defined area of persistent redness in lightly pigmented skin, whereas in darker skin tones the ulcer may appear with persistent red, blue, or purple hues (p. 181).[5]
2007	Intact skin with non-blanchable redness of a localised area usually over a bony prominence. Darkly pigmented skin may not have visible blanching; its colour may differ from the surround area. In 2007 this was added to the definition: The area may be painful, firm, soft, warmer, or cooler as compared to adjacent tissue. Stage I may be difficult to detect in individuals with dark skin tones. May indicate at risk persons (a heralding sign of risk).[6]
2009, 2014	Intact skin with non-blanchable redness of a localised area usually over a bony prominence. Darkly pigmented skin may not have visible blanching; its colour may differ from the surrounding area. The area may be painful, firm, soft, warmer, or cooler as compared to adjacent tissue. Category/Stage I may be difficult to detect in individuals with dark skin tones. May indicate "at risk" persons (a heralding sign of risk). [7]
April 2016	Intact skin with localised area of non-blanchable erythema, which may appear differently in darkly pigmented skin. Presence of blanchable erythema or changes in sensation, temperature, or firmness may precede visual changes. Colour changes do not include purple, or maroon discolouration; these may indicate deep tissue injury.[8]

Abbreviations: NPIAP, National Pressure Injury Advisory Panel; NPUAP, National Pressure Ulcer Advisory Panel.

attributed to difficulty in detection of stage 1 PIs in patients with dark skin. More severe PIs (stage 3, stage 4, eschar) were also found among patients with dark skin tones as compared with those with light or medium skin tones (11% vs 6-7%, 13% vs 6-7%, and 9% vs 5-6%, respectively). In a large, multiyear national study using the National Inpatient Sample (NIS) database from 2008 to 2012, Bauer and colleagues[37] reported that among patients who identified as African American, PI rates were significantly higher than among all other racial groups at 2.4%; patients who identified as White had the second highest incidence reported at 1.8%. Moreover, the PI stage was more severe in African American patients (stage 3), whereas stage 2 was the most common stage among White patients.[37]

Using the NIS database from 2003, Fogerty and colleagues[38] identified that African Americans were more likely to be discharged from US hospitals with PIs in comparison with non-African Americans (OR, 2.3; no CI provided). No analysis of PI stage was conducted in this investigation. A recent investigation conducted by Cox and Thomas-Hawkins[39] echoes the results of these previous works. In this investigation of 17,781 patients with PIs using the 2018 Healthcare Cost and Utilization Project (HCUP) State-Specific Database (New Jersey), a higher proportion of patients identifying as Black had an admitting diagnosis of PIs (5.0% vs 3.5%; $P < .05$) as well as a higher proportion of stage 4 PIs (3.3% vs 2.3%) when compared to all other races combined. When secondary diagnoses of PIs were examined, Black patients had a significantly lower proportion of stage 1 PIs (4.7% vs 18%; $P < .05$) but a higher proportion of stage 4 PIs (28.7% vs 16.9%; $P < .05$) when compared to all other races combined. Limitations cited for this study include the single state nature of the data and a lack of multivariate analysis.

Collectively, this limited body of work highlights some important considerations with regard to PI reporting across healthcare settings. First, there is the paucity of recent studies that have considered or examined race or dark skin tones as a potential risk factor for PI development. This is important because the change in racial diversity across the US warrants investigation. Second, the similarities in PI rates across these limited studies are striking and may highlight the need for specific clinical and diagnostic tools in practice to identify impending PIs in patients with dark skin tones.

ANATOMY AND PHYSIOLOGY OF SKIN

The skin consists of two distinct layers: epidermis and dermis.[40-43] The epidermis is cellular and avascular, consisting of 90% keratinocytes which synthesize the strong, water-insoluble structural protein, keratin. The epidermis protects from water loss, shear, friction, and toxic irritants. It also prevents invasion of bacteria and other pathogens by three mechanisms: 1) a mechanical barrier, 2) an acid mantle (pH, 4-6.6) suppressing bacterial growth, and 3) shedding of skin cells to minimize bioburden.

