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Friction Blisters of the Feet: A New Paradigm to Explain Causation

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Friction blisters on the feet commonly occur when individuals engage in active pursuits such as running, hiking, and military training. The high prevalence of blisters in active individuals underscores the fact that the pathomechanics of this condition are not fully understood. The traditional blister causation paradigm revolves around heat, moisture, and friction. In reality, foot friction blisters are caused by repetitive shear deformation. The 3 fundamental elements of blister-inducing shear deformation are (1) motion of bone, (2) high friction force, and (3) repetition of the resulting shear events. Rubbing at the skin surface is not a mechanism for friction blister formation. To that end, prevention of the friction blister continues to be an elusive quest for both the patient and the treating clinician. In this article, we aimed to highlight the limitations of the long-held blister-causation paradigm and offer a new explanation.

Key Words: shear, skin injury, foot injury

Key Points

- Repetitive shear deformation, not rubbing at the skin surface, causes foot friction blisters.
- Minimizing any of the 3 causative factors—motion of bone, high friction force, and repetition—will reduce the risk of friction blisters.

Individuals who pursue activities such as running, hiking, and military training often experience friction blisters on the feet. Researchers have documented that between 16% and 76% of runners[1–7] and between 29% and 95% of hikers[8–11] experience foot blisters. In many of these reports, friction blisters were the single most common injury. In an examination of the medical risks of wilderness hiking, Boulware et al[12] found that foot blisters occurred 4 times more frequently than tendinitis and 6 times more frequently than ankle sprains. Of note, a foot blister was associated with a 50% increased likelihood of Marine recruits experiencing an additional training-related musculoskeletal injury.[13] Clearly, preventing friction blisters in active people is an important goal. In this article, we present a new model for blister causation. Specific prevention techniques regarding friction blister mechanisms of action and the evidence base are discussed in a companion article.[14]

HISTOLOGY OF THE FRICTION BLISTER

The skin is designed to provide protection from external stressors and preserve internal water-rich molecules. It consists of the outer epidermis and the underlying dermis. The epidermis predominantly contains keratinocytes generated at the basal layer. As these cells progress superficially, they produce more keratin, dry out, and flatten before finally being shed (Figure 1). The epidermis adheres to the dermis via interlocking projections called *epidermal ridges*. The dermis provides a deformable but highly elastic physical support for the epidermis.[15] The plantar dermis has a higher collagen content than the dorsal dermis.[16] This is advantageous for the plantar surface of the foot because collagen fibers are able to store, transmit, and dissipate elastic energy during mechanical deformation, thus protecting it from mechanical failure.[17]

Fluid-filled friction blisters are most common on the "thick skin" of the plantar and palmar surfaces, where the epidermis is up to 10 times thicker than "thin skin."[18] Thick skin contains an additional epidermal layer called the *stratum lucidum*, is devoid of hair, and exhibits ridges and abundant eccrine sweat glands that aid in temperature regulation.[19,20] These unique features are directly relevant to the functional requirements of the anatomic location.[21–24] For example, a thicker corneum allows exfoliation without abrasion. In addition, hairless, moist, and ridged skin enhances the frictional properties of the skin surface with the environment, increasing traction.

Friction blisters are an injury within the epidermis, specifically the stratum spinosum, just above the basal layer.[25] The stratum spinosum consists of keratinocytes that are attached to one another by small interlocking cytoplasmic processes, desmosomes, and an intercellular cement of glycoproteins and lipoproteins.[18] In early blister research, investigators[21,26,27] demonstrated that friction blister formation has 2 stages: an intraepidermal tear and filling of the void with fluid. A consistent pattern of clinical signs occurs during blister development: initial redness, followed by blanching, development of a small pleat in the epidermis, and subsequent filling of the pleat with fluid.[28] The blister

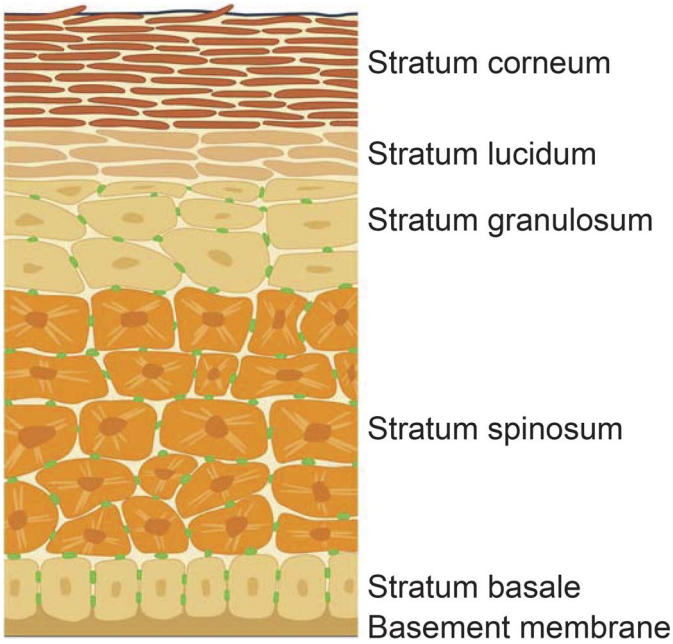

Figure 1. A schematic representation of the 5 layers of the epidermis of "thick skin" present on the plantar and palmar surfaces, from deep to superficial: stratum basale, stratum spinosum, stratum granulosum, stratum lucidum (absent in "thin skin"), and the stratum corneum.

