



Perspective



Cite this article: Hayes J, Santos MM.L.D, Higgins CA, Masen M. 2026 Perspectives on the future of skin–prosthetic interactions. *J. R. Soc. Interface* **23**: 20250549.
<https://doi.org/10.1098/rsif.2025.0549>

Received: 6 June 2025

Accepted: 13 January 2026

Subject Category:

Life Sciences–Engineering interface

Subject Areas:

bioengineering, biomedical engineering, biomechanics

Keywords:

perspectives, future, skin, medical, devices, interactions

Author for correspondence:

Jack Hayes
e-mail: jh6720@ic.ac.uk

Perspectives on the future of skin–prosthetic interactions

Jack Hayes^{1,2}, Margarida M.L.D. Santos², Claire A. Higgins¹ and Marc Masen²

¹Department of Bioengineering, and ²Department of Mechanical Engineering, Imperial College London, London, UK

JH, 0000-0003-1203-1229; MM, 0000-0002-9423-7193

Skin is filled with a rich network of sensory neurons, and consequently skin interaction with, and perception of the external environment, is a continual phenomenon experienced by all people in society. For medical devices such as prosthetics, the mechanical interaction between skin and the device is an essential aspect for device function, but it must be minimized to ensure continued skin health, which is inherently intertwined with biological processes. Numerous factors influence the interface properties of skin. For example, hydration, which influences skin morphology and composition, in turn influencing skin mechanics that may lead to tissue inflammation and skin injury. Here, we review the current state-of-the-art in skin medical device tribology with a particular focus on skin–prosthetic interactions. We split this article into traditional approaches to skin friction and a biology-first approach to skin friction, with a paradigm shift of skin as an engineered material. The field of tribology has historically been an interdisciplinary field comprising engineers, chemists and physicists, but future developments are needed in skin biology to drive meaningful change. We envisage that the integration of both clinical and biological perspectives will drive future innovations towards improved medical device interactions with the skin, when paired with engineering perspectives.

1. Introduction

Skin plays an important role in how we perceive and interface with our environment [1], ranging from heat and moisture management to tactile or pain stimulus detection. Critically, skin and its appendages provide a barrier to abrasion, infection or UV damage and protect underlying soft tissues [2]. Previous reviews have focused on the role of biotribology in everyday life [3], fingerpad and product interactions [4], artificial joints and dental implants [5]. However, the specific role that skin plays in medical device interactions has received less attention than articulating artificial joints such as hip or knee replacements. In particular, the impact that system properties have on the developed skin friction is an active area of research [6]. Much of the literature in skin biotribology takes an engineering-first approach which neglects the fact that skin is a dynamic and adaptive biological material. Additionally, the skin's response to loading will be biological in nature, and inflammation and skin dysregulation may onset before the visible rupturing of skin. Medical devices provide substantial benefits to users but have some major limitations, where much attention has been directed towards the manufactured medical device and less afforded to the device–skin interface. In this review, an outline of current research into skin tribology with medical devices will be presented, with a particular focus on prosthetics and an overview of the interdisciplinary challenges present in prosthetic socket design.

2. The role of friction on skin

Skin responds in two very distinct manners to friction depending on loading modality, environmental conditions and anatomical location. Friction against the skin's surface can elicit pleasurable sensations with the most pleasant responses reported at speeds of 3 cm s^{-1} and normal forces of 0.2 N [7]. In contrast, higher pressures lead to skin irritation—this effect is exacerbated by the presence of shear stresses [8,9]. While the exact mechanisms that lead to skin breakdown are still being investigated, it is generally accepted that prolonged pressure and the addition of shear lead to skin breakdown. Klaassen summarizes two distinct mechanisms which lead to tissue inflammation, degradation and injury onset [10]. Firstly, combinations of pressure and shear lead to occlusion of blood vessels, build-up of waste products within the skin coupled with restriction of oxygen and nutrients provided by the vasculature leading to tissue damage over a longer time course (few hours). Secondly, high strains on individual cells lead to programmed cell death (apoptosis) or spontaneous cell death (necrosis), resulting from events such as rupture of the cell membrane, leading to subsequent degradation of the tissue over a shorter time course (few minutes) [10]. In parallel, inflammation caused by these two mechanisms leads to an increase in inflammatory cytokines. Research in type I pressure ulcers, so before skin breakdown, has shown elevated IL-1 α , IL-1RA, IL-8, G-CSF and an increased ratio of IL-1 α /IL-1RA in ulcer sites compared with patient-matched control skin [11].

Onset of skin irritation and injury can be exacerbated by high friction and shear stress on the skin due to the presence of high deviatoric stresses, which cause large deformations in soft tissues [12]. Developed skin friction is influenced by intrinsic factors such as sex, anatomical location and skin hydration status [13]. It is important that future skin research considers the varying needs of diverse populations, in terms of age, skin tone and sex [14]. Previous studies have reported that eumelanin-rich skin shows different structures and cutaneous responses to inflammation and scarring [15,16], meaning device–skin interactions pose larger risks for some. However, dermatological literature on darker skin phototypes is still limited. The majority of research studies are performed in Fitzpatrick scale I–II skin, making it harder for researchers and designers to build equitable products that cater to different needs [14]. Sex is also another consideration. Women's monthly hormonal cycles, or hormonal changes during perimenopause and menopause, are known to affect the skin, impacting skin thickness and wound healing capacity among other factors [17,18]. However, most tribological studies do not take this into account or attempt to link skin mechanics with these hormonal changes.

In addition to these intrinsic factors, extrinsic biophysical factors such as environmental temperature and relative humidity [19] and contacting material properties [13,20] also alter skin friction. These intrinsic and extrinsic factors influence the biological function of the skin and in turn alter its mechanical properties, subjecting the skin to greater deformation while also impairing repair and remodelling mechanisms essential for barrier reformation [2].

Despite advances in prosthetic devices, skin injuries remain an unmet clinical need for lower limb amputees. Pressure has been reported in several studies in the prosthetic socket, with direct pressures measured at 34–149 kPa during standing and 58–342 kPa during walking [21–25]. However, owing to the largely incompressible nature of soft tissue, these high peak pressures recorded on the surface give little indication of underlying tissue health [26]. Instead, shear stress may give a better indication to tissue health in a prosthetic socket. An amputee's residual limb is subjected to high levels of friction and shear to keep the prosthetic in contact with the skin and transfer the body load efficiently. Lower limb amputees using suspension systems require friction for device functionality, despite friction's role in skin breakdown. Socket shear stresses have been measured at 27–134 kPa and increase the likelihood of developing skin injuries [21,23]. Consequently, reports of skin problems in lower limb amputees range from 34% to as high as 75% [27–31], suffering a disproportionately higher skin injury rate (65%) when compared with the general population [32].