The epithelium is composed of five layers of cells: stratum corneum (SC), stratum lucidum, stratum granulosum, stratum spinosum, and stratum basale.[40] Available evidence about epidermal layers with known differences in persons with dark versus light skin tones include the stratum corneum, stratum spinosum, and stratum basale.

The stratum basale is the deepest of the five layers of the epidermis.[44] A basal cell is a stem cell that is a precursor of the keratinocytes of the epidermis. All keratinocytes are produced from this single layer of cells, which are constantly undergoing mitosis to produce new cells. As basal cells divide, one cell moves toward the surface and the other remains to continue reproduction.

In addition to basal cells, Merkel cells and melanocytes are also present in this layer. The Merkel cell functions as a receptor responsible for stimulating sensory nerves that the brain perceives as touch. Melanocytes produce the melanin pigment, which gives hair and skin its colour and protects living cells of the epidermis from ultraviolet radiation damage.

Differences in skin anatomy and physiology between dark and light skin tones

Pigmentation is the most obvious difference in the skin among racial groups.[45] Racial variation is dependent on the quantity of melanin, amount of UV exposure, genetics, melanosome content, and type of pigments found in the skin. Four chromophores are responsible for the differences in colour found in human skin: hemoglobin, oxyhemoglobin, melanin, and carotenoids. Hemoglobin and oxyhemoglobin are responsible for the pinkish colour of white skin. Melanin accounts for the various brown shades in black and sun-tanned skin. Carotenes underlie yellow-orange pigmentation. Individuals with the most lightly pigmented skin have approximately half as much epidermal melanin as the most darkly pigmented skin types.

Differences in the SC have been reported between darkly pigmented skin (Fitzpatrick Classification Scale V and VI) and lightly pigmented skin (Fitzpatrick I/II/III).[46] Darkly pigmented skin has more layers of corneocytes than lightly pigmented skin at a mean of 21.8 cells versus 16.7 cells, respectively. No differences have been reported in the size or thickness of the cells, although the cell layers in dark skin tones are thought to be more compact, reflecting greater intercellular cohesion. Although study results vary, the rate of spontaneous desquamation may be 2.5 times higher in persons with dark skin tones than those with lighter skin tones, accounting for the higher frequency of xerosis in individuals with dark skin tones.[47] Differences in desquamation also vary by body site between individuals with dark versus light skin tones (eg, higher rates of desquamation of lightly pigmented skin on the cheeks and forehead).

Corneocyte size, quality, and phenotype are important because smaller cells usually correlate with epidermal (keratinocyte) hyperproliferation and development of dry skin from reduced lipid levels. Although study results vary, individuals with

dark skin tones have the lowest levels of ceramides (lipid) at approximately 50% of the SC ceramide levels of individuals with lighter skin tones.[2] Consequently, dark skin tones are linked to higher transepidermal water loss (TEWL) yielding lower water content (WC) of the SC. In addition, darkly pigmented skin has lower skin vascular reactivity to external factors (eg, vasodilators). Cumulatively, higher TEWL, lower WC, and reduced thermoregulatory skin response heighten the vulnerability of individuals with dark skin tones to PI development.[48] Although studies are inconclusive, reduced vascular reactivity also may hinder visualization of blanching erythema of stage 1 PIs.

Gefen proposes a mechanical strain model for dark SC.[48] Reduced WC in the SC may increase SC stiffness, resulting in less effective dispersion of friction and greater mechanical stress. Thus, a vicious cycle of shear damage occurs in persons with dark SC leading to progressive increases in TEWL. The WC in dark damaged SC decreases with increasing TEWL, further elevating mechanical stress concentrations. As TEWL increases, the skin becomes drier and more inflamed, heightening risk of skin injury from pressure, shear, and friction.