does not fill with fluid immediately after the epidermal tear occurs, but it is fully filled within 2 hours.[21,29,30] Blister fluid is thin and colorless, similar to plasma with a lower protein level.[31,32]

PATHOMECHANICS OF THE FRICTION BLISTER

In this section, we describe friction and shear in the human foot during ambulation. During walking and running, the foot approaches the ground at an anteriorly directed angle to the supporting surface. Application of force to the foot at initial contact is therefore tangentially directed and is termed a *shear force* (Figure 2). If the foot strikes the ground in a purely vertical or perpendicular direction to the ground, the force exerted on the foot is termed *compressive force*, also known as *vertical ground reaction force*. In reality, the human foot is subject to both shear force and vertical ground reaction force during ambulation.[34,35] Researchers[35,36] have shown that plantar pressures are increased across the entire foot when running versus walking and that peak plantar pressure is higher in the rearfoot than the forefoot in both activities.

During push-off, the foot experiences a second shear force that is posteriorly directed (Figure 3). This shear force is generated by elastic recoil of the Achilles tendon pushing the forefoot against the ground in a plantar and posterior direction.

Yavuz et al[37] characterized the biphasic shear event in a person walking with anterior shear force during the loading response and midstance phases and posterior shear force during terminal stance and preswing phases (Figure 4). In addition to the plantar surface, shear forces are experienced over the whole foot, including the common blister sites of the posterior heel, interdigital spaces, and dorsum of the digits.

Figure 2. Initial contact creates anterior-directed shear force. Illustration courtesy of Kevin Rosenbloom, CPed. Reproduced with permission from SNCSC.[33]

Consider the scenario of a runner's heel striking the ground. Before touch-down, the foot approaches the ground from a direction that is angulated from vertical. The foot impacts the ground at an angle that is neither perpendicular

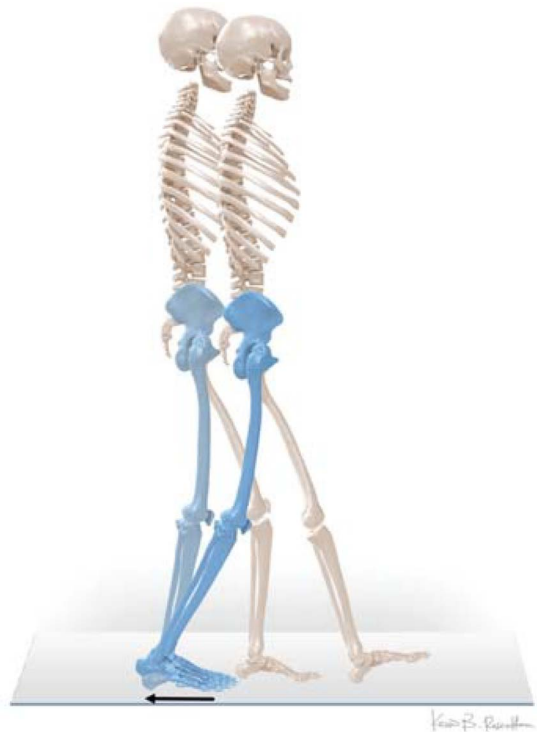

Figure 3. Push-off creates posterior-directed shear force. Illustration courtesy of Kevin Rosenbloom, CPed. Reproduced with permission from SNCSC.[33]

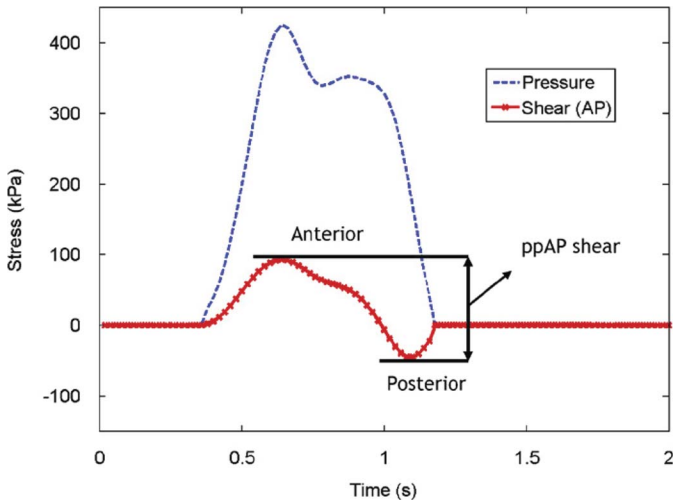

Figure 4. Pressure and shear (AP) curves of a representative diabetic subject obtained by a single transducer. Peak-to-peak AP shear was determined by adding the absolute values of maximum anterior and posterior shear magnitudes for each transducer. Abbreviations: AP, anteroposterior; pp, peak-to-peak. Used with permission of Elsevier; permission conveyed through Copyright Clearance Center, Inc.[37]