3. Traditional approaches to skin–medical device interactions

Medical devices, such as prosthetic limbs, personal protective equipment (PPE), non-invasive ventilation masks, splints, braces and bandages/medical adhesive tapes to secure the devices, can be attributed to one-third of hospital-acquired pressure ulcers [33,34]. Several groups have outlined the importance of regular skin assessments at areas of medical device contact and suggest a need for interdisciplinary medical teams and engagement with medical device designers to reduce risk of skin injury [35,36]. Skin friction in a prosthetic socket is a complicated and multifactorial problem consisting of an interplay between contact mechanics and biological feedback. Approaches to characterize and investigate skin friction highlight the importance of system properties [6] (figure 1). In the case of the residual limb, this interfaces with the prosthesis through a sock and silicone liner; this interaction helps keep the prosthetic device in place and allows load transfer to enable the user to walk.

3.1. Engineered material

Material properties of the countersurface ('Engineered Material' in figure 1) are known to influence friction intensity. In particular, higher friction coefficients are recorded in prosthetic socks made from wool and nylon due to coarse fabric weaves and protruding fibres, which lead to microscopic skin trauma [37]. Properties of contacting engineered materials also have effects on reducing friction, where Klaassen *et al.* found that engineered surfaces with a $4 \text{ }\mu\text{m}$ surface roughness and higher stiffness (0.91 MPa) are more beneficial in scenarios where static friction needs to be minimized, and conversely softer materials (0.45 MPa) would ensure sticking (high static friction) and prevent dynamic sliding [38]. Altering interface friction in response to altering user requirements and biophysical conditions could help improve device efficacy. Efforts to reduce interface pressures by using softer contacting materials in hospital beds have shown limited efficacy in reducing prevalence of pressure

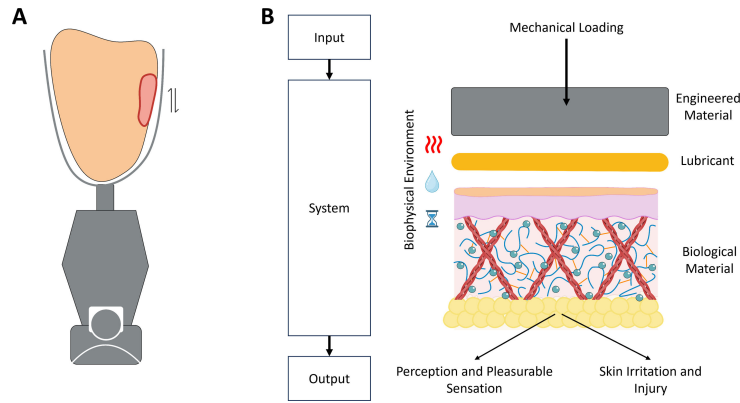


Figure 1. Skin tribology system in a prosthetic socket. (A) Schematic of shear present at skin surface of prosthetic socket. (B) Schematic demonstrating the crosstalk of engineering and biology that leads to skin discomfort and injury. All diagrams created in BioRender and Inkscape.

ulcers [39]. Oomens *et al.*'s computational analysis of a seated buttock showed that reducing the stiffness of the engineered material by a factor of 16 has minimal effects on the internal strains in the deep tissue [26]. However, Boyle *et al.* find that the addition of lateral pressure counterintuitively reduces internal deformations, providing a potential solution to ulcer risk patients in wheelchairs or bed-bound individuals [12]. Tanriverdi *et al.* trial the use of an adaptive prosthetic socket to account for volume fluctuations in residual limbs and find improved self-reported residual limb health [40]. While reducing interface pressures have yielded inconsistent results, Reuvekamp *et al.* provide a comprehensive review of future research directions and desirable outcome measures for functional surface engineering to control the microclimate of the skin–medical device interface [41]. The ideal microclimate for healthy skin is defined as 40–65% relative humidity, skin hydration levels of 10–20% and skin temperature at 16–33°C [41]. Socket microclimates have been reported at 80–90% relative humidity [42]; Reuvekamp *et al.* hypothesize the use of surface engineering techniques, such as patterned and porous surfaces to harvest and transport unwanted moisture, could alleviate these negative conditions [41].

3.2. Lubricant

Skin friction is defined by a non-interacting two-term equation [43] and is thought to be more strongly influenced by surface adhesion and surface chemistry [44]. Sebum is a fluid film on the surface of the skin and is a mixture of lipids released by sebaceous glands, located near hair follicles, that spreads across the surface of the skin [45]. Sebum is believed to play a role in UV protection, antibacterial capacity and have a function in inflammatory responses [46]. It has also been suggested that sebum acts as natural lubrication for hair follicles [47] and surrounding skin [46]. Preliminary research into the effects of sebum on skin friction has indicated weak correlations between lipid content and friction [48–50]. In sebum-poor areas such as the abdomen, the sebum thickness was less than 0.5 µm and the coefficient of friction was measured at 0.12 [51], while the forehead, where sebum thickness was greater than 4 µm, measured 0.34 [45]. While there is little correlation between poor and rich sebum areas, a deficiency of sebum does have a dramatic effect with a complete loss of surface sebum leading to a threefold reduction in the recorded coefficient of friction (0.3 versus 0.9) [48].

We (M.M.) previously argued that contributions of adhesion and deformation to skin friction are load dependent. The adhesive contribution to the coefficient of friction reduces with increasing load, while the deformation contribution to skin friction increases with increasing load [6]. Influencing the shear strength of the interface by addition of a lubricant can also reduce skin friction. Investigations by Yap *et al.* into healthcare PPE highlight optimal mixtures as 20 wt% beeswax, 40 wt% olive oil and 40 wt% mineral oil, which led to an 87% reduction in shear [52]. However, the complex nature of how lubricants interact with skin and alter the contact's system properties mean that emollients and creams should be avoided due to their secondary effects of altering tissue mechanics [53]. In the case of skin–prosthetic interactions, friction is a design requirement in non-osseo integrated designs. Reducing friction at the prosthetic–skin interface would lead to reductions in functionality, secondary pain in posture resulting from misalignment of the prosthetic and greater risk of falls from device misalignment.

3.3. Biophysical environment

Skin mechanics are influenced by the biophysical environment and as such must be controlled due to the relationship between soft skin mechanics, shear and resulting inflammation. Boyle *et al.* also highlight the interplay of skin mechanics on excessive deformations and friction in ulcer aetiology, which they term 'deformation ulcers' [12]. Skin tribology studies have been conducted in healthcare workers evaluating the use of PPE on soft tissue health. These revealed that short-term changes such as altered temperature and humidity levels greatly affect skin behaviour as seen when using facial PPE for prolonged periods of time [52–54]. Biophysical environments with elevated humidity also act to soften the skin, causing skin to conform more to the mask, increasing contact area and the associated adhesive forces [55] and leading to reduced tissue tolerance [56].

