

Healthy peristomal skin: a foundation for successful voice and pulmonary rehabilitation

Understanding peristomal skin

The structure of the skin

The skin is the largest organ in the body and renews itself roughly every 28 days. The thickest skin is found on the soles of the feet, where it measures around 1.5mm. Skin has several key functions:

- Protection against trauma, UV light, temperature changes, dehydration, toxins, and microorganisms
- Temperature regulation through circulation, sweating, and insulation
- Sensation (pain, pressure, vibration, and temperature)
- Metabolism, including melanin production, vitamin D synthesis, and excretion of small amounts of waste through sweat
- Communication, such as facial expression and emotional signalling and touch.

The skin provides a semi permeable barrier that protects the body from the external environment and helps maintain homeostasis.

Clinical insight

A clear understanding of how the skin is structured and how it functions provides the foundation for recognising early signs of peristomal irritation, explaining why specific injury patterns occur, and choosing appropriate strategies to protect the epidermis during adhesive wear.

The skin consists of 3 layers:

- The epidermis – traps moisture
- The dermis – provides functionality due to variety of structures in this layer
- The hypodermis or subcutaneous tissue provides insulation and cushion

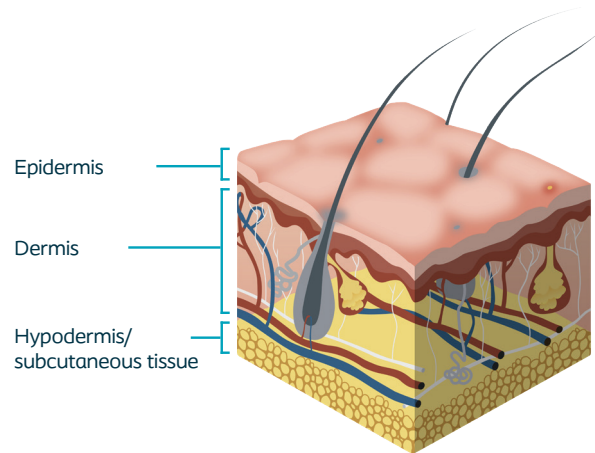


FIGURE. The three layers of the skin: Epidermis, dermis and hypodermis/ Subcutaneous tissue.

Definitions

Intact injured skin (superficially injured skin): presents with superficial pathological changes or changes following disease or trauma but where the basement membrane is unbroken.

Non-intact injured skin (breached skin): presents with pathological changes or changes following disease or trauma where the basement membrane is breached.

Understanding peristomal skin

Structure and stress points

Peristomal skin is not uniform. Different layers of the epidermis contribute distinctive protective functions and demonstrate different patterns of failure when exposed to mechanical forces, moisture or adhesive trauma. Damage to these outer layers can compromise barrier integrity and increase permeability of the skin^{1,2}. The following overview outlines how each layer behaves under stress and why certain injury types appear the way they do.

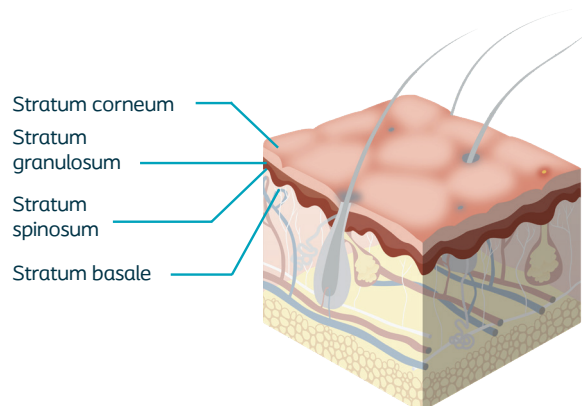


FIGURE. The epidermis and its four layers.

The stratum corneum: The sacrificial layer

The stratum corneum is the outermost layer of the skin and forms the barrier between the body and the external environment. It is made of tightly packed, flattened, non-living cells called corneocytes held together by lipids made up of ceramides, cholesterol and fatty acids. This "brick and mortar" arrangement helps to:

- Prevent moisture loss also known as transepidermal water loss (TEWL)
- Protect the living skin beneath from friction and irritants
- Absorb the impact of external forces such as wiping, shaving, or adhesive removal^{3,4}.

