

Skin manifestations of running

Erica A. Mailler-Savage, MD,^a and Brian B. Adams, MD, MPH^{a,b}
Cincinnati, Ohio

As the United States comes increasingly closer to being the heaviest nation on earth, many people are turning to exercise, especially running, to lose weight. Most runners, whether novice or professional, will have a skin disorder that may prompt them to seek medical attention. Although case reports and sports reviews have discussed, in a cursory fashion, the nature of these skin lesions, to our knowledge there has never been an extensive review of the literature that specifically addresses the skin diseases of runners. In this article, we present the epidemiology, origin, clinical characteristics, treatment, and prevention of skin diseases inherent to runners. (J Am Acad Dermatol 2006;55:290-301.)

The skin conditions affecting runners can be divided into infections, inflammatory conditions, trauma, and environmental injuries. Although many are benign, some seriously affect the runner's performance, and a few are potentially life threatening.

INFECTIONS

Tinea pedis

There is an epidemic of tinea pedis in athletes and runners. The Achilles Project, which included 87,793 study participants, found that sports-active individuals were roughly twice as likely to develop tinea pedis compared with nonactive individuals.¹ Two other studies, specifically targeting marathon runners, detected a prevalence of 22%² and 31%³ by potassium hydroxide microscopic examination and culture (Table I).

Trichophyton rubrum and *T mentagrophytes* are responsible for most cases of tinea pedis.⁴⁻¹² *T rubrum* causes the interdigital and scaly moccasin variants whereas *T mentagrophytes* causes the inflammatory or vesicular type (Fig 1).^{4,10} Risk factors for the development of tinea pedis include occlusion of the skin with increased carbon dioxide tension, perspiration with secondary maceration of the epidermis, trauma, and using showers with colonized floors.^{1,5,7,8,10,12-19}

Several treatment options are available for tinea pedis. Astringent soaks may be used in the web spaces to debride macerated tissue that is a common site for secondary infection.^{15,19-21} Topical antifungals are frequently used but are less effective used alone because of frequent reinfection and the necessity to apply them multiple times per day. The optimal approach to therapy for tinea pedis consists of topical antifungals, meticulous foot and shoe hygiene, and oral drying agents to decrease exertional focal hyperhidrosis. Oral antifungals should be reserved for extensive disease and treatment failures (Table II).^{7,8,10,18,19,22-25} Prevention of tinea pedis is best accomplished by removing moist socks, wearing socks made of synthetic material, wearing ventilated shoes, wearing sandals in the locker room and showers, and applying powder to the feet after bathing or before workouts.*

Gram-negative toe web infections

Although not specifically reported in runners, individuals frequenting dressing rooms, swimming pools, and hot tubs risk gram-negative bacterial toe web infection.²⁸ Clinically, athletes will present with erythematous, eroded, severely macerated and foul-smelling areas on the toes extending to the soles. Existing dermatophyte infection predisposes for these bacterial infections.²⁸ Topical and oral antibacterial therapies along with attentive foot care (including daily warm water soaks) clear the eruption.

Plantar verruca

Human papillomavirus, a papovavirus,^{14,15,29} causes warts on the feet that can interfere with

From the Department of Dermatology, University of Cincinnati,^a and Veterans Administration Medical Center.^b

Funding sources: None.

Conflicts of interest: None identified.

Reprint requests: Brian B. Adams, MD, MPH, University of Cincinnati, College of Medicine, Department of Dermatology, PO Box 670592, Cincinnati, OH 45267-0592. E-mail: adamsbb@email.uc.edu.

0190-9622/\$32.00

© 2006 by the American Academy of Dermatology, Inc.

doi:10.1016/j.jaad.2006.02.011

*References^{4,5,8,10,15,17,20,26,27}

Table I. Injuries reported during marathon events

Event (y) author	Study method	No. of race entrants	No. of study entrants	Lesions studied	No. of people affected
The Sun City-to-Surf Runs (1976-1979) Richards et al ⁸³	Review of medical records from first-aid tent	9800 (1976)	184	Blisters	47 (26%)*
		11,450 (1977)	226		32 (14%)*
		16,800 (1978)	196		35 (18%)*
		16,200 (1979)	237		59 (25%)*
				Chafing	60 (33%)* 78 (35%)* 59 (30%)* 66 (28%)*
1973 Classical Marathon Race of Athens (1973) Orava ⁸⁴	Survey sent to runners 1 mo after race	139	94	Blisters and chafing (during race)	24 (26%)*
				Jogger's nipples (during race)	2 (2%)*
				Blisters, chafing, loss of toenails with persistence >1 wk (after race)	13 (14%)*
Mayor Daley Marathon (1977) Nequin ⁸⁵	Injuries reported at first-aid station	4300	372	Jogger's nipples	20 (5.4%)*
Women's National Marathon (1977) Nequin ⁸⁵	Informal survey at marathon	98	98	Jogger's nipples	16 (16.3%)*
Midnight Sun Marathon (The Mayor's Marathon) (1980) Caldwell ⁸⁶	Survey sent 3 wk after race	252	84	Blisters	11 (19%)*
Big M Melbourne Marathon (1980) Duras et al ⁸⁷ and Kretsch et al ⁸⁸	Survey of those who sought treatment during the race	5423	97	Blisters	13 (13%)*
			(Men only) 84	Blisters, corns	7 (8.5%)*
London Marathon (1982-1984) Temple ⁸⁹ and Cerio et al ⁹⁰	Report of runners treated at first-aid stations during race	1700 (1982)	Unknown	Blisters	12 (0.7%) [†]
				Chafing	10 (0.5%) [†]
				Toe injury (1982)	2 (0.1%) [†]
		Unknown (1983, 1984)		Blisters	0 (1983) 1 (1984)
Sheffield Marathon (1982) Nicholl et al ⁹¹⁻⁹³	Report of runners treated at first-aid stations	2289	409	Skin lesions, including blisters	94 (4.2%) [†]
Twin Cities Marathon (1982-1994) Roberts ⁹⁴	Review of medical records from first-aid tents	81,277	1534	Abrasions	27 (1.9%)*
		(Total for all y)		Blisters	289 (19.9%)*