Occlusive environments are a pertinent problem with non-invasive ventilator masks [57–59] and incontinence products [60,61], with the effects being particularly pronounced in paediatric patients [58]. The use of accurate three-dimensional facial scans allows the creation of customizable masks, catering to diverse sizes and facial shapes [57]. Users of absorbent incontinence

products, such as back sheets, have increased risk of incontinence-associated dermatitis (IAD); nursing home residents have IAD incidence rates of 5.5% [60], while critical care patients have 29–36% [62] making it a prevalent issue. The TENA SmartCare Change Indicator tracks urine saturation in incontinence pads, reducing the time skin spends in contact with urine under pressure by indicating when a pad needs to be changed. When tested in a large-scale clinical study, it improved care efficiency and sleep quality of care home residents [63]. While the use of technology to help with incontinence is still new and developing, it is an example of how technology and biological understanding can be synergized to improve skin health.

Efforts to reduce skin injuries from prosthetic use have been focused on engineering-first solutions to reduce pressure and shear at vulnerable locations. A recent review by Zhang *et al.* suggests that skin friction is often required to maintain functionality of the medical device [64]. However, shear negatively impacts soft tissue health with the environment created by a prosthetic socket impacting skin mechanics, increasing vulnerability to skin irritation and injury. Some promising advancements in adaptive sockets that can grow with the user, or engineered surfaces and control measures that can dynamically modify the microclimate, can mitigate exacerbating factors that worsen skin health. However, a major part of the interface remains unaltered and under-investigated. Here, we explore the potential to vary skin properties in the prosthetic environment as a novel approach in skin tribology research.

4. Biology-first approach to the medical device interface

4.1. Skin biology

Skin is an inhomogeneous adaptive material with three main functional layers: the epidermis which interfaces with the external environment, the underlying dermis which provides mechanical support, houses functional structures like glands, hair follicles and vasculature and the hypodermis (fat layer) which dissipates mechanical force (figure 2) [47,65]. Skin principally acts as a two-way barrier providing mechanical protection from the external environment and retaining water within the body [65]. Skin structure and barrier function are established and maintained by a complex interplay of biological materials such as lipids and continued expression of keratins, a structural intermediate filament found in cells within the skin epidermis [66] (figure 2).

4.2. Dermis

The dermis is approximately 2–5 mm thick depending on body site [67] and the predominant cell type is fibroblasts. Fibroblasts play a role in tissue architecture [68], wound repair [2] and orchestrate overlying epidermal development [69,70]. The dermis can be split into two distinct regions. The papillary dermis lies directly beneath the epidermis and comprises approximately 10% of the dermal thickness [71]. Here, fibroblast cell density is highest and elastin and collagen content is lower [47]. Below lies the remaining 90% of the dermis known as the reticular dermis. This layer is characterized by a higher abundance of collagen fibres and lower cell density [47]. Fibroblasts are traditionally defined as being derived from the reticular or papillary dermis; however, single-cell sequencing of human skin has highlighted four distinct fibroblast subpopulations [72], while spatial transcriptomics have outlined six fibroblast subpopulations in healthy skin [73].

4.3. Epidermis

In contrast to the dermis, the epidermis is avascular and therefore relies on diffusion through the barrier and between cells for oxygen, nutrients and water (figure 3). This layer is stratified squamous epithelium consisting mainly (approx. 90%) of keratinocytes, with a small subpopulation of specialized cells [47]. Cells delaminate (exit the basal layer) then undergo differentiation coupled with stratification to move up through the epidermis to reach the stratum corneum.

The stratum corneum is the most superficial layer of the human epidermis (figure 3) which acts as a barrier and interacts with the external environment. Cells in the stratum corneum are sloughed off approximately 28 days after they have delaminated from the basal layer [47]. The stratum corneum comprises a lipid matrix aggregated with protein-rich anucleated keratinocytes termed corneocytes (figure 3). Vyumvuhore *et al.* found that at 9% tensile extension of excised human abdominal stratum corneum, intercellular lipids become more disorganized (gauche transition) [74] and *in vivo* rubbing of skin leads to reductions in lipid content in the superficial stratum corneum [75,76]. Maintaining a healthy barrier is pivotal to preventing skin dermatoses in lower limb amputees. However, recent work has shown prosthesis use leads to reduced skin health [76].

4.4. Skin as an engineering material

Skin is a heterogeneous composite structure, which is pre-tensioned *in vivo*, exhibits time-dependent behaviour, hyperelastic strain behaviour and direction-dependent mechanics. In addition, all of these factors vary depending on anatomical location, current general health status, skin pathologies, biophysical environment and demographic of individual.

Skin layers contribute uniquely to bulk tissue mechanics. The stratum corneum has the highest elastic modulus followed by the viable epidermis and the more compliant dermis [77–81]. The stratum corneum plays an important role in the overall mechanical response of the skin; however, a large range of elastic moduli of all skin layers has been recorded in the literature [78,79]. This has been largely attributed to extraction methods for measuring isolated stratum corneum and the hydration state of the tissue. The effective modulus of the stratum corneum has been shown to vary between wet and dry conditions, being over

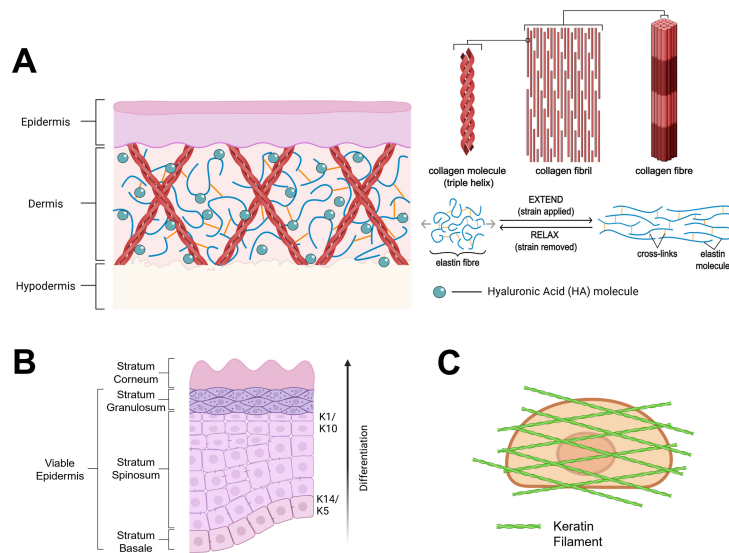


Figure 2. Skin organization and the load-bearing structures present. (A) Whole skin diagram, with collagen and elastin architecture. (B) Stratification and keratin distribution in the epidermis. (C) Keratin reinforcement of the cytoskeleton in individual keratinocytes. All diagrams created in BioRender and Inkscape.

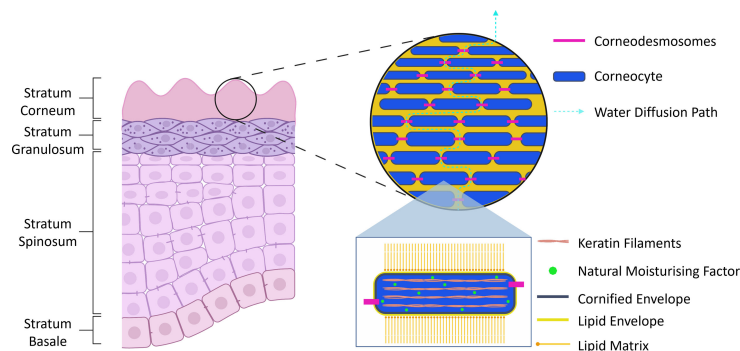


Figure 3. Human skin epidermal architecture. All diagrams created in BioRender.