SKIN TONE ASSESSMENT AND MEASUREMENT

Variations in skin pigmentation, condition (dry or moist), and temperature; the presence of fluid or products; the visual acuity of the observer; and lighting are some of the factors that can influence the subjective nature of skin assessments performed by clinicians.[49] Visual inspection of skin changes related to pressure, such as blanching, redness, or erythema is complemented by touch and technology (Figure 4).[50,51] However, for visual inspection practices, standardization in terms and technique is key to ensure consistency among various clinician assessments. It is important to note that visual inspection alone is unreliable.[22,51] Table 2 provides key information on visual assessment for diverse skin tones to assist clinicians in identifying early skin changes.[22,52,53]

Skin tone classification scales

Classification of skin colour was often previously based on self-reported ethnicity and race. Colour categories based on observers' descriptions have not been widely systematised. The use of classification scales offers some regularity; however, the function is varied. In addition to the validity and scientific rigor in the development and design of the scales, the effectiveness of classification models is dependent on individual competence and inter-rater reliability, which varies from setting to setting. These scales offer a constitutive (baseline) skin colour, facultative skin colour (sun or UV exposure), and PI-induced change in skin colour.[22,54]

The Fitzpatrick Classification Scale was developed to identify complexion and tolerance to sunlight and designed as a measure of sun sensitivity.[22,53-57] The Fitzpatrick Scale is not reliable when evaluating skin exposed to UV radiation (environmental factors) and would be best used to assess skin not exposed.

The Munsell Colour System (MCS), designed by an artist for painting, describes qualities of hue, lightness (value), and intensity (chroma), and was initially used in soil research.[58] In healthcare settings, this scale has undergone minimal validity and reliability testing despite its use in skin tone assessments.[53,57,59]

The Massey-Martin Scale (MMS), also known as the New Immigrant Survey (NIS) Skin Colour Scale, is a skin tone survey instrument designed for skin tone observation.[53,60] The initial instructions for use avoided the comparison of the pictorial guide to the person's skin side-by-side, as this was intended for social surveys conducted in person (by memory for the observer) or by phone (results indicated as unknown).

The Skin Tone Colour Scale (STCS) system by Konishi et al[59] was designed based on the MCS and is intended for skin and lesion tone assessment. The system is vast in the selection of diverse skin tones making it the most comprehensive classification available to clinicians.

Overall, current terms to describe skin colour tone are subjective, imprecise, non-standardised, and can have offensive connotations. Visual assessment scales, such as the Munsell[61,62] and Fitzpatrick scales,[63] which use images of skin colour tones are limited in that they cannot represent the full range of tones among individuals or even between body locations of the same individual in a feasible and useful way.

Table 2. Visual skin assessment for diverse skin tones[22,54]

Baseline skin tone should be established in an area not frequently exposed to ultraviolet radiation
Use adequate lighting; the best lighting includes ambient or natural: - Avoid fluorescent lighting - Appropriate lighting includes halogen lamp or flashlight (eg, cell phone, or pen light)
Thoroughly cleanse area to be assessed: - Remove bodily fluids - Remove skin care products
Compare skin area to be assessed to surrounding unaffected area: - Compare same area to be assessed to opposite laterality when possible - Compare skin area to be assessed (eg, sacrum) to alternative unaffected area (eg, abdomen)
Compare moist skin area to be assessed to dry skin: - Note differences for taut and/or shining skin changes
Implement a standardised, valid, and reliable skin tone classification system intended for skin pigmentation
Visual inspection should be accompanied with assessment for temperature, erythema, and blanching via tactile inspection and palpation
Visual inspection should be accompanied with augmented visual technology when possible; consider standardising throughout the entire healthcare setting

Recognising these problematic issues of assessing skin colour tone, the British Association of Dermatologists (BAD) recommended that terminology about skin colour tone be neutral, based on objective measurements, and reflective of multiethnic populations.[6,64] The most objective measure is via genetically established skin tone by melanogenesis. Pigmentation presents where melanocyte cells produce melanin in the skin. Dadzie et al[6] and BAD proposed the use of eumelanin pigment nomenclature for skin tone description and developed the Eumelanin Human Skin Colour Scale (ESCS) specifically for visual skin assessment. The scale is based on and named after eumelanin, which comprises 90% of the pigment found in human skin. The ESCS has five categories of melanin index that is measured by light reflectance: eumelanin low (<25), eumelanin intermediate low (25-<50), eumelanin intermediate (50-<75), eumelanin intermediate high (75-<100), and eumelanin high (≥100).[6] There are small, lightweight, portable, easy-to-use instruments for measuring melanin index. The ESCS has potential for use in classifying outcomes of PIs such as incidence and healing and monitoring for disparities/inequities.