nor parallel to the ground, while the ground pushes back against the foot in the opposite direction. Given that the impact force is directed tangential to the plantar surface of the foot, shear force results. The magnitude of the shear force is called *shear stress*, which is an objective measure of shear force expressed in dynes per square centimeter.

During walking or running while shod, shear force is applied to the bottom of the shoe at initial contact from an anterior-to-posterior direction while the shoe is moving into the ground from a posterior-to-anterior direction. These shear forces on the shoe are therefore applied in opposite but parallel directions. The force that opposes the motion of the 2 bodies (shoe and ground) is called the *friction force*. The terms *friction* and *friction force* are synonymous and describe a parallel force that opposes the movement of 1 surface across another. The friction forces at the skin-footwear interfaces and the shoe-ground interface provide critical contributions to the braking force generated by muscular contraction during initial contact.[38] The motion of the shoe sliding across the ground at initial contact is termed *rubbing* or *slipping. Sliding*, *rubbing*, and *slipping* describe a motion, not a force.

The amount of sliding of the shoe on the ground depends on the coefficient of friction (COF) that exists between the surfaces. The *coefficient of friction* is a dimensionless value describing the ratio of friction force between 2 surfaces and the "normal force" (that is, compressive force) pressing them together. It is represented by the formula $\mu = F/N$, where μ is the COF, F is friction force, and N is normal force. A low COF produces low friction force with earlier and increased sliding between the 2 surfaces. A high COF produces high friction force with little or no sliding between the surfaces.

An interface exists between the shoe and the ground, established by 2 materials in parallel contact. Sliding or rubbing can occur at this interface, which is resisted by a friction force that depends on the COF between the surfaces.

Shear force is also exerted on the material within the sole of the shoe and on the entire foot inside the shoe. The rubber material of the midsole of a running shoe distorts as the ground pushes backward against the shoe at contact while the shoe is moving forward. These conflicting motions within the material of the sole of the shoe create shear strain, also described as *shear deformation* or *shear distortion*. This is characterized by stretching or deformation of the rubber material. *Shear strain* is the deformation response to the applied shear force in a material per unit area. *Shear modulus* is a measure of the elastic stiffness of the material that is deforming under the applied shear force.

The integument on the bottom of the foot receives the same shear forces as the shoe does during ambulation. An interface exists between the pedal skin surface and the sock worn by the individual. Another interface exists between the sock surface and the lining material of the shoe. Friction force determines how much sliding or rubbing occurs between the materials at various interfaces.

The cellular material within the pedal integument and soft tissues undergoes shear deformation or strain as shear force is applied to the foot (Figure 5). No movement interface is present deep to the skin surface, so no friction is present within the cellular layers of the pedal integument. The cellular layers of the integument are secured with connective tissue that has elastic properties, measured by a shear modulus that determines the ability of the tissue to stretch and rebound when subjected to shear stress. When the shear force or stress exceeds the elastic limits of the integument, a yield point occurs where the connections between the cellular layers (material) fail and tear.[25] This is the initiation of the blister injury. The higher the shear magnitude, the fewer the repetitions required to cause a blister.[27]

In the friction blister, repetitive shear deformation results in mechanical fatigue, failure, or a tear within a specific layer of the epidermis, the stratum spinosum.[21,23,25,26,28,39–41] The *stratum spinosum* (Figure 1) appears to be the zone where either maximal shear deformation occurs or resistance to shear deformation is least, leading to mechanical tearing of tissue and formation of the friction blister. Symptoms reported by participants during experimental blister research[28] have followed a pattern of "rubbing" during load application, a "stinging" sensation, and finally, a "sharp" pain indicating the intraepidermal tear.

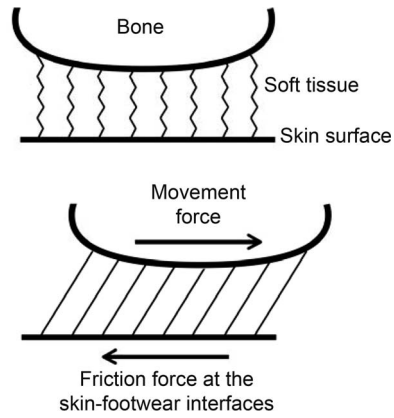

Figure 5. Simple shear diagram showing the critical influences of friction force and bone movement to the development of shear deformation.