600 times stiffer in the dry condition compared with hydrated condition (4000 versus 6 MPa) [82] which further implicates skin hydration and microclimate as an important player in stratum corneum health and mechanics.

Mechanics of the viable epidermis are driven by keratinocyte mechanics [83] where the main structural proteins are keratins. Isolated keratinocytes have an indentation stiffnesses of 1–3 kPa [84], but knockdown of *in vitro* keratin 5 expression led to keratinocytes with half the stiffness of control keratinocytes (412 versus 752 Pa) [85]. Within the dermis, the mechanics are dominated by collagen, produced by resident fibroblasts, which themselves have mechanics of 0.9 kPa [86]. Elastic energy storage occurs in the flexible regions of the collagen fibrils and stress transfer occurs through rigid sections of the molecule [87]. Mechanical load is bore through collagen, which is 10-fold stiffer than elastin [88]. The mechanics of the dermis reduces in stiffness by a factor of 3.5 as a result of age-induced disruption of collagen fibres [87].

Skin is a dynamic biological system and its mechanics can change because of medical device interaction. For example, after long-term prosthetic use, total lipid contents (ceramide and fatty acids) are significantly ($p < 0.05$) lower in the stratum corneum of residual limb skin compared with the intact limb, suggesting impaired barrier function after interaction with the medical device [76]. Natural moisturizing factor is also reduced at the skin's surface due to prosthesis use, which in turn leads to higher water content near the skin's surface. These biological changes have large-scale effects on soft tissue mechanics, causing a softening of skin by a factor of 3 and an increase of skin friction coefficient from 0.6 to 0.7 [76]. In turn, this leads to higher levels of inflammatory cytokines IL-8, IL-2 and IL-1 β in residual limbs compared with intact limbs [76]. In a cycle of events, the augmented tissue mechanics (softer skin and increased friction coefficient) after prosthesis use makes skin more susceptible to deformation-based injuries caused by prosthesis use. This is reflected in high skin injury statistics for prosthesis users and highlights that the role of the tissue in skin–medical device interaction plays just as an important role as the device itself.

5. Augmenting skin–medical device interaction by changing the skin

As skin–prosthetic interactions depend on properties of both the device and the skin, research to date has largely focused on altering medical device interface properties to augment the interaction. However, we argue here that another approach should be considered in parallel with innovative engineering solutions in future research and that is to alter the skin instead [89]. Skin is the largest organ on the body and as such exhibits large regional heterogeneity enabling it to fulfil its function according to location. For example, skin on the soles of the feet is 1.6-fold stiffer in indentation and 2.4-fold stiffer under shear loading [78].

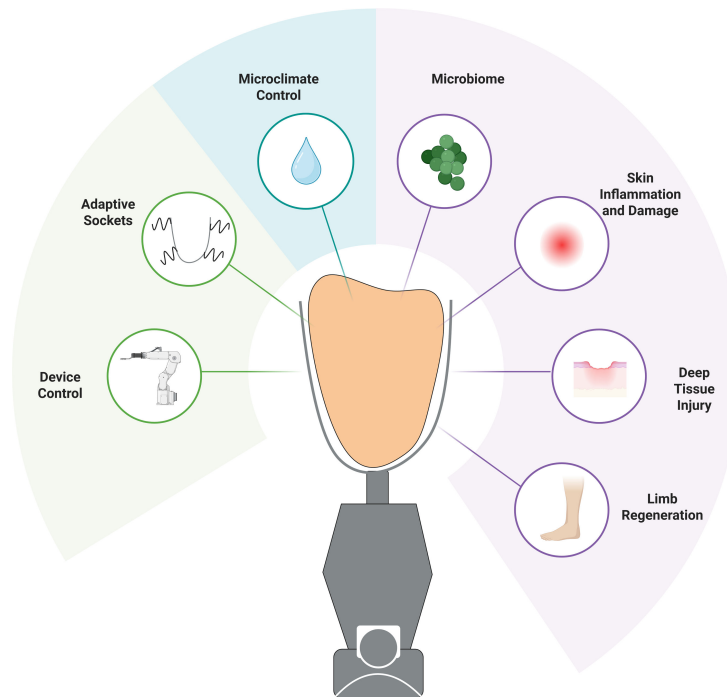


Figure 4. Interdisciplinary challenges in prosthetic provision.

The composition of palmoplantar skin protects it from a diverse range of adverse mechanical stimuli [78]. In contrast to the features present on the plantar heel, skin on a lower limb amputee's residuum lacks unique keratin expression and morphological features to confer improved load bearing, resulting in high skin injury prevalence. Re-engineering of non-load-bearing skin, such as that on the residuum, towards a load-bearing phenotype that mimics plantar skin mechanics has been predicted to reduce shear strains by 47%, which would consequently reduce failure by biological fatigue [89]. Altering the mechanics of residual limb skin would allow keratinocytes in the epidermis to resist higher stresses without rupture, while increased stiffness in the dermis would allow greater resistance to distortion under the shear loading. One approach for augmenting the skin is using cyanoacrylate liquid skin protectant treatment which leads to an increase in the stiffness of the skin and an approximately 40% decrease of the coefficient of friction between the treated skin and contacting material [90]. These changes, however, are not permanent and ultimately could reduce effectiveness of a prosthesis.

Another approach is to graft plantar, load-bearing skin, directly to the residual limb which would enable skin to be weight bearing. Fuchs *et al.* demonstrated that when human plantar skin-derived microtissue columns were transplanted to the trunk of a mouse, they retained their plantar skin phenotype [91]. However, the key question is how can you re-engineer non-plantar skin to a plantar phenotype. For this, we can learn from nature as it is well accepted that skin fibroblasts orchestrate both dermal and epidermal identity during skin development [92]. Yamaguchi *et al.* first investigated the effects of co-culturing plantar fibroblasts with non-plantar keratinocytes, showing that the plantar fibroblasts could induce expression of a palmoplantar-specific keratin (KRT9) in non-plantar keratinocytes in as little as 30 min [93]. When plantar and scalp fibroblasts are compared in an *in vitro* three-dimensional skin equivalent model, changes in the epidermis are induced including increases in epidermal thickness (80 versus 60 μm) and increased plantar-specific keratin expression (1.5-fold versus 1.0-fold) for plantar and scalp fibroblasts, respectively [94]. Collectively, these data suggest that plantar fibroblasts have the capacity to reprogramme non-plantar keratinocytes to a plantar, and therefore theoretically load-bearing, phenotype.