Early visual detection technologies and dark skin tones

In individuals with dark skin tones, it is especially challenging to visually identify early skin changes that may herald an impending PI. The use of augmented visual technologies such as subepidermal moisture (SEM) assessment technology and long wave infrared technology (LWIT) hold promise for early identification of skin and tissue changes before they are visible to the naked eye. Thus, use of these technologies may create a window of opportunity for targeted interventions before the visible and tactile manifestations of tissue damage occur.[65] Absent these technologies, this window is invisible to direct care clinicians. The subclinical nature of developing PIs has resulted in diagnostic latency, which then contributes to interventional latency. In individuals with dark skin tones, this lag results in a higher probability of PIs remaining undetected.[66] Early detection technologies may help resolve this problem.

SEM technology

Subepidermal moisture assessment technology is based on the contemporary understanding of PI etiology (Figure 5).[67,68] The onset of microscopic damage in the early development of PIs and DTPIs is consistent regardless of skin pigmentation. Cell and tissue damage triggered by sustained pressure, shear, and friction signal acute inflammatory responses. As the level of tissue damage increases, so does the inflammatory response.[67] This immune response results in interstitial edema. Localised edema or SEM is one of the earliest indicators of nonvisible pressure damage. The ICD-10 coding for stage 1 PIs characterizes this early damage as 'pre-ulcer skin changes limited to persistent focal edema.'[69] This subclinical progression of tissue damage is further described in the etiology chapter of the 2019 clinical practice guidelines for the prevention and treatment of PIs.[51]

The SEM scanner device is FDA approved as a PI management tool and is indicated for adults of all skin tones at risk of PI development. The noninvasive point-of-care device detects persistent focal edema and reports the results as a SEM delta (Δ) value (Figure 1). The SEM assessment technology measures changes in SEM between healthy and inflamed tissue.[70] Increased SEM values may indicate an anatomy-specific increased risk for PI development in all skin tones.[68]

Meta-analyses from systematic reviews report early detection of PI development via SEM assessments by a median of 5 days before visual assessments ($P \leq .001$).[71,72] In a dual-arm study of 175 participants (n = 48/175, non-White), SEM assessments reported a diagnostic sensitivity and specificity of 86.8% and 88% in detecting PIs, resulting in an area-under-the-curve significantly exceeding clinical judgement ($P < .0001$).[70] In a cohort study, SEM assessments detected developing stage 1 PIs 1 week earlier than a visible diagnosis of a stage 1 erythema (OR, 5.3; CI 1.87-15.11; $P < .001$) in individuals with dark skin tones (n = 11/66).[73] In a multi-ethnic clinical study, SEM measurements were statistically significant in detecting concurrent and future stage 1 PIs and DTPIs in both heels in residents with dark skin tones as per Munsell value (n = 68/417; $P < .001$).[74] An observational study of 15 patients (n = 4/15, Fitzpatrick type III and above) reported early indication of tissue damage based on SEM measurements that agreed with a later confirmation of suspected DTPIs via ultrasound-based identification of hypoechoic lesions and visual assessments.[75] In a retrospective study of 69 patients in surgical intensive care, nurses indicated SEM assessments enabled more accurate skin assessments in patients with dark skin tones (n = 29/69).[76]

Long-Wave Infrared Thermography device

Long-wave infrared thermography (LWIT) is a noninvasive, multimodal device for use in clinical environments. It incorporates LWIT with a camera to detect PIs before visual or tactile changes occur. The device assesses changes in skin temperature because localised heat, edema, and changes in tissue consistency are all typical warning signs for PI development.[77] The device can be useful in patients with light or dark skin tones. It is particularly helpful in detecting DTPIs, which can remain undetected on the skin for up to 72 hours (Figure 6).[9,78] This feature is particularly important for patients with dark skin tones because dark skin pigmentation can mask the typical deep colours of purple and maroon that serve as the heralding visual signs for a DTPI.