Because the stratum corneum is designed to be expendable, it sheds over time. When exposed repeatedly to mechanical stress or excess moisture, it can be removed faster than it can be replaced.

How it responds under stress

Skin responds to stress in different ways. Repeated traction, such as frequent adhesive removal, progressively thins the surface, which may appear pink or shiny once the protective outer layer is stripped. Dry friction or overheating, for example from abrasive cleaning, leaves the skin dull, rough or flaky and prone to cracking. In contrast, excessive moisture from sweat or mucus build-up causes the surface to swell and soften, appearing pale and wrinkled before breaking down.

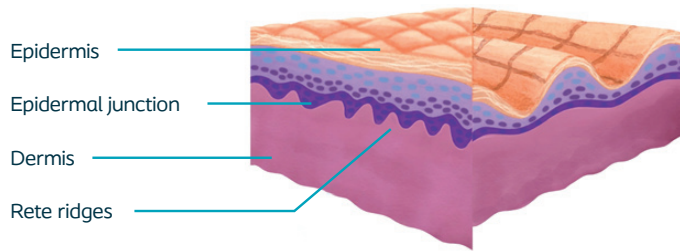
When this layer remains intact, it protects the underlying epidermis from chemical, microbial and mechanical insults. When it is lost more quickly than it can recover, the surface becomes more sensitive to touch, more prone to moisture loss, and less tolerant of direct contact, including adhesives and allows irritants to penetrate more easily⁵.

Clinical insight

Damage to the stratum corneum is often the first step in peristomal skin problems. Even mild erosion from repeated adhesive removal or moisture exposure can reduce tolerance to adhesives and increase the risk of longer-term irritation. Recognising early changes in this layer allows timely adjustments in cleaning routines, adhesive choice, or wear patterns to prevent deeper injury and maintain comfort.

Derma-Epidermal Junction (DEJ)

The Anchor/Shear Point



The dermo-epidermal junction (DEJ) is not a visible layer but a specialised basement membrane interface where the epidermis is anchored to the underlying dermis. This structure forms a complex attachment system that maintains skin integrity and mechanical stability^{6,7}.

Its key roles are to:

- Prevent the epidermis from sliding over the dermis⁶
- Distribute shear forces during movement or traction^{8,9}
- Provide a pathway for nutrients from the vascular dermis to the non-vascular epidermis¹⁰

Rete ridges (or rete pegs) play a key role in skin strength. They are downward projections of the epidermis that interlock with upward projections of the dermis (dermal papillae) at the DEJ. Rather than the epidermis sitting flat on the dermis, the two layers form a ridged interlocking junction where the epidermis anchors into the dermis¹¹.

How it responds to stress

When the skin is exposed to strong or rapid traction, such as aggressive adhesive removal, this anchoring layer can partially separate, causing the outer skin to lift away in sheets rather than flake at the surface. Prolonged moisture from sweat or mucus softens the skin and weakens the bonds between these layers meaning that less force is needed for them to pull apart. Inflammatory heat, such as during a dermatitis flare or following radiotherapy, increases blood flow and tissue reactivity, which can make the junction more fragile and more vulnerable to mechanical stress.

When intact, the DEJ allows the surface to move and flex without detaching. When the DEJ is weakened, the layers can separate at this anchoring interface rather than at the surface. In these cases, the outer skin may lift as a single layer, and the exposed tissue often appears raw, pink, or damp rather than dry or flaky.

Clinical insight

Damage at the DEJ leads to deeper and more painful injury than loss of the stratum corneum alone. Adhesive removal, moisture softening, or inflammatory skin changes can all weaken this anchoring point, increasing the risk of sheet-like skin lifts. Recognising early signs of shear stress allows clinicians to modify adhesive technique or product choice before more significant detachment occurs.