This description focuses on the first shear event occurring on the plantar surface of the foot during the loading-response phase of the gait cycle. A second event occurs during terminal stance with all forces now exerted in the opposite direction. In Figure 4, we can appreciate that the terminal stance and preswing shear event lasts longer than the initial contact shear event. This may account for friction blisters being far more frequent at the plantar aspect of the forefoot than at the plantar aspect of the heel of active individuals.[11,42,43] Given that plantar pressures are higher in the heel than in the forefoot during walking and running, the role of shear forces rather than pressure must be emphasized in the pathomechanics of foot friction blisters.[35,36]

The anteriorly and posteriorly directed shear forces on the human foot are generated by gravity as the foot strikes the ground from an angle. Muscles then contract to provide "braking" or constraining inertia to prevent a forward fall. They generate contractile force that is exerted on tendons that attach to bone and thus restrain the motion of the skeletal segments.

During walking and running, push-off is the result of elastic recoil or release of elastic energy that has been stored in tendons that are attached to bones. The primary tendons generating push-off force in the foot are the Achilles tendon, which attaches to the calcaneus, and both the flexor digitorum longus and flexor hallucis longus, which attach to the phalanges.[44] During push-off, these tendons pull at their insertions on the respective bones in a posterior direction, creating a tangential shear force between the entire foot and the ground. The "pull" or recoil of the tendons is exerted on specific bones of the foot, which then transmit the push-off force through various soft tissue layers to the skin surface.

For maximal efficiency, the foot should not slip within the shoe, and the shoe should not slip against the supporting surface as the tendons contract and pull the bones of the forefoot in a downward and posterior direction.[38] Zero slippage would occur if maximal friction force was present between the interface of the skin and the sock, the sock and the shoe, and the shoe and the ground. If this occurred, then maximal shear force would be exerted on the material sandwiched between the bones and the superficial layer of skin. In this scenario of zero slippage, the bones are moving backward and the skin is not. A conflict results, with deep movement of the bones and no movement external to the skin surface. Therefore, maximal shear distortion occurs in the soft tissue located between the bones and the skin surface (Figure 5).

On the plantar surface of the foot, the soft tissue material that is sandwiched between the bones and the skin surface consists of the 5 layers of the epidermis, the dermis, the superficial fascia, and the deep fascia. These layers do not behave like a rigid body because they have a low shear modulus and deform and recoil under shear stress. As the bones are pulled backward, motion occurs within the layers of soft tissue, especially when no motion is present at the skin surface. Each layer of the soft tissue sandwich has different material properties, so the transmission of force from the bones to the skin surface causes varying degrees of deformation. In this regard, the bones as well as the various layers of soft tissue will move "out of synch" with each other, creating a shear distortion. If the elastic limits of the soft tissue exceed the yield point on the stress-strain curve, structural failure will occur as tearing of the interconnective tissue elements. Thus, the greatest potential for shear deformation within the soft tissue material sandwiched between the bone and skin surface would arise if the skin did not move with the bone. In this situation, the skin surface and footwear interfaces remain stationary while the adjacent bone moves. The more the skin moves with the bone, or in synch with the bone, the lower the magnitude of shear deformation. For this to happen, slippage must occur at one of the interfaces to allow skin motion in response to bone motion. Both the timing and magnitude of skin slippage will determine the overall shear stress that occurs within the soft tissue layers. The earlier the slippage response, the lower the shear stress within the layers of the epidermis. Hence, these layers are moving in synch with the underlying bone at an earlier point in that individual shear distortion.

However, greater skin slippage reduces efficiency for propulsion. The fact that most humans can walk and run leisurely without sustaining skin injury, either barefoot or in shoes, demonstrates that the human foot has innate features allowing shear distortions to occur without exceeding the yield point of elastic deformation within the material layer of soft tissue, particularly on the plantar surface of the foot.

Shear forces during push-off are generated by muscles pulling the bones of the feet, not directly pulling on the skin. Shear forces are not generated by the skin or at the skin surface but by bone movement under the skin. Frictional force acts on the skin surface as it keeps the foot in stable contact with the footwear for maximal braking and propulsive efficiency. Shear forces from bone motion create shear deformation in the deeper layers of the soft tissue beneath the skin. The injury known as a friction blister occurs not at the skin surface but within the layers of material sandwiched between the bone and the skin surface. Specifically, it is an injury at a specific level of the epidermis, the stratum spinosum.

Even though excessive friction force on the skin surface is easily blamed as the primary cause of friction blisters, the roles of the bones and deep fascia that actually generate the shear forces in the soft tissue layers of the foot need to be evaluated. Further researchers should determine whether physical properties of the epidermis, dermis, superficial fascia, and deep fascia differ between individuals or are altered on a day-to-day basis by hydration or other whole-body factors. In addition, the roles of walking and running form, such as stride length and striking patterns, should be considered for their effects on bone excursion in the feet, because these may be critical factors in creating shearing force in discrete locations and may render certain individuals more vulnerable to friction blisters.