The idea that plantar fibroblasts could reprogramme skin on the residuum to a plantar, load-bearing phenotype was recently tested in a phase 1 clinical trial with 39 lower limb amputees [94]. The study investigated the therapeutic potential of injection three times per week of 10 million isolated and cultured human plantar fibroblasts. The authors found that 17 months of plantar fibroblast injection leads to increased elastin content, increases in KRT9 expression and increases in epidermal thickness [94], which suggest a transition towards a plantar identity. The authors quantified skin 'firmness', with a Durometer (Buffalo Grove, IL, USA), confirming previous studies [78] that palm skin is substantially firmer than non-palmoplantar skin. However, they found that injection of fibroblasts, regardless of the anatomical location from which they were harvested (plantar skin or scalp), led to increases in skin 'firmness' [94]. This suggests that using plantar fibroblasts to re-engineer skin on the residual limb has merit, although more comprehensive studies need to be conducted looking at their role in augmenting skin mechanics.

6. Conclusion

Skin is a dynamic regenerative organ that interacts mechanically with the external environment. Its main function is as a barrier to mechanical damage; however, it also acts to mechanically interact and sense the environment. Skin tribology is inherently intertwined with biological processes. Numerous factors influence the interface properties of skin, namely, hydration influences morphology and composition which in turn influence mechanics leading to inflammation and skin injury. Highly orchestrated

cellular processes allow conservation of the epidermal barrier through spatial differentiation. Skin morphology and composition vary based on anatomical location, with the most pronounced contrast occurring between loading skin (palmoplantar) and non-loading skin (non-palmoplantar). Recent characterization of skin covering an amputee's limb shows substantial adaptation occurs with wearing of the prosthesis that does not constitute greater resistance to load [76] and instead results in biological fatigue.

Characterizing skin of different pathologies and different mechanical environments is essential in soft tissue problems. It is important to improve the breadth of skin characterization to encompass groups that have been researched less, such as women in menopause. Additionally, moving away from studies focused solely on a single sex and/or genetic ancestry (usually males of European ancestry) is key to obtaining a more holistic and equitable view and understanding of skin mechanics. This research must be conducted with an integrated approach (figure 4) due to the interdependence on skin biology and skin mechanics. Throughout this piece, we have argued that a biology-first approach to skin tribology is required to complement the engineering-first approach currently used. We highlight the viewpoint of skin as an engineering material, which can open exciting possibilities and solutions to mechanical problems in healthcare.

Ethics. This work did not require ethical approval from a human subject or animal welfare committee.

Data accessibility. This article has no additional data.

Declaration of AI use. We have not used AI-assisted technologies in creating this article.

Authors' contributions. J.H.: conceptualization, funding acquisition, investigation, project administration, resources, visualization, writing—original draft, writing—review and editing; M.M.L.D.S.: visualization, writing—original draft, writing—review and editing; C.A.H.: supervision, writing—review and editing; M.M.: conceptualization, project administration, resources, supervision, visualization, writing—original draft, writing—review and editing.

All authors gave final approval for publication and agreed to be held accountable for the work performed therein.

Conflict of interest declaration. We declare we have no competing interests.

Funding. Funding provided by British Skin Foundation (grant reference 012_R_24) funding to C.A.H.

References

- Zhou X, Li Y, Tian Y, Masen MA, Li Y, Jin Z. 2023 Friction and neuroimaging of active and passive tactile touch. *Sci. Rep.* **13**, 13077. (doi:10.1038/s41598-023-40326-y)
- Wilkinson HN, Hardman MJ. 2020 Wound healing: cellular mechanisms and pathological outcomes. *Open Biol.* **10**, 200223. (doi:10.1098/rsob.200223)
- Dowson D. 2009 A tribological day. *Proc. Inst. Mech. Eng. J. Eng. Tribol.* **223**, 261–273. (doi:10.1243/13506501JET557)
- Van Kuilenburg J, Masen MA, van der Heide E. 2015 A review of fingerpad contact mechanics and friction and how this affects tactile perception. *Proc. Inst. Mech. Eng. J. Eng. Tribol.* **229**, 243–258. (doi:10.1177/1350650113504908)
- Jin ZM, Zheng J, Li W, Zhou ZR. 2016 Tribology of medical devices. *Biosurf. Biotribol.* **2**, 173–192. (doi:10.1016/j.bsbt.2016.12.001)
- Masen MA. 2011 A systems based experimental approach to tactile friction. *J. Mech. Behav. Biomed. Mater.* **4**, 1620–1626. (doi:10.1016/j.jmbbm.2011.04.007)
- Löken LS, Wessberg J, Morrison I, McGlone F, Olsson H. 2009 Coding of pleasant touch by unmyelinated afferents in humans. *Nat. Neurosci.* **12**, 547–548. (doi:10.1038/nn.2312)
- de Wert LA, Bader DL, Oomens CWJ, Schoonhoven L, Poeze M, Bouvy ND. 2015 A new method to evaluate the effects of shear on the skin. *Wound Repair Regen.* **23**, 885–890. (doi:10.1111/wrr.12368)
- de Wert LA, Geerts M, van der Brug S, Adriaansens L, Poeze M, Schaper N, Bouvy ND. 2019 The effect of shear force on skin viability in patients with type 2 diabetes. *J. Diabetes Res.* **2019**, 1973704. (doi:10.1155/2019/1973704)
- Klaassen M. 2018 The static friction behavior of skin with relevance to pressure ulcer prevalence. PhD Thesis, University of Twente, Enschede, The Netherlands. (doi:10.3990/1.9789036546072)
- Jayabal H, Abiakam NS, Filingeri D, Bader DL, Worsley PR. 2023 Inflammatory biomarkers in sebum for identifying skin damage in patients with a Stage I pressure ulcer in the pelvic region: a single centre observational, longitudinal cohort study with elderly patients. *Int. Wound J.* **20**, 2594–2607. (doi:10.1111/iwj.14131)
- Boyle CJ, Carpanen D, Pandelani T, Higgins CA, Masen MA, Masouros SD. 2019 Lateral pressure equalisation as a principle for designing support surfaces to prevent deep tissue pressure ulcers. *PLoS One* **15**, e0227064. (doi:10.1371/journal.pone.0227064)
- Veijgen NK, Masen MA, van der Heide E. 2013 Relating friction on the human skin to the hydration and temperature of the skin. *Tribol. Lett.* **49**, 251–262. (doi:10.1007/s11249-012-0062-1)
- Abbas AA, Dlova N, Higgins CA. 2025 Beyond the skin surface: melanocyte biology and the spectrum of health inequities. *J. Invest. Dermatol.* **145**, 2973–2980. (doi:10.1016/j.jid.2025.06.1586)
- Rawlings AV. 2006 Ethnic skin types: are there differences in skin structure and function? *Int. J. Cosmet. Sci.* **28**, 79–93. (doi:10.1111/j.1467-2494.2006.00302.x)
- Lim H, Kang S, Chien AL. 2019 Photodamage in skin of color. In *Cutaneous photoaging* (eds REB Watson, CEM Griffiths), pp. 31–58. London, UK: The Royal Society of Chemistry.
- Dao H Jr, Kazin RA. 2007 Gender differences in skin: a review of the literature. *Gen. Med.* **4**, 308–328. (doi:10.1016/S1550-8579(07)80061-1)
- Lee DH, Oh JH, Chung JH. 2016 Glycosaminoglycan and proteoglycan in skin aging. *J. Dermatol. Sci.* **83**, 174–181. (doi:10.1016/j.jdermsci.2016.05.016)
- Klaassen M, Schipper DJ, Masen MA. 2016 Influence of the relative humidity and the temperature on the in-vivo friction behaviour of human skin. *Bio. Tribol.* **6**, 21–28. (doi:10.1016/j.biotri.2016.03.003)
- Yeung CYC, Holmes DF, Thomason HA, Stephenson C, Derby B, Hardman MJ. 2016 An ex vivo porcine skin model to evaluate pressure-reducing devices of different mechanical properties used for pressure ulcer prevention. *Wound Repair Regen.* **24**, 1089–1096. (doi:10.1111/wrr.12481)
- Devin KM, Tang J, Moser D, Jiang L. 2023 Assessing socket fit effects on pressure and shear at a transtibial residuum/socket interface. *Appl. Bionics Biomech.* **2023**, 3257059. (doi:10.1155/2023/3257059)
- Zhang M, Turner-Smith AR, Tanner A, Roberts VC. 1998 Clinical investigation of the pressure and shear stress on the trans-tibial stump with a prosthesis. *Med. Eng. Phys.* **20**, 188–198. (doi:10.1016/S1350-4533(98)00013-7)
- Laszczak P, McGrath M, Tang J, Gao J, Jiang L, Bader DL, Moser D, Zahedi S. 2016 A pressure and shear sensor system for stress measurement at lower limb residuum/socket interface. *Med. Eng. Phys.* **38**, 695–700. (doi:10.1016/j.medengphy.2016.04.007)