As a combination photographic and LWIT device, it uses two imaging modalities by measuring long-wave infrared radiation (energy emitted from the human body) to create the final digital images. The energy, or lack thereof, is created from blood flow, perfusion, and, ultimately, metabolic activity. The device uses a relative temperature differential to compare the environmental temperature with the adjacent skin temperature and adapts for intrinsic and extrinsic factors (eg, elevated core body temperature, room temperature). A cooler temperature in comparison with the adjacent skin indicates less perfusion and deeper ischemic damage; warmer temperatures indicate increased metabolic activity and inflammation.

The reliability and validity of using LWIT to detect PIs have been confirmed in several studies.[79-84] The 2019 International Pressure Injury Prevention and Treatment Clinical Practice Guideline identified thermography as an area of high research priority.[85]

PI PREVENTION IN PERSONS WITH DARK SKIN TONES

Pressure injury prevention is rooted in both risk assessment and routine, comprehensive skin and soft tissue assessment.[51] Current evidence-based prevention practices apply to persons with dark skin tones and should be implemented.[51,86] Because early PI detection is challenging in patients with dark skin tones, the identification of a later stage PI stage (at the time of discovery), has been reported when compared to lighter pigmented persons.[87] Incorporating enhanced skin assessment techniques and visual augmentation devices into clinical practice should be a considered to enhance PI prevention in an effort to close the gap in early-stage identification.

PI TREATMENT AND HEALING IN PERSONS WITH DARK SKIN TONES

The process of healing a PI includes hemostasis, inflammation, proliferation, and maturation involving numerous molecular mechanisms.[88,89] Healing is influenced by a patient's clinical factors and treatments received. In patients with dark skin tones, healing PIs and especially the surrounding skin may appear differently to clinicians than in patients with lighter skin tones. For example, hypopigmentation of newly re-epithelialised tissue may be visible at the wound margins of a healing full thickness PI in patients with dark skin tones. (Figure 7). Although a few studies have reported differences and disparities in the prevalence or incidence of PIs by race or ethnicity,[32,90] studies focused on the healing of PIs are far less common. Bliss et al[91] analysed a nation-wide dataset of MDS records of older adults admitted to nursing homes. Of 10,862 older (65+ years) individuals admitted to a nursing home with a PI, 44% had healed by 90 days. However, there was a significant overall disparity of 6% in the healing of PIs (stages 2 to 4) present on admission at the required 90-day assessment among Black residents. In a study reporting on time to PI development during nursing home stays,[90] 99% of all residents with a PI received treatment for it, thus no disparity was found in the number of treatments by race or ethnic group. However, in other healthcare settings, such as acute care or the home care setting, there is a lack of evidence regarding disparities with regard to PI healing rates and treatment among patients with dark skin tones.

SOCIAL DETERMINANTS OF HEALTH

The impacts of SDOH on PI development and treatment are largely understudied and, thus, unknown. The US Department of Health and Human Services defines SDOH within five domains: economic stability, education access and quality, healthcare access and quality, neighborhood and built environments, and social and community context.[92] Examination of these domains reveals potential health disparities and inequities across racial groups. Overall, people of colour have been disproportionately affected by and fare worse within all domains of SDOH compared with White individuals.[93] *Communities in Action: Pathways to Health Equity* states that health equity is crucial for the well-being of communities.[94] Although large gains have been made in healthcare coverage across racial/ethnic groups under the Affordable Care Act, people of colour remain more likely to be uninsured.[94] Disparities exist with regard to income as well. In 2021, higher median incomes were reported for households headed by Asian or White individual, whereas households headed by persons identifying as Black or Hispanic reported median incomes lower than the national median.[95]