Vertical compression force is a necessary component of blister formation. Higher pressure contributes to higher friction force, which, in the presence of moving bones, leads to larger shear distortions. Pressure is created by gravity, muscular contraction, bony architecture, and the position of bones relative to one another. These factors contribute to the overall mechanism of bone movement causing friction blisters on the feet.

Finally, the repetition of shear deformation within the skin is a fundamental factor in blister causation.[23,25,45] This

repetition factor is reinforced by the observation that blisters are more common in activities where more steps are taken, such as long-distance running, hiking, and endurance military training. In their experimental blister studies, Sulzberger et al,[21] Naylor,[27] and Hashmi et al[28] used the number or duration of shear cycles required to precipitate the initial blister injury as the endpoint of data collection. Indeed, an inverse relationship exists between the magnitude of shear deformation and the number of shear cycles required to produce the intraepidermal mechanical fatigue.[41,45]

HISTORICAL OVERVIEW

In this section, we discuss traditional beliefs that do not consider all the contributing factors of blister formation. The traditional blister causation paradigm revolves around a relationship among heat, friction, and moisture.

Heat has a role in blister formation. Friction blisters are more frequent in hotter environmental conditions.[46,47] Furthermore, an increase in skin temperature precedes blister formation, and a prolonged increase in temperature is present after a blister forms.[28] However, histologic studies have shown that friction blisters are not a thermal injury.[23,25,27–30,46,48]

Moisture from perspiration is also relevant to blister formation because it increases friction force.[26,27,30,49–51] Naylor[27] confirmed that higher ambient temperatures are associated with increased perspiration. Researchers[52–54] have confirmed that faster walking speeds increase in-shoe temperature and perspiration.

Authors have verified that increased heat creates increased moisture on the feet, which then increases friction force inside the shoe. This paradigm is widely accepted in the sporting community. However, the concept of *friction* is often misunderstood and requires some clarification. The scientific definition is clear: the resistance to movement of 1 surface across another.[55–59] Nevertheless, others[57–59] have equated friction with *rubbing*, which is the movement of 1 surface across another. This second definition is assumed by the lay population but is also used in some medical publications on shear-related skin injury, including blisters.[60–62] These conflicting definitions of *friction* contribute to a common misunderstanding that friction blisters are caused by an object (shoe or sock) moving across the skin. This misconception may have led to the popularity of tape application as a method of blister prevention despite a lack of evidence.[40,63] The expectation is that the tape functions as a physical barrier to objects rubbing against the skin. However, the presence of tape adhered to the skin does not negate the shear deformation of the soft tissues directly under it. An example of this misunderstanding about how blisters form was presented by Krabak et al[61(p187)]:

> Understanding the mechanism of a friction blister injury can help in addressing prevention. The main etiology is the repeated action of skin rubbing against another surface. As the external contact of either sock or footwear moves across the skin, the frictional force (F_f) opposes this movement. When horizontal shear forces overcome this resistance, repeated sliding as a friction point causes exfoliation of the stratum corneum and erythema in and around this zone.

Another more recent example from DeBois et al[64(pp1–2)] suggested blisters result from a superficial abrasion:

> Friction blisters are formed as a result of abrasion caused by the frictional forces applied directly to the skin's top layer of the epidermis, the stratum corneum. The frictional force applied to the stratum corneum is transmitted through the stratum granulosum into the stratum spinosum of the epidermis causing micro tearing in between skin layers.

The movement force or shear force that generates blister formation originates from the bones moving relative to the soft tissues within the foot. This concept of movement of skeletal segments within the foot during gait is rarely recognized as a critical element for the development of shear force within the soft tissues. In reality, rubbing at the skin surface is not a mechanism for the formation of friction blisters, and most prevention strategies, such as lubricants and all moisture management strategies, actually encourage rubbing by reducing the friction force at the skin-sock interface.

THE NEW PARADIGM

Understanding the pathomechanics of the friction blister is essential to effective selection and implementation of prevention strategies. A change in beliefs is necessary to implement newer prevention strategies based on research findings that verify how friction blisters in the feet actually occur. The notion of blisters as a superficial-to-deep wear injury from external rubbing needs to be abandoned. Furthermore, no evidence that friction blisters are a thermal burn exists.[23,28–30,46] Instead, friction blisters must be viewed as a mechanical injury in the deeper layers of the epidermis resulting from repetitive shear deformation. For the clinician, the following succinct description of the friction blister is proposed: Blisters are an intraepidermal tear caused by repetitive shear deformation. During ambulation, the muscles pull the bones of the feet while high friction forces between the pedal skin, multiple footwear interfaces, and the ground maximize efficiency for push-off. Motion of the bones relative to the stationary skin creates shear deformation within the tissues beneath the skin surface. Repetitive shear deformation results in mechanical failure or tearing within the stratum spinosum layer of the epidermis, which later fills with plasma-like fluid to create the actual blister.