Clinical insight

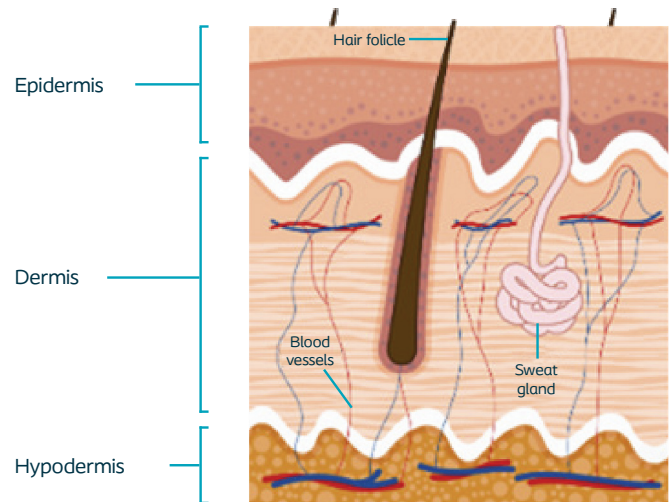
If separation occurs at the DEJ, the area can heal through re-epithelisation and gradual re-anchoring of the epidermis to the dermis. Protecting the site from further traction, managing moisture, and allowing a short break from adhesives support this process and help the layers reattach safely.

Epidermis

The Living Barrier Layer

The epidermis is the outer epithelial layer of the skin and consists of several sublayers, including the stratum basale, stratum spinosum, stratum granulosum, and the stratum corneum which forms the most superficial barrier exposed to the external environment¹² and is responsible for continuous cellular renewal and repair¹³. Within the deeper epidermal layers keratinocytes continuously proliferate and migrate toward the surface, replacing cells that are naturally shed from the stratum corneum. This process enables ongoing cellular renewal and barrier repair¹⁴.

When intact, this regenerative process helps replace superficial cells lost during routine cleansing or adhesive removal and the epidermis recovers quickly from this mild surface disruption. However, mechanical injury to the skin barrier damages keratinocytes and reduces barrier function¹⁵. Once the epidermis becomes exposed, inflamed or stripped, its tolerance to mechanical stress is significantly reduced.



Clinical Characteristics

Status of the Epidermis	Appearance	Clinical Implication
Protected beneath the stratum corneum.	Normal matte texture, no shine.	Routine adhesive use is generally tolerated.
Exposed (after stripping/erosion of stratum corneum).	Pink, smooth or shiny surface.	Indicates early injury, baseplate adhesion may still be possible but should be modified (gentle removal, less frequent changes, barrier support).
Inflamed or sensitised	Red, sore, reactive to touch or cleansers.	Indicates that contact tolerance is declining, continued stripping will accelerate breakdown.
Chronically overstressed	Thickened (hyperkeratotic) or persistently fragile.	Suggests repair attempts are ongoing but inefficient. Indicates need for protection or rotation of attachment methods.

Clinical insight

Superficial epidermal injury does not represent full-thickness tissue damage, but it is a critical decision point. When protected early, the epidermis can rapidly restore barrier function. However, if the area remains inflamed and continues to be exposed to mechanical stress, the risk of progression to deeper dermal injury increases.

Dermis

The Structural Support and Vascular Supply

The dermis lies beneath the epidermis and provides the mechanical strength and blood supply needed for skin stability and repair. It contains collagen, elastic tissue, and other extracellular components that include vasculature, nerve endings, hair follicles, and glands¹⁶.

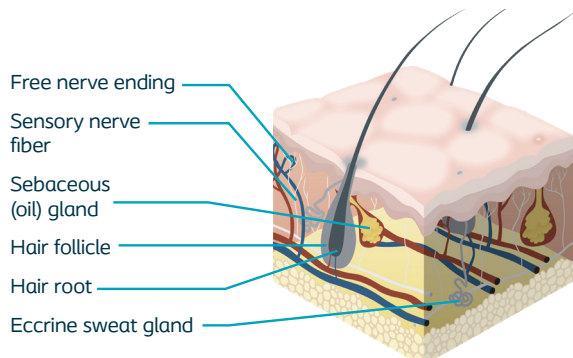


FIGURE. The dermis with blood vessels, nerve endings, hair follicles, sebaceous glands and sweat glands.