Continued

Table I. Cont'd

Event (y) author	Study method	No. of race entrants	No. of study entrants	Lesions studied	No. of people affected
Glasgow Marathon (1982-1985, 1987) Ridley et al ⁹⁵	Review of medical records from first-aid tents	8256 (1982)	Unknown	Blisters	152 (1.8%) [†]
		14,271 (1983)			81 (0.6%) [†]
		15,353 (1984)			54 (0.4%) [†]
		20,052 (1985)			33 (0.2%) [†]
		8600 (1987)			48 (0.3%)
Boston Marathon (1985, 1987) Adner et al ⁹⁶	Review of medical records from first-aid tents	5122 (1985)	536	Blisters	102 (19%)*
		6364 (1987)	93		9 (10%)*
Wonderful Copenhagen Marathon and Danish National Marathon Championship (1986) Hölmich et al ⁹⁷ and Darre et al ⁹⁸	Review of medical records from medical tent	2520 (Total in race)	40	Blisters	Most common presenting symptom
	Two questionnaires given to championship participants: one for injuries during race and one for injuries after race	60 (Championship participants)	60	Blisters (during run) Other skin injuries (during run) Blisters (after run) Other skin injuries (after run)	10 (16%)* 10 (16%)* 16 (26%)* 11 (18%)*
Arhus Marathon (1986) Jakobsen et al ⁹⁹	Questionnaire at race that asked about injuries during training	Unknown	161	Blisters	40 (25%)*
International Marathon of Montreal (1988) Auger et al ²	Interviewed before race	Unknown	405	Tinea pedis	89 (22%)*
Grandma's Marathon (1989-1991, 1993-1995) Crouse et al ¹⁰⁰	Review of medical records from first-aid tent	3742 (1989) [‡]	385	Blisters	89 (23%)*
		5334 (1990) [‡]	405		89 (22%)*
		5150 (1991) [‡]	242		61 (25%)*
		5640 (1993) [‡]	141		31 (22%)*
		5193 (1994) [‡]	343		72 (21%)*
6528 (1995) [‡]	261	60 (23%)*			
Grandma's Half Marathon (1991, 1993-1995) Crouse ¹⁰⁰	Review of medical records from first-aid tent	1500 (1991)	24	Blisters	7 (29%)*
		2000 (1993)	30		7 (23%)*
		2100 (1994)	74		13 (18%)*
		2472 (1995)	40		16 (41%)*
Auckland Citibank Marathon (1993) Satterthwaite et al ^{101,102}	Review of medical records from first-aid tent	1219	75	Blisters	11 (14.5%)*

Continued

Table I. Cont'd

Event (y) author	Study method	No. of race entrants	No. of study entrants	Lesions studied	No. of people affected
	Cohort study; questionnaire 2 d before marathon to assess risk factors, postrace questionnaire 1 wk after race		875	Blisters Chafing Abrasions	345 (39%)* 140 (16%)* 13 (2%)*
New York City Marathon (1994) Caselli et al ¹⁰³	Survey for people who presented to first-aid stations for podiatry care	29,735	265	Corns/calluses/ blisters	109 (41%)*
Médoc Marathon (1998) Lacroix et al ³	Random interviews at end of run; feet examined and tissue taken to determine if dermatophytes present	7500	147	Tinea pedis	45 (31%)*
Great North Run (unknown) Sainsbury ¹⁰⁴	Review of medical records from first-aid tents	57,570 (Half marathon)	3006		513 (0.9%) [†]
		9330 (marathon)	445	Skin problems	62 (0.7%) [†]

Adapted from Mailler and Adams.¹² Reproduced with permission from the BMJ Publishing Group.

*Percentage based on number of entrants in study.

[†]Percentage based on number of entrants in race.

[‡]Number of finishers.

running.⁴ The average incubation period for the virus is 6 months.^{8,10,23,24} Plantar verruca are well-defined, hyperkeratotic papules and plaques on the soles of the feet.^{5,14,20} On pressure sites, the lesions may become endophytic and very painful.^{20,23,24} Runners may be predisposed to this type of infection because of maceration of the epidermis caused by warmth and perspiration of the skin.^{8,10,15,18,30}

Warts should be differentiated from calluses and corns, which were reported by 8.5% to 41% of runners on marathon day (Table I). Warts can be identified by removing the overlying hyperkeratotic material to reveal pericapillary hemorrhages.^{8,10,15,18,30} Calluses should be examined to be sure that they are not harboring warts, because callused skin is more susceptible to papillomavirus.^{4,15}

Destroying warts will help prevent them from growing and causing pain, and may furthermore decrease transmission of the virus to others.¹³ Warts can be destroyed with cryosurgery, cantharidin, trichloroacetic acid, or keratolytic agents such as salicylic acid; however, these procedures can prevent the runner from immediately returning back

to training.* Topical imiquimod under occlusion is an effective and potentially less painful method of wart destruction that will decrease a runner's downtime.^{4,5,32} Topical tretinoin and topical fluorouracil applied twice daily for 2 to 3 months have also anecdotally been reported to work; daily paring may also be effective to reduce pain.^{8,15,23,24} Runners should wear sandals in the locker room and shower to prevent acquisition of the virus.^{4,5,13,26,32,33}

INFLAMMATORY CONDITIONS

Allergic contact dermatitis

Well-defined, erythematous, vesicular, crusted or eroded plaques characterize allergic contact dermatitis.^{4,26} It occurs in runners after contact with components in running shoes or in topical medicines used for injuries.^{4,8,18,26} Excessive moisture in the runner's socks predisposes him or her to shoe dermatitis by allowing chemicals in the rubber soles

*References^{5,10,17,20,23,24,31}



Fig 1. Inflammatory tinea pedis on the plantar surface of the foot.

such as ethylbutylthiourea,^{5,8,32,34-36} dibenzothiazyl disulfide,^{35,36} and mercaptobenzothiazole^{5,26,32,36,37} to seep through to the skin. Runners may also be allergic to formaldehyde resin in athletic or rubber-backed varieties of tape.^{5,8,19,36,37} Methyl salicylate^{36,37} and trolamine salicylate^{36,37} in topical analgesics, polymyxin B in topical antibiotics^{31,34} and arnica³⁸ in jogging cream may lead to dermatitis in some cases.