For the patient, the following simple description of the friction blister is proposed: Blisters are a tear under the skin surface caused by the skin and bone moving out of synch. As the bones in the foot move with each step, the skin does not immediately follow, so the soft tissues located between the skin surface and the bones stretch and distort. This is called *shear deformation*. If there is too much shear, a tear develops within the layers of skin that later fills with fluid to form the actual blister.

CONCLUSIONS

Foot friction blisters are caused by repetitive shear deformation. The 3 fundamental elements of blister formation are (1) motion of the bone, (2) high friction force, and (3) repetition of the resulting shear events. Minimizing any of these 3 elements will potentially reduce the risk of friction blisters. The first element of bone movement has been

largely ignored or unrecognized, especially regarding the potential it holds for preventing blisters. The second element of friction must be clarified because its role in blisters is based on lack of motion at the skin-footwear interfaces. Finally, the third element of repetition should be recognized as one that can be controlled and modulated to allow adaptation and reduced risk of blister injury. These concepts are expanded in a subsequent article devoted to implementing strategies for preventing friction blisters in the feet.

REFERENCES

1. Pasquina PF, Griffin SC, Anderson-Barnes VC, Tsao JW, O'Connor FG. Analysis of injuries from the Army Ten Miler: a 6-year retrospective review. *Mil Med*. 2013;178(1):55–60. doi:10.7205/milmed-d-11-00447
2. Scheer BV, Murray A. Al Andalus Ultra Trail: an observation of medical interventions during a 219-km, 5-day ultramarathon stage race. *Clin J Sport Med*. 2011;21(5):444–446. doi:10.1097/JSM.0b013e318225b0df
3. Mailler EA, Adams BB. The wear and tear of 26.2: dermatological injuries reported on marathon day. *Br J Sports Med*. 2004;38(4):498–501. doi:10.1136/bjsm.2004.011874
4. Mailler-Savage EA, Adams BB. Skin manifestations of running. *J Am Acad Dermatol*. 2006;55(2):290–301. doi:10.1016/j.jaad.2006.02.011
5. Hoffman MD, Fogard K. Factors related to successful completion of a 161-km ultramarathon. *Int J Sports Physiol Perform*. 2011;6(1):25–37. doi:10.1123/ijspp.6.1.25
6. Krabak BJ, Waite B, Schiff MA. Study of injury and illness rates in multiday ultramarathon runners. *Med Sci Sports Exerc*. 2011;43(12):2314–2320. doi:10.1249/MSS.0b013e318221bfe3
7. Scheer BV, Reljic D, Murray A, Costa RJ. The enemy of the feet blisters in ultraendurance runners. *J Am Podiatr Med Assoc*. 2014;104(5):473–478. doi:10.7547/0003-0538-104.5.473
8. Gardner TB, Hill DR. Illness and injury among long-distance hikers on the Long Trail, Vermont. *Wilderness Environ Med*. 2002;13(2):131–134. doi:10.1580/1080-6032(2002)013[0131:iaiald]2.0.co;2
9. Knapik JJ, Reynolds K, Barson J. Influence of an antiperspirant on foot blister incidence during cross-country hiking. *J Am Acad Dermatol*. 1998;39(2, pt 1):202–206. doi:10.1016/S0190-9622(98)70075-1
10. Boulware DR. Gender differences among long-distance backpackers: a prospective study of women Appalachian Trail backpackers. *Wilderness Environ Med*. 2004;15(3):175–180. doi:10.1580/1080-6032(2004)15[175:gdalba]2.0.co;2
11. Choi SC, Min YG, Lee IS, et al. Injuries associated with the 580 km university student grand voluntary road march: focus on foot injuries. *J Korean Med Sci*. 2013;28(12):1814–1821. doi:10.3346/jkms.2013.28.12.1814
12. Boulware DR, Forgey WW, Martin WJ II. Medical risks of wilderness hiking. *Am J Med*. 2003;114(4):288–293. doi:10.1016/S0002-9343(02)01494-8
13. Bush RA, Brodine SK, Shaffer RA. The association of blisters with musculoskeletal injuries in male Marine recruits. *J Am Podiatr Med Assoc*. 2000;90(4):194–198. doi:10.7547/87507315-90-4-194
14. Rushton R, Ritchie D. Friction blisters on the feet: a critical assessment of current prevention strategies. *J Athl Train*. 2024;59(1):8–21. doi:10.4085/1062-6050-0341.22
15. Elias PM, Williams ML. *A Primer on the Skin Barrier*. Elias Williams Medical Corp; 2013.
16. Vela-Romera A, Carriel V, Martín-Piedra MA, et al. Characterization of the human ridged and non-ridged skin: a comprehensive histological, histochemical and immunohistochemical analysis. *Histochem Cell Biol*. 2019;151(1):57–73. doi:10.1007/s00418-018-1701-x