Functional Characteristics and Clinical Relevance^{17,18,19}

Collagen / Elastin Network

Changes to dermal collagen and elastin (e.g. ageing, scarring or radiotherapy-induced fibrosis) reduce elasticity and increase skin fragility, making the skin more susceptible to shear forces and uneven lifting during adhesive removal.

Blood Supply

Adequate dermal perfusion supports repair following superficial injury, whereas impaired circulation delays healing and prolongs inflammation.

Immune Cells (e.g. mast cells, macrophages)

These immune cells mediate inflammatory skin reactions such as irritant or allergic contact dermatitis and wound healing inflammation.

Sensory Nerve Endings

The dermis contains sensory nerve endings. Increased pain, burning, or stinging during cleansing or adhesive removal may indicate deeper tissue irritation involving the dermis rather than superficial epidermal disruption.

Behaviour Under Stress

When the dermis is intact and well perfused, it will present as soft and pliable, this generally allows for routine adhesive removal without incident. When the skin is fibrotic, the skin can feel firm and inflexible, this increases the chances of traction related lifting. When the skin is inflamed or moist it will present as red, swollen or weeping, this will provide poor surface stability, and adhesion may be reduced or inconsistent.

Clinical insight

Changes within the dermis affect how well the skin tolerates traction, friction, and adhesive use. Reduced elasticity, poor perfusion, or active inflammation all lower the skin's resilience and increase the risk of deeper injury. Identifying these dermal characteristics early helps guide safer adhesive techniques and protective strategies.

Hypodermis

Subsurface Cushioning and Contour Layer

The hypodermis (also referred to as subcutaneous tissue) primarily functions as a mechanical cushioning and insulating layer. It is composed largely of adipose tissue with a loose connective tissue framework, which helps absorb mechanical stress, conserve heat, and anchor the skin to underlying fascia and structures.

The distribution and thickness of subcutaneous fat also contribute to surface contour, influencing how the overlying skin sits against underlying anatomical structures²⁰.

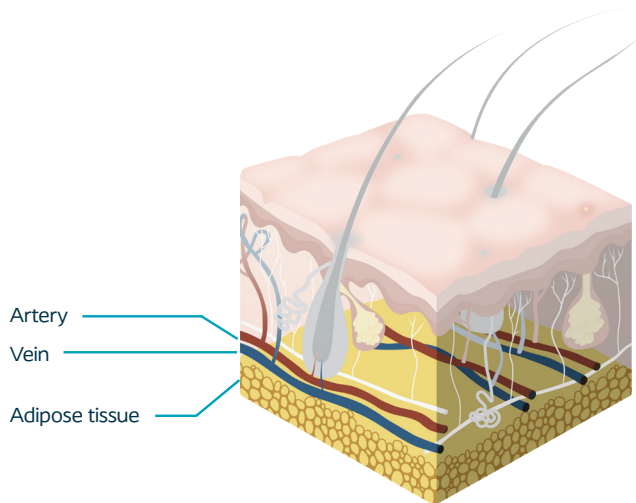


FIGURE. Hypodermis/Subcutaneous tissue.

Clinical characteristics

- When the hypodermis is thinner (after radiation or scarring): there is less natural cushioning. Force travels more directly to the surface. The tissue becomes tighter and less flexible²¹.
- When the hypodermis is uneven or reconstructed (e.g. Flap reconstruction): some areas are softer or thicker than others. The tissue does not move evenly. Stress builds up where the different tissues meet²².
- Where fibrosis or tethering is present, increased stiffness and reduced tissue mobility prevent normal force distribution, causing mechanical forces to become concentrated in specific areas rather than being evenly dispersed²³.

Behaviour under stress

When the skin is soft and even it provides a stable surface and contour which allows for a consistent adhesive contact area. If the hypodermis is irregular (e.g. flap edges, scar bands) this creates dips and ridges which lead to tension points where moisture can pool and adhesive edges can lift.

Clinical insight

Unlike the upper skin layers, the hypodermis is rarely damaged directly by adhesives. However, it strongly influences how the upper layers respond to pressure, movement, and moisture. Variations in subcutaneous thickness or mobility explain why the same adhesive may perform differently from one region to another in the same patient.