The diagnosis of allergic contact dermatitis is often clear from the patient's history and physical findings; however, patch testing may be necessary to determine the cause.²⁶ Topical steroids and systemic antihistamines may be used for relief from the pruritic eruption if necessary, although avoidance of the offending substance is the best treatment.^{4,5,8,20,26,32,39} Short courses of systemic steroids may be used for severe reactions.^{8,20,23,24,39} Although some authors suggest that wearing heavy cotton absorbent socks and changing socks more frequently to decrease moisture are the best ways to prevent contact dermatitis,^{19,23,24,31} others believe that sweating and subsequent allergen leaching usually will overcome these measures and that nonallergenic shoes are the best option.¹⁸ Shoe insoles can be made from polyurethane as an alternative to rubber-based products, and paper tape or coban may be used instead of athletic tape.⁵

Physical urticarias

A total of 14% of athletes, in comparison with 2.4% of the general population, have physical urticaria.^{4,26} Cholinergic urticaria, commonly reported in runners, is characterized by 2- to 4-mm, well-defined, erythematous, pruritic wheals that develop within 2 to 30 minutes after a general overheating of the body from environmental temperature, exertion, exercise, or stress.^{4,20,26,39,40} This type of urticaria has a predilection for the upper thorax and neck, but may spread to the rest of the body.⁴⁰ Systemic symptoms may include abdominal cramps, diarrhea, nausea, vomiting, salivation, headache, syncope, and

Table II. Treatment options for tinea pedis^{22,25}

Topical treatments for tinea pedis	Oral treatments for tinea pedis
Azoles*	Allylamines*
Undecenoic acid	Griseofulvin
Allylamines	Azoles
Tolnaftate	

*First-line agent (based on findings by the Cochrane Library Systematic Reviews).

hypotension.^{8,15,20,39,40} Antihistamines, specifically hydroxyzine, especially if taken before exposure to the offending agent, may be helpful.^{4,19,20,26,40}

Vibratory angioedema is a rare form of physical urticaria that seems to occur with increased frequency in people who also have cholinergic urticaria.^{41,42} Transient erythema and wheals form at the site of the vibration secondary to intraepidermal or intercellular edema and increased blood flow.^{43,44} Increased plasma histamine levels are present.^{43,44} This condition has been reported in a 28-year-old woman who experienced the symptoms in the back aspect of her thighs because of repetitive pounding related to running.⁴³ Prophylactic terfenadine (120 mg) allowed her to exercise without symptoms.⁴³

Exercise-induced anaphylaxis

Exercise-induced anaphylaxis (EIA) is a distinct but rare form of physical allergy, and is the most life threatening of the dermatologic manifestations of runners.^{36,37,45-47} The pathophysiology of EIA is unclear, but two mechanisms have been proposed. The first is activation of the alternative complement pathway and the second is IgE, lactate, or creatine phosphokinase mediated mast cell degranulation that results in increased serum levels of histamine.^{5,41}

Patients who are atopic and those with food allergies seem to be at highest risk.^{19,36,37,40,46,48} Eating before exercise seems to predispose athletes to developing lesions.^{5,45,47-49} Various inciting foods include shellfish, alcohol, tomatoes, cheese, milk, celery, wheat, and nuts.^{5,45,47-49} One case of cold-induced anaphylaxis has been reported in a runner.⁴⁹ An unusual case reported in the literature similar to EIA was a man who developed urticaria with subsequent anaphylaxis after running through a wheat field.⁵⁰ It is unknown what triggered the reaction, but it was believed to be some form of physical urticaria after contact with the wheat stalks.⁵⁰

In one study, 78% of individuals noticed that running induced their lesions.^{5,41,47} Runners develop both cutaneous and systemic symptoms in EIA, with pruritus being a regular finding.^{5,48} The reaction begins with sensation of cutaneous warmth, pruritus,

and erythema, and progresses subsequently to urticaria or angioedema. Some athletes experience gastrointestinal symptoms (nausea, diarrhea, colic) and headaches.^{32,40,48} Angioedema may involve the face, hands, feet, tongue, lips, eyelids, and oropharynx and may persist for up to 72 hours.^{4,40,41} Respiratory distress and vascular collapse may occur.^{5,19,36,37,40,48} The symptoms typically begin within the first 5 minutes of exercise but may begin after exercise is complete.^{39,41}

Runners should stop immediately if they suspect the onset of EIA.^{19,27,40,45,48} The acute treatment of EIA focuses on vascular support and airway patency.^{4,5,27,32,41,48} A long-acting, non-sedating antihistamine taken 1 hour before exercising may help in preventing or alleviating exercise-induced urticaria.^{19,36,37,41,46,48} If antihistamines are not effective, 40 mg of prednisone 12 hours before exercise may be helpful.^{19,36,37,46,47} Half of those with EIA can prevent it by avoiding exercise in extremely hot, humid, or cold weather, whereas one third can reduce attacks by not eating 4 to 6 hours before exercise.^{4,5,32,40,41,45} Avoidance of medications such as aspirin and nonsteroidal anti-inflammatory drugs also decreases attacks of EIA.^{5,40,41} Women may be predisposed around the time of menses or during pregnancy.^{40,47,49} Cromolyn has been used to prevent pulmonary symptoms and ketoprofen has been used to prevent dermatologic symptoms.^{5,32} Runners with EIA should be advised that they will not always experience symptoms, but carrying epinephrine and never running alone can prevent disastrous consequences.^{32,40,45}