17. Shah RG, Pierce MC, Silver FH. Morphomechanics of dermis—a method for non-destructive testing of collagenous tissues. *Skin Res Technol*. 2017;23(3):399–406. doi:10.1111/SRT.12349
18. Hunter JAA, Svain JA, Dahl MV. *Clinical Dermatology*. Blackwell; 1989.
19. Odom RB, Baker S, Shaps R. Dermatology. In: Zier BG, ed. *Essentials of Internal Medicine in Clinical Podiatry*. WB Saunders; 1990:386–435.
20. Koruga D, Bandić J, Janjić G, Lalović Č, Munćan J, Vukojević D. Epidermal layers characterisation by opto-magnetic spectroscopy based on digital image of skin. *Acta Physiol Pol A*. 2012;121(3):606–610. doi:10.12693/APhysPolA.121.606
21. Sulzberger MB, Cortese TA, Fishman L, Wiley HS. Studies on blisters produced by friction, I: results of linear rubbing and twisting technics. *J Invest Dermatol*. 1966;47(5):456–465. doi:10.1038/jid.1966.169
22. Comaish S, Bottoms E. The skin and friction: deviations from Amonton's laws and the effects of hydration and lubrication. *Br J Dermatol*. 1971;84(1):37–43. doi:10.1111/j.1365-2133.1971.tb14194.x
23. Knapik JJ, Reynolds KL, Duplantis KL, Jones BH. Friction blisters: pathophysiology, prevention, and treatment. *Sport Med*. 1995;20(3):136–147. doi:10.2165/00007256-199520030-00002
24. Zhang M, Mak AF. In vivo friction properties of human skin. *Prosthet Orthot Int*. 1999;23(2):135–141. doi:10.3109/03093649909071625
25. Comaish JS. Epidermal fatigue as a cause of friction blisters. *Lancet*. 13;1(7794):81–83. doi:10.1016/s0140-6736(73)90472-8
26. Naylor PF. The skin surface and friction. *Br J Dermatol*. 1955;67(7):239–246. doi:10.1111/j.1365-2133.1955.tb12729.x
27. Naylor PF. Experimental friction blisters. *Br J Dermatol*. 1955;67(10):327–342. doi:10.1111/j.1365-2133.1955.tb12657.x
28. Hashmi F, Richards BS, Forghany S, Hatton AL, Nester CJ. The formation of friction blisters on the foot: the development of a laboratory-based blister creation model. *Skin Res Technol*. 2013;19(1):e479–e489. doi:10.1111/j.1600-0846.2012.00669.x
29. Cortese TA, Griffin TB, Mc M, Layton LL, Hutsell TC. Experimental friction blisters in Macaque monkeys. *J Invest Dermatol*. 1969;53(2):172–177.
30. Akers WA, Sulzberger MB. The friction blister. *Mil Med*. 1972;137(1):1–7.
31. Schmid P. Quantitation of specific proteins in blister fluid. *J Invest Dermatol*. 1970;55(4):244–248. doi:10.1111/1523-1747.ep12259948
32. Cortese TA II, Sams WM II, Sulzberger MB. Studies on blisters produced by friction, II: the blister fluid. *J Invest Dermatol*. 1968;50(1):47–53. doi:10.1038/jid.1968.7
33. Richie D. Human walking: the gait cycle. In: *Pathomechanics of Common Foot Disorders*. 1st ed. Springer; 2021:45–61. doi:10.1007/978-3-030-54201-6
34. Malisoux L, Delattre N, Meyer C, Gette P, Urhausen A, Theisen D. Effect of shoe cushioning on landing impact forces and spatiotemporal parameters during running: results from a randomized trial including 800+ recreational runners. *Eur J Sport Sci*. 2021;21(7):985–993. doi:10.1080/17461391.2020.1809713
35. Lung CW, Mo PC, Cao C, et al. Effects of walking speeds and durations on the plantar pressure gradient and pressure gradient angle. *BMC Musculoskelet Disord*. 2022;23(1):823. doi:10.1186/s12891-022-05771-2
36. Chuckpaiwong B, Nunley JA, Mall NA, Queen RM. The effect of foot type on in-shoe plantar pressure during walking and running. *Gait Posture*. 2008;28(3):405–411. doi:10.1016/j.gaitpost.2008.01.012
37. Yavuz M, Tajaddini A, Botek G, Davis BL. Temporal characteristics of plantar shear distribution: relevance to diabetic patients. *J Biomech*. 2008;41(3):556–559. doi:10.1016/j.jbiomech.2007.10.008
38. Morio CYM, Herbaut A. Neuromechanical adaptations to slippery sport shoes. *Hum Mov Sci*. 2018;59:212–222. doi:10.1016/j.humov.2018.04.016
39. Spence WR, Shields MN. Prevention of blisters, callosities, and ulcers by absorption of shear forces. *J Am Podiatr Med Assoc*. 1968;58(10):428–434. doi:10.7547/87507315-58-10-428
40. Richie D II. How to manage friction blisters. *Podiatry Today*. 2010;23(6):42–49.
41. Carlson JM. Functional limitations from pain caused by repetitive loading on the skin: a review and discussion for practitioners, with new data