24. Sanders JE, Zachariah SG, Jacobsen AK, Ferguson JR. 2005 Changes in interface pressures and shear stresses over time on trans-tibial amputee subjects ambulating with prosthetic limbs: comparison of diurnal and six-month differences. *J. Biomech.* **38**, 1566–1573. (doi:10.1016/j.jbiomech.2004.08.008)
25. Lee VSP, Solomonidis SE, Spence WD. 1997 Stump-socket interface pressure as an aid to socket design in prostheses for trans-femoral amputees—a preliminary study. *Proc. Inst. Mech. Eng. H J. Eng. Med.* **211**, 167–180. (doi:10.1243/0954411971534287)
26. Oomens CWJ, Bressers OFJT, Bosboom EMH, Bouten CVC, Blader DL. 2003 Can loaded interface characteristics influence strain distributions in muscle adjacent to bony prominences? *Comput. Method. Biomech. Biomed. Eng.* **6**, 171–180. (doi:10.1080/1025584031000121034)
27. Lyon CC, Kulkarni J, Zimerson E, Van Ross E, Beck MH. 2000 Skin disorders in amputees. *J. Am. Acad. Dermatol.* **42**, 501–507. (doi:10.1016/s0190-9622(00)90227-5)
28. DesGroseilliers JP, DesJardins JP, Germain JP, Krol AL. 1978 Dermatologic problems in amputees. *Can. Med. Assoc. J.* **118**, 535–537.
29. Meulenbelt HE, Geertzen JH, Jonkman MF, Dijkstra PU. 2009 Determinants of skin problems of the stump in lower-limb amputees. *Arch. Phys. Med. Rehabil.* **90**, 74–81. (doi:10.1016/j.apmr.2008.07.015)
30. Dudek NL, Marks MB, Marshall SC, Chardon JP. 2005 Dermatologic conditions associated with use of a lower-extremity prosthesis. *Arch. Phys. Med. Rehabil.* **86**, 659–663. (doi:10.1016/j.apmr.2004.09.003)
31. Koc E, Tunca M, Akar A, Erbil AH, Demiralp B, Arca E. 2008 Skin problems in amputees: a descriptive study. *Int. J. Dermatol.* **47**, 463–466. (doi:10.1111/j.1365-4632.2008.03604.x)
32. Highsmith MJ, Kahle JT, Klenow TD, Andrews CR, Lewis KL, Bradley RC, Ward JM, Orriola JJ, Highsmith JT. 2016 Interventions to manage residual limb ulceration due to prosthetic use in individuals with lower extremity amputation: a systematic review of the literature. *Technol. Innov.* **18**, 115–123. (doi:10.21300/18.2-3.2016.115)
33. Worsley PR, Stanger ND, Horrell AK, Bader DL. 2018 Investigating the effects of cervical collar design and fit on the biomechanical and biomarker reaction at the skin. *Med. Device Evid. Res.* **11**, 87–94. (doi:10.2147/MDER.S149419)
34. Ham W, Schoonhoven L, Schuurmans MJ, Leenen LPH. 2014 Pressure ulcers from spinal immobilization in trauma patients: a systematic review. *J. Trauma Acute Care Surg.* **76**, 1131–1141. (doi:10.1097/TA.0000000000000153)
35. Black JM, Cuddigan JE, Walko MA, Didier LA, Lander MJ, Kelp MR. 2010 Medical device related pressure ulcers in hospitalized patients. *Int. Wound J.* **7**, 358–365. (doi:10.1111/j.1742-481X.2010.00699.x)
36. Bader DL, Worsley PR, Gefen A. 2019 Bioengineering considerations in the prevention of medical device-related pressure ulcers. *Clin. Biomech.* **67**, 70–77. (doi:10.1016/j.clinbiomech.2019.04.018)
37. Li W, Liu XD, Cai ZB, Zheng J, Zhou ZR. 2011 Effect of prosthetic socks on the frictional properties of residual limb skin. *Wear* **271**, 2804–2811. (doi:10.1016/j.wear.2011.05.032)
38. Klaassen M, de Vries EG, Masen MA. 2019 Friction in the contact between skin and a soft counter material: Effects of hardness and surface finish. *J. Mech. Behav. Biomed. Mater.* **92**, 137–143. (doi:10.1016/j.jmbbm.2019.01.006)
39. McInnes E, Jammali-Blasi A, Bell-Syer SEM, Dumville JC, Middleton V, Cullum N. 2015 Support surfaces for pressure ulcer prevention. *Cochrane Database Syst. Rev.* **2015**, CD001735. (doi:10.1002/14651858.CD001735.pub2)
40. Tanriverdi U *et al.* 2025 Dynamically adaptive soft metamaterial for wearable human-machine interfaces. *Nat. Commun.* **16**, 2621. (doi:10.1038/s41467-025-57634-8)