TRAUMA

Jogger's toe

The nail plate of the second toe, the periungual areas of the third through fifth toes, and the entire hallux suffer significant trauma during running (Fig 2).^{5,8,18,51-53} Approximately 0.1% to 14% of runners reported injuries to the toenails on marathon day (Table I). Subungual hematoma and subungual hyperkeratosis develop because of repeated contact of the nail on the distal toe with the front of the toe box. Nail trauma is exacerbated with increased force as a result of downhill running.^{5,12,19,32,51,53,54} When hemorrhage occurs in the nail matrix, it is incorporated into the nail plate, whereas bleeding distal to the lunula is found in the nail bed.⁸ Onycholysis, thickening, and secondary fungal infection of the nail may occur with continual injury.³¹

Onychomycosis and subungual malignant melanoma should be in the differential diagnosis.^{4,5,41,51,53} Potassium hydroxide testing, culture, or period acid-Schiff staining of the subungual debris



Fig 2. Jogger's toe.

can differentiate onychomycosis from jogger's toe.^{4,5,12,41,51,53} The clinician should be suspicious of melanoma if discoloration of the nail is also seen on the periungual region (Hutchinson's sign), if there is a lateral extension of the pigment, or if there are different hues within the pigmented area.^{5,12,53,55} Suspicion for melanoma warrants a biopsy.^{4,12,51,53,55} Radiography may be necessary to rule out a fractured toe.^{18,19,56}

Time is the best treatment for subungual hematoma; however the patient should be advised that the toenail may remain black for several months.^{4,5,51,53,57,58} Evacuation of the blood through an incision with a scalpel, unguinal fenestration with punch biopsy, hot wire puncture, or Geiger cautery is traumatic and controversial, but may prevent spread of the hemorrhage and loss of the nail in acute injuries.^{8,20,54,59,60} Rest and soaking in warm water may be helpful for chronic lesions.^{15,19,54,58,59} Teams of collegiate runners have painted their nails dark colors for cosmetic purposes.^{5,57}

To prevent jogger's toe, lacing should be tight enough to keep the foot from sliding forward without restricting circulation, and the anterior toe box should be high and long enough to allow unrestricted dorsal flexion of the toes and minor forward slippage.^{19,54,57,59} In addition, nails should be cut straight and close to the skin.* Referral to a podiatrist may be necessary if over-the-counter methods are ineffective.⁵³

Talon noir

Talon noir, also known as calcaneal petechiae or black heel, is a condition characterized by discrete brown or blue-black macules.^{9,15,32,54,59,61,62} Talon noir relates to intraepidermal and ultimately intracorneal bleeding from lateral shearing forces of the epidermis sliding over the rete pegs of the papillary dermis thereby damaging the delicate papillary dermal capillaries.^{8,9,19,54,58,59,62} Repeated stop-and-start motions, changes in direction, and constant

*References^{4,5,19,51,53,54,58,59}



Fig 3. Large blister on the sole at a friction point.

pounding on hard surfaces cause injury of the heel against the back of the shoe.^{8,14,20,26} Young runners seem to be particularly vulnerable.^{8,62}

The macules of talon noir are composed of petechiae and are found on the posterior, medial, and lateral sides of the heel, just above the thick plantar skin at places where blood vessels are minimally protected by fatty tissue.^{15,19,26,34,54} The lesions may be confused with melanoma; however, paring the skin with a surgical blade removes the old hemorrhage.* Any suspicion for melanoma should prompt a biopsy.^{19,32} Runner's petechiae usually resolve on their own with 2 to 3 weeks of rest; skin lubrication, heel cups, a change in footwear, wearing two pairs of thick socks, and a hiatus from training may reduce the incidence of these lesions.^{8,9,19,26,51,60}

Runner's purpura

Purpura and petechiae have been noted in runners on the lower extremities and face during vigorous exercise in hot weather conditions.^{63,64} Erythematous urticarial or purpuric plaques on the lower legs that may be painful, paresthetic, or pruritic characterize exercise-induced purpura.⁶³ Purpura may extend to the thighs but usually spares the skin of the lower leg compressed by socks.⁶³ One case of ankle petechiae has been reported in an asymptomatic 29-year-old novice jogger.⁶⁵

An overloading of the thermoregulatory process with excessive venodilation and secondary failure of the fatigued calf muscle to pump this additional flow is thought to be the cause of these lesions.⁶³ There is no relation to chronic venous insufficiency or sun exposure.⁶³ Skin biopsy specimen may demonstrate leukocytoclastic vasculitis with C3 and IgM deposits.⁶³ Exercise-induced purpura spontaneously resolves in 3 to 10 days with rest and without residual hyperpigmentation.⁶³ Venous

compression, venoactive drugs, or topical steroids before running may prevent purpura and petechiae from forming.⁶³

Solar purpura typically occurs in skin in which surrounding connective tissue support of cutaneous blood vessels has been altered by age, actinic damage, or corticosteroids.⁶⁴ A 41-year-old male fighter pilot developed solar purpura on his malar prominences after a 6-mile run.⁶⁴ In this case, the exercise-induced displacement of blood from the central to peripheral circulation with resulting increased venous capacitance and transmural capillary pressure purportedly caused increased extravasation of blood cells.⁶⁴

Blisters

Blisters plague the running enthusiast. A review of the marathon literature showed that between 0.20% and 39% of runners reported to a medical tent at some point in their race as a result of blisters (Table I). Horizontal shearing forces cause epidermal splits in the mid to lower malpighian layer of the epidermis or through the lamina lucida at the dermoepidermal junction. These forces create blisters most frequently at the distal phalanges, under the metatarsals, and behind the calcaneus (Fig 3).^{9,14,18,19,66,67} The space created by the separated layers then fills with blood or tissue transudate.^{14,20,54,59} Risk factors for blister development include heat, moisture, overtraining, and ill-fitting shoes (either too tight or too loose).[†] Extensive blistering should raise the clinician's suspicion for epidermolysis bullosa.