for limiting friction loads. *J Prosthet Orthot*. 2006;18(4):93–103. doi:10.1097/00008526-200610000-00002
42. Herring KM, Richie DH II. Friction blisters and sock fiber composition: a double-blind study. *J Am Podiatr Med Assoc*. 1990;80(2):63–71. doi:10.7547/87507315-80-2-63
43. Herring KM, Richie DH II. Comparison of cotton and acrylic socks using a generic cushion sole design for runners. *J Am Podiatr Med Assoc*. 1993;83(9):515–522. doi:10.7547/87507315-83-9-515
44. Perry J, Burnfield JM. *Gait Analysis: Normal and Pathological Function*. 2nd ed. SLACK Inc; 2010.
45. Hoffman MD. Etiological foundation for practical strategies to prevent exercise-related foot blisters. *Curr Sports Med Rep*. 2016;15(5):330–335. doi:10.1249/JSR.0000000000000297
46. Griffin TB, Cortese TA Jr, Layton LL, Sulzberger MB. Inverse time and temperature relationship in experimental friction blisters. *J Invest Dermatol*. 1969;52(4):391.
47. Knapik J, Reynolds K, Staab J, Vogel JA, Jones B. Injuries associated with strenuous road marching. *Mil Med*. 1992;157(2):64–67.
48. Patterson HS, Woolley TW, Ledner WM. Foot blister risk factors in an ROTC summer camp population. *Mil Med*. 1994;159(2):130–135.
49. Nacht S, Close J-A, Yeung D, Gans EH. Skin friction coefficient: changes induced by skin hydration and emollient application and correlation with perceived skin feel. *J Soc Cosmet Chem*. 1981;32(2):55–65.
50. El-Shimi AF. In vivo skin friction measurements. *J Soc Cosmet Chem*. 1977;28(2):3751.
51. Kirkham S, Lam S, Nester C, Hashmi F. The effect of hydration on the risk of friction blister formation on the heel of the foot. *Skin Res Technol*. 2014;20(2):246–253. doi:10.1111/srt.12136
52. Covill D, Guan Z, Bailey M, Pope D. Effects of varying the environmental conditions on in-shoe temperature. In: Milburn P, ed. *Proceedings of the 6th Symposium on Footwear Biomechanics*. Footwear Biomechanics Group; 2003:18–19.
53. Hall M, Shurr DG, Zimmerman MB, Saltzman CL. Plantar foot surface temperatures with use of insoles. *Iowa Orthop J*. 2004;24:72–75.
54. Li P-L, Yick K-L, Yip J, Ng S-P. Influence of upper footwear material properties on foot skin temperature, humidity and perceived comfort of older individuals. *Int J Environ Res Public Health*. 2022;19(17):10861. doi:10.3390/ijerph191710861
55. Friction. Cambridge University Press. Accessed August 18, 2021. https://dictionary.cambridge.org/dictionary/english/friction
56. Friction. Wikipedia. Accessed August 18, 2021. https://en.wikipedia.org/wiki/Friction
57. Friction. Merriam-Webster Inc. Accessed August 18, 2021. https://www.merriam-webster.com/dictionary/friction
58. Friction. Oxford University Press. Accessed August 18, 2021. https://www.oxfordlearnersdictionaries.com/definition/english/friction?q=friction
59. Friction. Macmillan Education Limited. Accessed August 18, 2021. https://www.macmillandictionary.com/dictionary/british/friction
60. Lipman GS, Sharp LJ, Christensen M, et al. Paper tape prevents foot blisters: a randomized prevention trial assessing paper tape in endurance distances II (Pre-TAPED II). *Clin J Sport Med*. 2016;26(5):362–368. doi:10.1097/JSM.0000000000000319
61. Krabak BJ, Waite B, Lipman G. Injury and illnesses prevention for ultramarathoners. *Curr Sports Med Rep*. 2013;12(3):183–189. doi:10.1249/JSR.0b013e3182913c98
62. Antokal S, Brienza D, Bryan N, et al. Friction induced skin injuries—are they pressure ulcers? A National Pressure Ulcer Advisory Panel white paper. National Pressure Ulcer Advisory Panel. Published November 20, 2012. Accessed 2013. https://cdn.ymaws.com/npiap.com/resource/resmgr/white_papers/1c._npuap-friction-white-pap.pdf
63. Brennan FH II. Managing blisters in competitive athletes. *Curr Sports Med Rep*. 2002;1(6):319–322. doi:10.1249/00149619-200212000-00003
64. DeBois IJ, Agarwal E, Kapoor A, Mathur K. Tribology of the sock-skin interface—the influence of different fabric parameters on sock friction. *J Foot Ankle Res*. 2022;15(1):61. doi:10.1186/S13047-022-00560-5

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