Peristomal skin is physiologically the same as the surrounding skin, but it is exposed to a unique combination of mechanical, chemical and environmental stressors, which increase its risk of barrier disruption and skin complications²⁴. Skin tolerance depends not only on surface barrier integrity, but also on underlying factors such as vascular supply, cellular activity, structural support, and tissue contour. Effective management must consider both the skin layer affected and the environment it is exposed to.

References

1. Jensen JM, Proksch E. The skin's barrier. *Giornale Italiano Di Dermatologia E Venereologia: Organo Ufficiale, Societa Italiana Di Dermatologia E Sifilografia* [Internet]. 2009 Dec 1;144(6):689–700. Available from: <https://pubmed.ncbi.nlm.nih.gov/19907407/>.
2. Kikuchi K, Shigeta S, Numayama-Tsuruta K, Ishikawa T. Vulnerability of the skin barrier to mechanical rubbing. *International Journal of Pharmaceutics* [Internet]. 2020 Sep 25;587:119708. Available from: <https://reader.elsevier.com/reader/sd/pii/S037851732030692X?token=AE6E171AB578B92704A2046DDC9A6BE93DD81FFEDFDF439AAABE9DC1BF46C9036917071148E5022C91B35CB1AA4A344C&originRegion=eu-west-1&originCreation=20220516164250>.
3. Smeden Jeroen van, et al., The important role of stratum corneum lipids for the cutaneous barrier function. *Biochimica et Biophysica Acta (BBA) - Molecular and Cell Biology of Lipids* [Internet]. 2014 Mar 1;1841(3):295–313. Available from: https://www.sciencedirect.com/science/article/pii/S1388198113002485?casa_token=4kQb_u4B3ykAAAAA:pj1ZFvbyQyTp-sH5Yk1eHxLh6-i5D4Y9orWiCnGrf0k6CDINbw9Xzj--faUPuileXvvyzhsKOA#bb0470.
4. Smeden, Jeroen van, and JA. Bouwstra. "Stratum Corneum Lipids: Their Role for the Skin Barrier Function in Healthy Subjects and Atopic Dermatitis Patients." *Skin Barrier Function*, vol. 49, 2016, pp. 8–26, <http://www.karger.com/Article/Abstract/441540#:~:text=The%20skin%20barrier%20is%20located,https://doi.org/10.1159/000441540>. Accessed 5 Mar. 2026.
5. Wertz, Philip W. "Synopsis of Barrier Function of Skin and Mucosa—Volume 2." *International Journal of Molecular Sciences*, vol. 24, no. 18, 5 Sept. 2023, p. 13690, <http://www.mdpi.com/1422-0067/24/18/13690>, <https://doi.org/10.3390/ijms241813690>. Accessed 5 Mar. 2026.
6. Has, Cristina, and Alexander Nyström. "Epidermal Basement Membrane in Health and Disease." *Current Topics in Membranes*, vol. 76, 2015, pp. 117–70, pubmed.ncbi.nlm.nih.gov/26610913/, <https://doi.org/10.1016/bs.ctm.2015.05.003>. Accessed 5 Mar. 2026.
7. Watt, F. M., and H. Fujiwara. "Cell-Extracellular Matrix Interactions in Normal and Diseased Skin." *Cold Spring Harbor Perspectives in Biology*, vol. 3, no. 4, 2 Feb. 2011, pp. a005124–a005124, <http://www.ncbi.nlm.nih.gov/pmc/articles/PMC3062212/>, <https://doi.org/10.1101/cshperspect.a005124>. Accessed 5 Mar. 2026.
8. Bush, Katie A., and George D. Pins. "Development of Microfabricated Dermal Epidermal Regenerative Matrices to Evaluate the Role of Cellular Microenvironments on Epidermal Morphogenesis." *Tissue Engineering Part A*, vol. 18, no. 21–22, Nov. 2012, pp. 2343–2353, <https://doi.org/10.1089/ten.tea.2011.0479>. Accessed 3 Nov. 2021.
9. Fenner J, Clark RAF. *Anatomy, Physiology, Histology, and Immunohistochemistry of Human Skin*. *Skin Tissue Engineering and Regenerative Medicine*. 2016;1–17.