Inappropriate treatment of blisters leads to delayed healing and increased time away from training.⁵ Correct technique for lancing blisters involves staying near the periphery and maintaining the blister roof.^{5,12,14,17,55,68} Undrained blisters create additional pressure on the peripheral fluid causing further separation and pain.⁵⁴ Furthermore, some authors showed that healing occurs fastest when the blister fluid is aspirated 3 times during the first 24 hours after occurrence.⁶⁹ Removal of the blister roof results in more discomfort and a greater propensity for developing secondary infections.^{20,54,67,68} Synthetic dressings adhere tightly to the affected area and provide adequate protection.^{5,8} The use of a small patch of hydrocolloid dressing often decreases pain and accelerates healing by promoting re-epithelialization.^{8,55,68}

Prevention of blisters requires a 3-pronged approach. First, the athlete may eliminate the mechanical aspect of friction by wearing two pairs of socks

*References^{8,19,26,32,48,55,60,62}

†References^{4,12,14,18,20,54,59,66,68}



Fig 4. Piezogenic pedal papules.



Fig 5. Jogger's nipples.

that are different materials, using neoprene insoles, buying appropriately fitting footwear, and applying petroleum jelly.[†] Second, athletes can decrease foot moisture by wearing dry synthetic moisture-wicking sports socks and applying antiperspirants to the feet.^{5,12,66} Finally, runners can promote the hardening of the skin with products such as 10% tannic acid soaks.^{12,26}

Piezogenic pedal papules

This entity was first described by Shelly and Rawnsley in 1968 with the term “piezogenic” to imply that the lesions were formed by pressure (piezo = pressure and genic = giving rise to).⁷⁰ One author suggests that as many as 10% to 20% of the population may be affected with both symptomatic and asymptomatic lesions.¹⁹ These papules represent herniations of subcutaneous fat through the collagen matrix of the reticular dermis.^{19,32,48,56} The papules, which may or may not be painful, are 2- to 5-mm skin- to yellow white-colored protuberances found on the medial or posterolateral heel of long-distance runners, particularly women and children (Fig 4).^{4,19} The pain can be significant enough to stop a runner's training.^{5,19,32,56} The pain and papules disappear after the feet are elevated for a few minutes.⁴⁸ Diagnosis is confirmed when the fat herniations are visualized while the athlete is standing and bearing weight on the affected foot.^{19,32,48,56} There is no satisfactory medical or surgical treatment, although heel cups have been reported to alleviate the symptoms.^{4,5,19,48,56,60}

Jogger's nipples and other forms of chafing

Chafing, a superficial inflammatory dermatitis caused by skin surfaces rubbing together, was reported by 0.4% to 16% of runners on marathon day (Table I). In one study of nonelite marathon runners, nearly one third of athletes used protection

with substances such as petroleum jelly to prevent chafing of areas including the nipples, feet, groin, and axillae.⁷¹

Jogger's nipples, a particular form of chafing, were reported by 2% to 16.3% of runners on marathon day (Table I). This cutaneous condition occurs in long-distance runners as a result of repetitive friction between runners' shirts and their nipples.^{4,5,18,26,31,55} Jogger's nipples occur not uncommonly in women who run without bras and in men who wear shirts made of coarse fibers.^{4,12,18-20} The lesions usually occur after long runs in cool conditions when the nipple is erect and the shirt is moist.^{27,31} Lesions present as painful, erythematous, crusted erosions of the areola and nipples, and may fissure with subsequent bleeding if they continue to be irritated (Fig 5).^{4,5,26,32}

Treatment for chafing consists of cleaning the affected areas gently with water, drying the areas thoroughly, and applying topical steroid ointment, if necessary, to alleviate inflammation.¹⁷ Jogger's nipples can be treated by applying petroleum jelly or antibiotic ointment such as erythromycin after the lesions occur.^{4,5,26,32}

Prevention of chafing is best accomplished by wearing dry, synthetic, well-fitting, moisture-wicking clothes.¹⁷ Talcum and alum powders are mildly helpful for drying, and petroleum jelly is effective for reducing friction.¹⁷ To prevent jogger's nipples specifically, friction can be reduced in both sexes by applying petroleum jelly, patches, or adhesive tape over the nipples.^{4,5,18,19,26,32} In addition, men can run without shirts, and women can wear semisynthetic bras.^{4,26} Men who run without shirts should be sure to apply sunscreen to prevent damage from UV light exposure.

Judo-jogger's itch

One case report discussed a jogger who developed pruritus the day after he had a vigorous judo workout.⁷² The itch started after sweating; began at the ankles and wrists with progression to the

[†]References^{12,14,26,34,56,59,67,68}

extremities, hips, and shoulders; and spared the head, torso, and genitalia.⁷² The author believed the pruritus may have been secondary to xerosis, which often affects runners in the winter when both the temperature and humidity are low, and the number of showers per week stays the same.^{18,73}

Runner's rump

Hyperpigmentation that results from small ecchymoses that occur on the superior portion of the gluteal cleft in long-distance runners characterizes runner's rump.^{19,54,58} The asymptomatic ecchymoses are produced by the continual contact of each buttock during each stride.^{54,58} Only certain runners are prone to develop this condition and, as such, it is assumed that a variation of running form contributes to its appearance.^{54,58} Runner's rump usually spontaneously resolves during periods of decreased training.^{54,58}

Jogger's alopecia

Linear, transverse, traction alopecia has been reported in a woman who jogged daily, wearing a tight-banded, wide-stripped, heavy headphone set.⁷⁴ Switching to a lighter headpiece stopped the hair loss.⁷⁴