In two studies focused on PI development and the impact of race, elements of SDOH (economic stability and healthcare access and quality) were operationalised through patients' income based on zip code of residence and health insurance payor status. Using the NIS database in 2009, Fogerty and colleagues[38] identified an increased risk for PIs among African Americans insured by Medicare and Medicaid compared with Caucasians. With regard to income, a higher proportion of African Americans were found in the lower income quartiles (50.6%) than Caucasians (21.4%). However, in multivariate analysis, the researchers reported no significant differences between races for PI risk based on either payor status or income. Cox and Thomas-Hawkins[39] reported a significantly higher proportion of Black individuals with PIs insured with Medicaid compared with patients with PIs from all other racial groups combined using 2018 HCUP state level data from New Jersey. With regard to income, reported by quartiles based on zip code of residence, a statistically significant higher proportion of Black persons with PIs resided in the lower income zip code quartiles and a lower proportion at the upper zip code income quartiles. In their sample, over 50% of Black patients with PIs lived in areas with reported incomes of $58,999 or less as compared with 19% of all other races combined. No multivariate analysis was conducted in this study. In a recent scoping review of the impact of SDOH on PI progression, Sasson and colleagues[96] found that detriments in SDOH related to food scarcity (as identified through ICD-10 codes) and Black race were both significant, independent predictors of longer PI duration.[96]

Two recent national reports have validated the health inequities experienced by patients with dark skin tones who have PIs. In 2021, the Urban Institute reported that Black patients in the US were 31.9% *less* likely to be admitted to hospitals considered high quality with regard to PI prevention.[97] Moreover, an Agency for Healthcare Research and Quality Disparities Report in 2019 identified that for both short- and long-stay nursing home patients, poorer quality of care associated with PIs was found among Black patients as compared with Whites patients.[98] It is plausible that the development or worsening of a PI is influenced by access to quality healthcare, and this may play a pivotal role in the higher rates of PIs among Black patients. Without access to

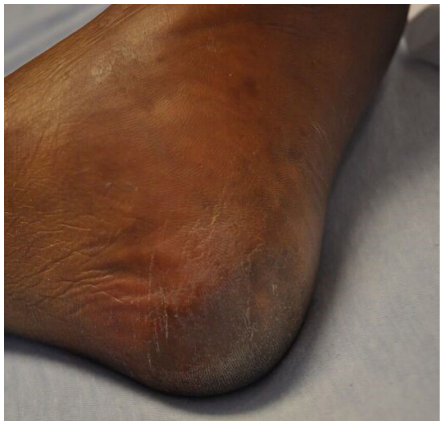

Figure 1. Deep tissue pressure injury of the heel. Image credit Joyce Black.

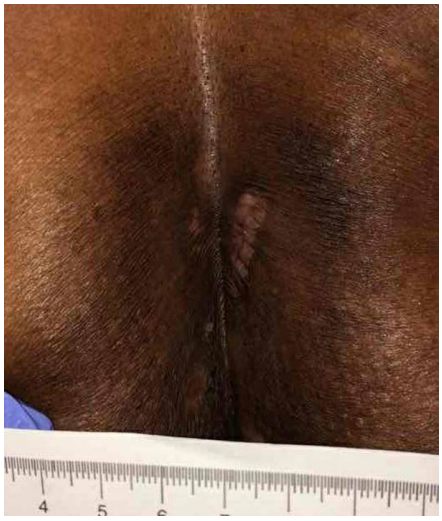

Figure 2. Deep tissue pressure injury of the buttocks with scarring from previous injury. Image credit Joyce Black.

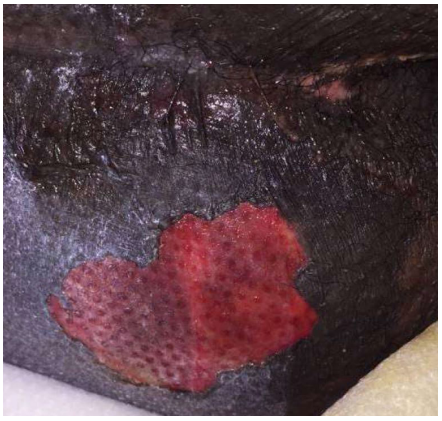

Figure 3. Deep tissue pressure injury evident by dusky, dark wound bed. Note the stippling pattern in the wound bed from the dressing. Image credit Joyce Black.