41. Reuvekamp H, Hekman EEG, van der Heide E, Matthews DTA. 2024 Strategies in surface engineering for the regulation of microclimates in skin-medical product interactions. *Heliyon* **10**, e25395. (doi:10.1016/j.heliyon.2024.e25395)
42. Gnyawali SC *et al.* 2023 Moisture mitigation using a vented liner and a vented socket system for individuals with transfemoral amputation. *Sci. Rep.* **13**, 16557. (doi:10.1038/s41598-023-43572-2)
43. Bowden FP, Tabor D. 2001 *The friction and lubrication of solids*. vol. 1. Oxford, UK: Oxford University Press.
44. Adams MJ, Briscoe BJ, Johnson SA. 2007 Friction and lubrication of human skin. *Tribol. Lett.* **26**, 239–253. (doi:10.1007/s11249-007-9206-0)
45. Sheu HM, Chao SC, Wong TW, Yu-Yun Lee J, Tsai JC. 1999 Human skin surface lipid film: an ultrastructural study and interaction with corneocytes and intercellular lipid lamellae of the stratum corneum. *Br. J. Dermatol.* **140**, 385–391. (doi:10.1046/j.1365-2133.1999.02697.x)
46. Makrantonaki E, Ganceviciene R, Zouboulis CC. 2011 An update on the role of the sebaceous gland in the pathogenesis of acne. *Dermatoendocrinol.* **3**, 41–49. (doi:10.4161/derm.3.1.13900)
47. Plotczyk M, Higgins CA. 2019 Skin biology. In *Biomaterials for skin repair and regeneration* (ed. E García-Gareta), pp. 3–25. Cambridge, UK: Woodhead Publishing. (doi:10.1016/B978-0-08-102546-8.00001-7)
48. Korbeld KT, Klaassen M, Jobanputra RD, de Vries EG, Masen MA. 2020 Effects of sebum properties on skin friction: investigation using a bench test. *Biosurf. Biotribol.* **6**, 43–47. (doi:10.1049/bsbt.2019.0015)
49. Pailler-Mattei C, Nicoli S, Pirot F, Vargiolu R, Zahouani H. 2009 A new approach to describe the skin surface physical properties *in vivo*. *Colloids Surf. B. Biointerfaces* **68**, 200–206. (doi:10.1016/j.colsurfb.2008.10.005)
50. Cua AB, Wilhelm KP, Maibach HI. 1995 Skin surface lipid and skin friction: relation to age, sex and anatomical region. *Skin Pharmacol. Physiol.* **8**, 246–251. (doi:10.1159/000211354)
51. Cua AB, Wilhelm KP, Maibach HI. 1990 Frictional properties of human skin: relation to age, sex and anatomical region, stratum corneum hydration and transepidermal water loss. *Br. J. Dermatol.* **123**, 473–479. (doi:10.1111/j.1365-2133.1990.tb01452.x)
52. Yap KK, Murali M, Tan Z, Zhou X, Li L, Masen MA. 2021 Wax-oil lubricants to reduce the shear between skin and PPE. *Sci. Rep.* **11**, 11537. (doi:10.1038/s41598-021-91119-0)
53. Masen MA *et al.* 2020 Evaluating lubricant performance to reduce COVID-19 PPE-related skin injury. *PLoS One* **15**, e0239363. (doi:10.1371/journal.pone.0239363)
54. Jobanputra RD, Hayes J, Royyuru S, Masen MA. 2021 A numerical analysis of skin-PPE interaction to prevent facial tissue injury. *Sci. Rep.* **11**, 16248. (doi:10.1038/s41598-021-95861-3)
55. Limbert G, Masen MA, Pond D, Graham HK, Sherratt MJ, Jobanputra R, McBride A. 2019 Biotribology of the ageing skin—why we should care. *Biotribology* **17**, 75–90. (doi:10.1016/j.biotri.2019.03.001)
56. Gefen A. 2011 How do microclimate factors affect the risk for superficial pressure ulcers: a mathematical modeling study. *J. Tissue Viability* **20**, 81–88. (doi:10.1016/j.jtv.2010.10.002)
57. Pigmans RRWP *et al.* 2024 Development of personalized non-invasive ventilation masks for critically ill children: a bench study. *Intensive Care Med. Exp.* **12**, 21. (doi:10.1186/s40635-024-00607-w)
58. Muller GJ, Hovenier R, Spijker J, van Gestel M, Klein-Blommert R, Markhorst D, Dijkman C, Bem RA. 2021 Non-invasive ventilation for pediatric hypoxic acute respiratory failure using a simple anesthetic mask with 3D printed adaptor: a case report. *Front. Pediatr.* **9**, 710829. (doi:10.3389/fped.2021.710829)
59. Visscher MO, White CC, Jones JM, Cahill T, Jones DC, Pan BS. 2015 Face masks for noninvasive ventilation: fit, excess skin hydration, and pressure ulcers. *Respir. Care* **60**, 1536–1547. (doi:10.4187/respcare.04036)