Friction burns

Three cases have been reported in the literature of children who, while running on treadmills, fell and trapped their hands between the conveyer belt and the base of the metal bar.⁷⁵ As a result of these accidents, the children sustained full-thickness burns requiring escharotomy and split-thickness grafts.⁷⁵ Friction burns result from a combination of mechanical and thermal abrasion.⁷⁵ Safety devices are present on most treadmills today to prevent this type of accident from occurring.⁷⁵

ENVIRONMENTAL INJURIES

Frostnip

One jogger developed penile frostnip after running in subfreezing temperatures with severe winds while he was only wearing polyester trousers and cotton underwear with an anterior opening.⁷⁶ Frostnip is the most common superficial skin injury caused by cold weather.^{15,18-20,59} The affected areas initially feel numb, become mottled bluish-purple, and then swell, sting, and burn.^{18,59} Blisters may form in 24 to 36 hours (indicating frostbite) and resolve with crusting over 2 weeks.^{18,59} A throbbing or burning sensation may last for several additional weeks and the affected skin may remain sensitive to cold for many months.^{18,19} A combination of below-freezing temperatures and a windchill factor often induce frostnip, which most commonly affects the

skin over the nose, cheeks, chin, and ears.^{8,15,20,59} As blood flow to the skin diminishes, the skin blanches and thermal loss is not adequately replaced.^{54,59} Rapid rewarming in a water bath of 38°C to 44°C for 20 minutes is the treatment of choice.^{4,5,54}

To prevent frostnip, athletes should dress in multiple layers and wear sufficient clothing for the outside temperature.^{4,5,18,19} A layering effect of loose-fitting clothing allows air trapped between the layers to serve as an insulator.^{18,46,54,55,59} A clean, absorbent terry-cloth towel wrapped around the neck or a synthetic, breathable, commercially available neck garment should be used to protect this vulnerable region.^{46,54,59} The runner should be aware that the combination of exercise, cold weather, and wind decrease the clothing's ability to insulate by about 10% and, therefore, wet clothes should be changed as soon as possible.^{4,15,46,59} A delay in bathing and shaving until after the day's outdoor activities have been completed is beneficial, especially for the face, as the skin's sebum serves as a natural insulator.^{8,20,46,54} Application of lotions, creams, or ointments, especially a heavier oil-based preparation such as petroleum or zinc oxide, helps to serve as a barrier and seal in moisture and heat.^{20,46,54}

Skin cancer

The sun is also a concern in runners because of sunburn and skin cancer.^{20,59} Cumulative exposure to UV radiation and multiple childhood blistering sunburns increases the risk for squamous and basal cell carcinoma and melanoma.^{4,26,31,61} Relative risk studies have been done in other sports such as cycling, mountaineering, water sports, skiing, and triathloning to assess sun exposure, but no studies have elucidated the risk strictly from running.^{32,77-82} Three male triathletes at the 1999 Ironman World Championships in Hawaii accumulated more than 30 times the recommended limit of UV exposure in an 8- to 10-hour race, despite the use of water-resistant sun protection factor 25+ sunscreen.⁷⁷ The mean personal UV exposure was 8.3 times the athlete's minimal erythema dose during the competition.⁷⁷

If one does develop sunburn, the long-term damage cannot be changed, but symptomatic treatment is possible.⁵ For minor burns, cool water or Burow's compresses reduce skin heat and inflammation.^{20,31} More severe burns can result in vesicle and bullae formation and must be treated to prevent secondary infection.^{31,59} Warm soaks, petrolatum jelly, sarna lotion, gauze, topical corticosteroids in a cream or spray, and nonsteroidal anti-inflammatories such as aspirin and indomethacin may help soothe the phototoxic effects.^{5,20,31,32,59,61} Systemic

toxicity with fever, nausea, chills, and prostration is seen in severe cases of sunburn and may require aggressive supportive treatment.^{15,20,31,59}

Preventative treatment consists of protective clothing and topical sunscreen.²⁰ Ideally, runners should avoid peak sun exposure between 10 AM and 4 PM.^{5,15,20,32,55,61} Running hats exist and are readily available in running specialty stores. A large number of chemical sunscreens numbered with sun protection factors are available to consumers.²⁰ Runners should use a sunscreen that has a sun protection factor of at least 15, is sweat proof, and is broadband blocking.^{15,20,26,55} Sunscreens should also have superior substantivity, meaning they are effective even with moisture; however, even sunscreens with substantivity should be reapplied after sweating.^{5,8,32,48} Finally, runners should be sure to choose a sunscreen that is nonirritating to the eyes and skin during perspiration.^{32,48}

Conclusion

The skin disorders presenting in runners are diverse and range from benign and irritating to severe and life threatening. Dermatologists should remember the skin problems unique to runners so as to focus the differential diagnosis and treat and prevent these disorders in a timely manner.