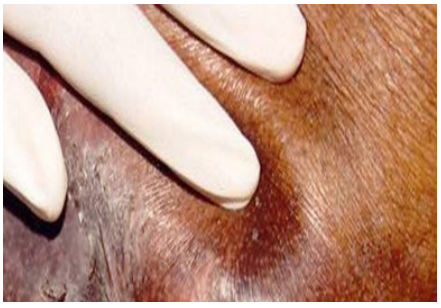

Figure 4. Palpation of the skin. Note, there is no visible change in skin colour. Image credit Joyce Black.

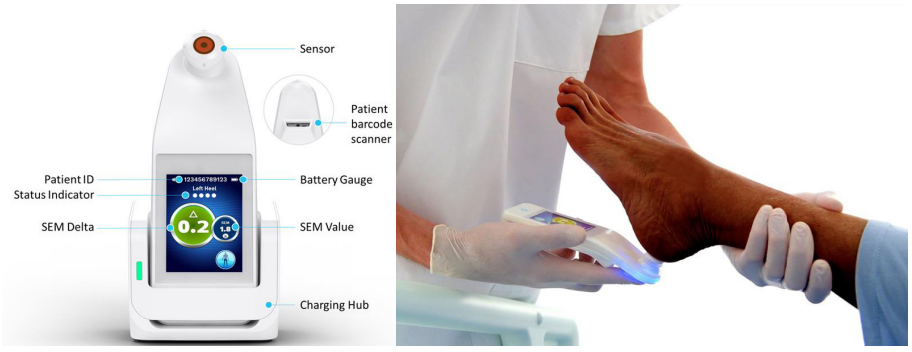

Figure 5. Subepidermal moisture technology. Images courtesy of Bruin Biometrics, LLC. Reprinted with permission.

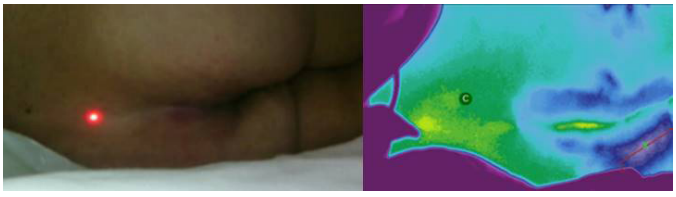

Figure 6. Thermal imaging of a developing deep tissue pressure injury Left), No visual changes are noted on a patient with darker skin tone. Right), Thermographic image illustrates an evolving deep-tissue pressure injury as seen by the yellow areas. The C marks the spot on the skin image that aligns with the camera image. Images courtesy of WoundVision. Reprinted with permission.

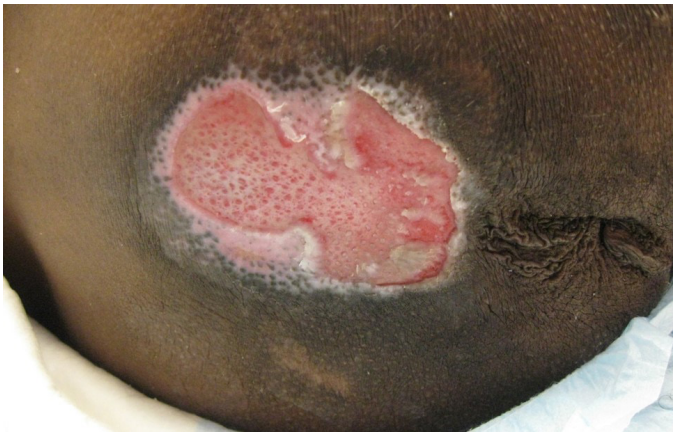

Figure 7. Healing stage 3 pressure injury. Note the hypopigmentation of the re-epithelialised tissue at the wound margins. Image credit Joyce Black.

evidence-based prevention or treatment modalities, the ability to prevent a PI or facilitate wound healing is compromised. At this time, the extent of this disparity has not been studied and is unknown. This is an area in need of further exploration.