60. Bliss DZ *et al.* 2017 Incidence and predictors of incontinence-associated skin damage in nursing home residents with new-onset incontinence. *J. Wound Ostomy Continence Nurs.* **44**, 165–171. (doi:10.1097/WON.0000000000000313)
61. Falloon SS, Abbas S, Stridfeldt C, Cottenden A. 2018 The impact of microclimate on skin health with absorbent incontinence product use. *J. Wound Ostomy Continence Nurs.* **45**, 341–348. (doi:10.1097/WON.0000000000000449)
62. Hall KD, Clark RC. 2015 A prospective, descriptive, quality improvement study to decrease incontinence-associated dermatitis and hospital-acquired pressure ulcers. *Ostomy Wound Manage.* **61**, 26–30. <https://europepmc.org/article/med/26185973>
63. Wagg A, Agholme F, Schwiigel H, Huige N, Hayder-Beichel D. 2025 A post-market, cluster randomised controlled trial of the effect of the TENA SmartCare Change Indicator on continence care efficiency and skin health in nursing homes. *Age Ageing* **54**, afaf236. (doi:10.1093/ageing/afaf236)
64. Zhang X, Zhang Y, Jin Z. 2022 A review of the bio-tribology of medical devices. *Friction* **10**, 4–30. (doi:10.1007/s40544-021-0512-6)
65. Byrne C, Hardman M, Nield K. 2003 Covering the limb—formation of the integument. *J. Anat.* **202**, 113–123. (doi:10.1046/j.1469-7580.2003.00142.x)
66. Fuchs E. 1995 Keratins and the skin. *Annu. Rev. Cell Dev. Biol.* **11**, 123–154. (doi:10.1146/annurev.cb.11.110195.001011)
67. Oltulu P, Ince B, Kokbudak N, Findik S, Kilinc F. 2018 Measurement of epidermis, dermis, and total skin thicknesses from six different body regions with a new ethical histometric technique. *Turk. J. Plast. Surg.* **26**, 56–61. (doi:10.4103/tjps.TJPS_2_17)
68. Driskell RR *et al.* 2013 Distinct fibroblast lineages determine dermal architecture in skin development and repair. *Nature* **504**, 277–281. (doi:10.1038/nature12783)
69. Rinn JL, Bondre C, Gladstone HB, Brown PO, Chang HY. 2006 Anatomic demarcation by positional variation in fibroblast gene expression programs. *PLoS Genet.* **2**, e119. (doi:10.1371/journal.pgen.0020119)
70. Rinn JL *et al.* 2008 A dermal *HOX* transcriptional program regulates site-specific epidermal fate. *Genes Dev.* **22**, 303–307. (doi:10.1101/gad.1610508)
71. Geerligs M. 2010 Skin layer mechanics. PhD Thesis, Technische Universiteit Eindhoven, Eindhoven, The Netherlands.
72. Philipeos C *et al.* 2018 Spatial and single-cell transcriptional profiling identifies functionally distinct human dermal fibroblast subpopulations. *J. Invest. Dermatol.* **138**, 811–825. (doi:10.1016/j.jid.2018.01.016)
73. Steele L *et al.* 2025 A single-cell and spatial genomics atlas of human skin fibroblasts reveals shared disease-related fibroblast subtypes across tissues. *Nat. Immunol.* **26**, 1807–1820. (doi:10.1038/s41590-025-02267-8)
74. Vyumvuhore R, Tfayli A, Duplan H, Delalleau A, Manfait M, Baillet-Guffroy A. 2013 Raman spectroscopy: a tool for biomechanical characterization of stratum corneum. *J. Raman Spectrosc.* **44**, 1077–1083. (doi:10.1002/jrs.4334)
75. Wong LS *et al.* 2018 Decrease of superficial serine and lactate in the stratum corneum due to repetitive frictional trauma. *Int. J. Dermatol.* **57**, 299–305. (doi:10.1111/ijd.13856)
76. Hayes J, Andrews J, Abdelwahab O, Andriuskevicius T, Briggs T, Gordon R, Worsley P, Higgins CA, Masen M. 2025 Skin adaptation in lower limb amputees assessed through Raman spectroscopy and mechanical characterization. *J. R. Soc. Interface* **22**, 20240475. (doi:10.1098/rsif.2024.0475)
77. Wei JCY, Edwards GA, Martin DJ, Huang H, Crichton ML, Kendall MAF. 2017 Allometric scaling of skin thickness, elasticity, viscoelasticity to mass for micro-medical device translation: from mice, rats, rabbits, pigs to humans. *Sci. Rep.* **7**, 15885. (doi:10.1038/s41598-017-15830-7)
78. Boyle CJ, Plotczyk M, Villalta SF, Patel S, Hettiaratchy S, Masouros SD, Masen MA, Higgins CA. 2019 Morphology and composition play distinct and complementary roles in the tolerance of plantar skin to mechanical load. *Sci. Adv.* **5**, eaay0244. (doi:10.1126/sciadv.aay0244)
79. Derler S, Gerhardt LC. 2012 Tribology of skin: review and analysis of experimental results for the friction coefficient of human skin. *Tribol. Lett.* **45**, 1–27. (doi:10.1007/s11249-011-9854-y)
80. Joodaki H, Panzer MB. 2018 Skin mechanical properties and modeling: a review. *Proc. Inst. Mech. Eng. H J. Eng. Med.* **232**, 323–343. (doi:10.1177/0954411918759801)
81. Mostafavi Yazdi SJ, Baqersad J. 2022 Mechanical modeling and characterization of human skin: a review. *J. Biomech.* **130**, 110864. (doi:10.1016/j.jbiomech.2021.110864)
82. van Kuilenburg J, Masen MA, van der Heide E. 2013 Contact modelling of human skin: what value to use for the modulus of elasticity? *Proc. Inst. Mech. Eng. J J. Eng. Tribol* **227**, 349–361. (doi:10.1177/1350650112463307)
83. Évora AS, Adams MJ, Johnson SA, Zhang Z. 2021 Corneocytes: relationship between structural and biomechanical properties. *Skin Pharmacol. Physiol.* **34**, 146–161. (doi:10.1159/000513054)
84. Puzzi L, Borin D, Martinelli V, Mestroni L, Kelsell DP, Sbaizero O. 2018 Cellular biomechanics impairment in keratinocytes is associated with a C-terminal truncated desmoplakin: an atomic force microscopy investigation. *Micron* **106**, 27–33. (doi:10.1016/j.micron.2017.12.005)
85. Ramms L *et al.* 2013 Keratins as the main component for the mechanical integrity of keratinocytes. *Proc. Natl Acad. Sci. USA* **110**, 18513–18518. (doi:10.1073/pnas.1313491110)
86. Efremov YM, Lomakina ME, Bagrov DV, Makhnovskiy PI, Alexandrova AV, Kirpichnikov MP, Shaitan KV. 2014 Mechanical properties of fibroblasts depend on level of cancer transformation. *Biochim. Biophys. Acta Mol. Cell. Res.* **1843**, 1013–1019. (doi:10.1016/j.bbamcr.2014.01.032)
87. Silver FH, Seehra GP, Freeman JW, DeVore D. 2002 Viscoelastic properties of young and old human dermis: a proposed molecular mechanism for elastic energy storage in collagen and elastin. *J. Appl. Polym. Sci.* **86**, 1978–1985. (doi:10.1002/app.11119)
88. Silver FH, Freeman JW, DeVore D. 2001 Viscoelastic properties of human skin and processed dermis. *Skin Res. Technol.* **7**, 18–23. (doi:10.1034/j.1600-0846.2001.007001018.x)
89. Boyle CJ, Higgins CA. 2021 Can plantar fibroblast implantation protect amputees from skin injury? A recipe for skin augmentation. *Exp. Dermatol.* **30**, 1829–1833. (doi:10.1111/exd.14419)
90. Margi R, Gefen A. 2022 Evaluation of facial tissue stresses under medical devices post application of a cyanoacrylate liquid skin protectant: an integrated experimental-computational study. *Int. Wound J.* **19**, 615–632. (doi:10.1111/iwj.13660)
91. Fuchs C, Wang Y, Wise E, Farinelli WA, Anderson RR, Cho S, Meyerle JH, Tam J. 2024 Structural and molecular characteristics of weight-bearing volar skin can be reconstituted by micro skin tissue column grafting. *FASEB J.* **38**, e23873. (doi:10.1096/fj.202400866R)
92. Higgins CA, Chen JC, Cerise JE, Jahoda CAB, Christiano AM. 2013 Microenvironmental reprogramming by three-dimensional culture enables dermal papilla cells to induce de novo human hair-follicle growth. *Proc. Natl Acad. Sci. USA* **110**, 19679–19688. (doi:10.1073/pnas.1309970110)
93. Yamaguchi Y, Itami S, Tarutani M, Hosokawa K, Miura H, Yoshikawa K. 1999 Regulation of keratin 9 in nonpalmoplantar keratinocytes by palmoplantar fibroblasts through epithelial-mesenchymal interactions. *J. Invest. Dermatol.* **112**, 483–488. (doi:10.1046/j.1523-1747.1999.00544.x)
94. Lee SS *et al.* 2024 The use of ectopic volar fibroblasts to modify skin identity. *Science* **385**, eadi1650. (doi:10.1126/science.adi1650)