REFERENCES

1. Caputo R, De Boule K, Del Rosso J, Nowicki R. Prevalence of superficial infections among sports-active individuals: results from the Achilles survey, a review of the literature. *J Eur Acad Dermatol Venereol* 2001;15:312-6.
2. Auger P, Marquis G, Joly J, Attye A. Epidemiology of tinea pedis in marathon runners: prevalence of occult athlete's foot. *Mycoses* 1993;36:35-41.
3. Lacroix C, Baspeyras M, de La Salmonière P, Benderdouche M, Couprie B, Accoceberry I, et al. Tinea pedis in European marathon runners. *J Eur Acad Dermatol Venereol* 2002;16:139-42.
4. Adams BB. Sports dermatology. *Dermatol Nurs* 2001;13:347-63.
5. Adams BB. Dermatologic disorders of the athlete. *Sports Med* 2002;32:309-21.
6. Brenner IKM, Shek PN, Shepard RJ. Infection in athletes. *Sports Med* 1994;17:86-107.
7. Hughes WT. The athlete: an immunocompromised host. *Adv Pediatr Infect Dis* 1998;13:79-99.
8. Kantor GR, Bergfeld WF. Common and uncommon dermatologic diseases related to sports activities. *Exerc Sport Sci Rev* 1988;16:215-53.
9. King MJ. Dermatologic problems in podiatric sports medicine. *Clin Podiatr Med Surg* 1997;14:511-24.
10. Sevier TL. Infectious diseases in athletes. *Sports Med* 1994;78:389-412.
11. Strong WB. The uniqueness of the young athlete: medical considerations. *Am J Sports Med* 1980;8:372-6.
12. Mailler EA, Adams BB. The wear and tear of 26.2: dermatological injuries reported on marathon day. *Br J Sports Med* 2004;38:498-501.
13. Adams BB. Transmission of cutaneous infections in athletes. *Br J Sports Med* 2000;34:413-4.
14. Bart B. Skin problems in athletics. *Minn Med* 1986;66:239-41.
15. Basler RSW. Skin lesions related to sports activity. *Prim Care* 1983;10:479-94.
16. Beck CK. Infectious diseases in sports. *Med Sci Sports Exerc* 2000;32(Suppl):S431-8.
17. Eiland G, Ridley D. Dermatologic problems in the athlete. *J Orthop Sports Phys Ther* 1996;23:388-402.
18. Levine N. Dermatologic aspects of sports medicine. *J Am Acad Dermatol* 1980;3:415-24.
19. Pharis DB, Teller C, Wolf JE. Cutaneous manifestations of sports participation. *J Am Acad Dermatol* 1997;36:448-59.
20. Atton AV, Tunnessen WW. The athlete and his skin. *Clin Rev Allergy* 1988;6:403-29.
21. Leyden JJ, Klingman AM. Aluminum chloride in the treatment of symptomatic athlete's foot. *Arch Dermatol* 1975;111:1004-10.
22. Bell-Syer SEM, Hart R, Crawford F, Torgerson DJ, Tyrrell W, Russel I. Oral treatments for fungal infections of the skin of the foot. *The Cochrane Database of Systematic Reviews*, 1999. doi: 10.1002/14651858.CD003584.
23. Bergfeld WF. Dermatologic problems in athletes. *Clin Sports Med* 1982;1:419-30.
24. Bergfeld WF. Dermatologic problems in athletes. *Prim Care* 1984;11:151-60.
25. Crawford F, Hart R, Bell-Syer SEM, Torgerson DJ, Young P, Russel I. Topical treatments for fungal infections of the skin and nails of the foot. *The Cochrane Database of Systematic Reviews*, 1999. doi: 10.1002/14651858.CD001434.
26. Adams BB. Sports dermatology. *Adolesc Med* 2001;12:305-22.
27. Mellman MF. Common medical problems in sports. *Clin Sports Med* 1997;16:635-62.
28. Aste N, Atzori L, Zucca M, Pau M, Biggio P. Gram-negative bacterial toe web infection: a survey of 123 cases from the district of Cagliari, Italy. *J Am Acad Dermatol* 2001;45:537-41.
29. Stauffer LW. Skin disorders in athletes: identification and management. *Phys Sportsmed* 1983;11:101-20.
30. Noffsinger J. Physical activity considerations in children and adolescents with viral infections. *Pediatr Ann* 1996;25:585-9.
31. Conklin RJ. Common cutaneous disorders in athletes. *Sports Med* 1990;9:100-19.
32. Adams BB. Skin and sports: common skin conditions in athletes and tips on treatments. *Skin and Aging* 2003;11:65-70.
33. Adams BB. Which skin infections are transmitted between athletes? *West J Med* 2001;174:352-3.
34. Bergfeld WF, Taylor JS. Trauma, sports, and the skin. *Am J Ind Med* 1985;8:403-13.
35. Fisher AA. Sports-related cutaneous reactions, part II: allergic contact dermatitis to sports equipment. *Cutis* 1999;63:202-4.
36. Scott MJ, editor. Sports dermatology. *Cutis* 1992;50(special issue):74-155.
37. Fisher AA. Sports-related allergic dermatitis. *Cutis* 1992;50:95-7.
38. de Leeuw J, den Hollander P. A patient with a contact allergy to jogging cream. *Contact Dermatitis* 1987;17:260-1.
39. Leshaw SW. Itching in active patients. *Phys Sportsmed* 1998;26:47-53.
40. Briner WW. Physical allergies and exercise. *Sports Med* 1993;15:365-73.
41. Adams BB. Exercise-induced anaphylaxis in a marathon runner. *Int J Dermatol* 2002;41:394-6.
42. Ting S, Reimann BEF, Rauls DO, Mansfield LE. Nonfamilial, vibration-induced angioedema. *J Allergy Clin Immunol* 1983;71:546-51.
43. Lawlor F, Black AK, Breathnach AS, Greaves MW. Vibratory angioedema: lesion induction, clinical features, laboratory