CLINICIAN EDUCATION AND SKIN ASSESSMENT IN DARK SKIN TONES

Inaccurate skin assessment and lack of knowledge of PI appearance in dark skin tones can delay early identification and treatment, resulting in more severe PIs and financial penalties to the healthcare organization.[99] Health inequities and racial disparities occur when healthcare professionals, whether through explicit or implicit biases, fail to adequately assess, identify, and prevent PIs in people of colour who are at risk of a PI.[100,101] Inaccurate skin assessment can also occur because of the difficulty of visualising skin colour changes and discolourations in people with dark skin tones. Education

provided to nurses and other clinicians related to assessment of people with diverse skin colours is sadly lacking.

As nurses, our reputation among the public is one of honesty, trust, and caring. Nurses have been ranked as the most honest and ethical of professions for more than 20 consecutive years.[102] Yet, often nursing students emerge from our education programs not adequately prepared to understand the consequences of SDOH and the emphasis on providing safe and equitable care across the diverse populations we serve. Despite widespread goals to include diversity, equity, and inclusion and SDOH into nursing education curricula, whether this has been accomplished is uncertain.[99] Recent evidence demonstrates there is a health disparity in undergraduate nurse education with education directed toward people with pale skin tones.[99] Oozageer and colleagues[99] conducted a documentary and observational study of nursing education and lectures on PIs at five undergraduate nursing programs in England. The investigators found an overwhelming focus on PIs in people with Caucasian skin tones with only brief, superficial information on people with dark skin tones.[99] In a qualitative study in these five nursing education programs using focus groups, the investigators found a predominant theme of White normativity. Specifically, the investigators identified a dominance of Whiteness in the teaching about PIs and the implications for student nurses of Whiteness as the norm.[99]

In 2023, Pittman and Black[103] examined health equity in nursing education textbooks, specifically examining physical assessment textbooks' skin and integument content relevant to skin colour tones. Using a convenience sampling of physical assessment textbooks for undergraduate and graduate level nursing programs, the investigators modified Oozageer's Diversity Observation Teaching Tool (DOTT) with permission to better fit their project aims. The textbooks' content (ie, Integument/Skin Chapter) were reviewed independently, then data were reviewed for consistency or differences. Each investigator also explored their university's simulation lab for evidence of diversity in manikin skin colour tones. Of the nine textbooks and 11 chapters, no chapter objectives included skin tone diversity. Six of the nine textbooks had visual descriptors of PI, including stages. However, of the six textbooks with photos, only three had photos with dark skin tones. The textbooks had 534 photos of various skin graphics or images but of those, only 35 (7%) were of dark skin tones. Conversely, 499 (93%) of the 534 images were of light skin tones. Both universities had simulation labs with 60 to 65% of the manikins having light skin tones and 35 to 40% having dark skin tones. However, it is not known if skin tone is discussed in the simulation scenarios. These findings support those of Oozageer and colleagues and demonstrate the lack of education regarding skin tone diversity that nursing students receive.

CONCLUSIONS

Racial disparities exist with regard to PI development and healing, especially highlighted among patients with dark skin tones. This article explored the current state of the science and identified gaps in the terms used to describe skin colour, making any data-based comparisons and trending impossible. With increasing racial diversity in the United States, including persons of mixed ethnic and racial backgrounds, the "race" of a patient should no longer be used as a demographic term or a risk factor for PIs. Skin tone colour must be more standardised and quantifiable in clinical education, practice, and research. In the face of a more racially diverse country in the upcoming decades, the ability to identify and treat developing PIs earlier will improve the quality of life for all patients. This work is urgently needed and support from private and governmental agencies is essential.

CONFLICT OF INTEREST

The authors declare no conflicts of interest.

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