10. Mescher, Anthony L. *Junqueira's Basic Histology: Text and Atlas*. 16th ed., New York, McGraw-Hill, 2021, pp. 372–379.
11. Zijl, Sebastiaan, et al. *Cell-Cell Signaling in Development; Chapter Four: Dynamic Regulation of Human Epidermal Differentiation by Adhesive and Mechanical Forces*. 150th ed., Academic Press, 9 July 2022, pp. 129–148.
12. Proksch E, Brandner JM, Jensen JM. The skin: an indispensable barrier. *Experimental dermatology*. 2008 Nov 11;17(12):1063–72.
13. Pastar, Irena, et al. "Epithelialization in Wound Healing: A Comprehensive Review." *Advances in Wound Care*, vol. 3, no. 7, July 2014, pp. 445–464, <http://www.ncbi.nlm.nih.gov/pmc/articles/PMC4086220/>, <https://doi.org/10.1089/wound.2013.0473>. Accessed 6 Mar. 2026.
14. Elias PM. Epidermal barrier function: intercellular lamellar lipid structures, origin, composition and metabolism. *Journal of Controlled Release*. 1991 Jun;15(3):199–208.
15. Breternitz M, Flach M, Präßler J, Elsner P, Fluhr JW. Acute barrier disruption by adhesive tapes is influenced by pressure, time and anatomical location: integrity and cohesion assessed by sequential tape stripping; a randomized, controlled study. *British Journal of Dermatology*. 2007 Feb;156(2):231–40.
16. 16.Tm B, K K. *Histology, Dermis* [Internet]. PubMed. 2020. Available from: <https://pubmed.ncbi.nlm.nih.gov/30570967/>.
17. Eming SA, Martin P, Tomic-Canic M. Wound repair and regeneration: Mechanisms, signaling, and translation. *Science Translational Medicine*. 2014 Dec 3;6(265):265sr6–6.
18. Krystel-Whittemore M, Dileepan KN, Wood JG. Mast Cell: a Multi-Functional Master Cell. *Frontiers in Immunology* [Internet]. 2016 Jan 6;6(1). Available from: <https://www.frontiersin.org/articles/10.3389/fimmu.2015.00620/full>.
19. Abraira Victoria E, Ginty David D. The Sensory Neurons of Touch. *Neuron* [Internet]. 2013 Aug;79(4):618–39. Available from: <https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3811145/>.
20. Proksch E, Brandner JM, Jensen JM. The skin: an indispensable barrier. *Experimental dermatology*. 2008 Nov 11;17(12):1063–72.
21. Borrelli MR, Shen AH, Lee GK, Momeni A, Longaker MT, Wan DC. Radiation-Induced Skin Fibrosis. *Annals of Plastic Surgery*. 2019 Oct;83(4S):S59–64.
22. Lv CL, Li B. Interface Morphodynamics in Living Tissues. *Soft Matter* [Internet]. 2025 [cited 2026 Mar 24];21(19):3670–87. Available from: <https://pubs.rsc.org/en/content/articlehtml/2025/sm/d5sm00145e>.
23. Kuehlmann B, Bonham CA, Zucal I, Prantl L, Gurtner GC. Mechanotransduction in Wound Healing and Fibrosis. *Journal of Clinical Medicine* [Internet]. 2020 May 11 [cited 2021 Mar 26];9(5):1423. Available from: <https://www.ncbi.nlm.nih.gov/pmc/articles/PMC7290354/pdf/jcm-09-01423.pdf>.
24. Gray M, Colwell JC, Doughty D, Goldberg M, Hoefloek J, Manson A, et al. Peristomal Moisture–Associated Skin Damage in Adults with Fecal Ostomies. *Journal of Wound, Ostomy and Continence Nursing*. 2013;40(4):389–99.

Atos (UK),
 Cartwright House, Tottle Road,
 Nottingham, NG2 1RT
 Tel: 0800 783 1659
 Email: info.uk@atosmedical.com
 Web: www.atosmedical.com

Atos and the Atos logo are trademarks of Coloplast A/S.
 © 2026-02 Coloplast A/S. All rights reserved.