- and ultrastructural findings and response to therapy. *Br J Dermatol* 1989;120:93-9.
44. Patterson R, Mellies CJ, Blankenship ML, Pruzansky JJ. Vibratory angioedema: a hereditary type of physical hypersensitivity. *J Allergy Clin Immunol* 1972;50:174-86.
 45. Aunhachoke K, Rojanametin K, Saengapaswiriya A. Food-dependent, exercise-induced anaphylaxis: first case report in Thailand. *J Med Assoc Thai* 2002;85:1014-8.
 46. Fisher AA. Sports-related cutaneous reactions, part I: dermatoses due to physical agents. *Cutis* 1999;63:134-6.
 47. Shadick NA, Liang MH, Partridge AJ, Wright E, Fossel AH, Sheffer AL. The natural history of exercise-induced anaphylaxis: survey results from a 10-year follow-up study. *J Allergy Clin Immunol* 1999;104:123-7.
 48. Levine N. Dermatologic aspects of sports medicine. *Dermatol Nurs* 1994;6:179-86.
 49. Ii M, Sayama K, Tohyama M, Hashimoto K. A case of cold-dependent exercise-induced anaphylaxis. *Br J Dermatol* 2002;147:368-70.
 50. Swaine IL, Riding WD. Respiratory arrest in a male athlete after running through a wheat field. *Int J Sports Med* 2001;22:268-9.
 51. Adams BB. Jogger's toenail. *J Am Acad Dermatol* 2003;48 (Suppl):S58-9.
 52. Scher RK. Jogger's toe. *Int J Dermatol* 1978;17:719-20.
 53. Adams BB. Running-related toenail abnormality. *Phys Sportsmed* 1999;27:85-7.
 54. Basler RSW. Skin injuries in sports medicine. *J Am Acad Dermatol* 1989;21:1257-62.
 55. Helm TN, Bergfeld WF. Sports dermatology. *Clin Dermatol* 1998;16:159-65.
 56. Klein AW, Rish DC. Sports related skin problems. *Compr Ther* 1992;18:2-4.
 57. Adams BB. More on jogger's toe. *Phys Sportsmed* 2000;28:20.
 58. Fisher AA. Sports-related cutaneous reactions, part III: sports identification marks. *Cutis* 1999;63:256-8.
 59. Basler RSW. Sports-related skin injuries. *Adv Dermatol* 1989;4:29-50.
 60. Powell FC. Sports dermatology. *J Eur Acad Dermatol Venerol* 1994;3:1-15.
 61. Houston SD, Knox JM. Skin problems related to sports and recreational activities. *Cutis* 1977;19:487-91.
 62. Wilkinson DS. Black heel—a minor hazard of sport. *Cutis* 1977;20:393-6.
 63. Ramelet AA. Exercise-induced purpura. *Dermatology* 2004;208:293-6.
 64. Latenser BA, Hempstead RW. Exercise-associated solar purpura in an atypical location. *Cutis* 1985;35:365-6.
 65. Cohen HJ. Jogger's petechiae. *N Engl J Med* 1968;279:109.
 66. Herring KM, Richie DH. Friction blisters and sock fiber composition: a double-blind study. *J Am Podiatr Med Assoc* 1990;80:63-71.
 67. Levine N. Friction blisters. *Phys Sportsmed* 1982;10:84-92.
 68. Knapik JJ, Reynolds KL, Duplantis KL, Jones BH. Friction blisters: pathophysiology, prevention and treatment. *Sports Med* 1995;20:136-47.
 69. Cortese TA, Fukuyama K, Edstein WL. Treatment of friction blisters. *Arch Dermatol* 1968;97:717-21.
 70. Shelley WB, Rawnsley HM. Painful feet due to the herniation of fat. *JAMA* 1968;205:308-9.
 71. Hölmich P, Christensen SW, Darre E, Jahnsen F, Hartvig T. Non-elite marathon runners: health, training and injuries. *Br J Sports Med* 1989;23:177-8.
 72. Sullivan SN. Judo-jogger's itch. *N Engl J Med* 1979;300:866.
 73. Thorpe LW. Judo-jogger's itch. *N Engl J Med* 1979;301:112.
 74. Copperman SM. Two new causes of alopecia. *JAMA* 1984;252:3367.
 75. Attalla MF, Al-Baker AA, Al-Ekiabi SA. Friction burns of the hand caused by jogging machines: a potential hazard to children. *Burns* 1991;17:170-1.
 76. Hershkovitz M. Penile frostbite, an unforeseen hazard of jogging. *N Engl J Med* 1977;292:178.
 77. Moehrle M. Ultraviolet exposure in the Ironman triathlon. *Med Sci Sports Exerc* 2001;33:1385-6.
 78. Gentile DA, Auerbach PS. The sun and water sports. *Clin Sports Med* 1987;6:669-84.
 79. Moehrle M, Garbe C. Does mountaineering increase the incidence of cutaneous melanoma? A hypothesis based on cancer registry data. *Dermatology* 1999;199:201-3.
 80. Moehrle M, Heinrich L, Schmid A, Garbe C. Extreme UV exposure of professional cyclists. *Dermatology* 2000;201:44-5.
 81. Moehrle M, Dennenmoser B, Garbe C. Continuous long-term monitoring of UV radiation in professional mountain guides reveals extremely high exposure. *Int J Cancer* 2003;103:775-8.
 82. Rigel EG, Lebwohl MG, Rigel AC, Rigel DS. Ultraviolet radiation in alpine skiing: magnitude of exposure and importance of regular protection. *Arch Dermatol* 2003;139:60-2.
 83. Richards R, Richards D, Schofield PJ, Sutton JR. Reducing the hazards in Sydney's the Sun City-to-Surf runs, 1971 to 1979. *Med J Aust* 1979;2:453-7.
 84. Orava S. About the strains caused by a marathon race to fitness joggers. *J Sports Med Phys Fitness* 1977;17:49-57.
 85. Nequin ND. More on jogger's ailments. *N Engl J Med* 1978;298:405.
 86. Caldwell J. Experience from the 1980 Midnight Sun marathon: injuries and training. *Alaska Med* 1981;23:18-21.
 87. Duras P, Russell JW, Kretsch A, Grogan R. Illness and injury during the 1980 Big M Melbourne marathon. *Aust J Sports Med Exerc Sci* 1983;15:35-9.
 88. Kretsch A, Grogan R, Duras P, Allen F, Sumner J, Gillam I. 1980 Melbourne marathon study. *Med J Aust* 1984;141:809-14.
 89. Temple C. Hazards of jogging and marathon running. *Br J Hosp Med* 1983;29:237-9.
 90. Cerio R, Moody A. The London marathon: 3 years in the running. *Arch Emerg Med* 1985;2:89-91.
 91. Nicholl JP, Williams BT. Injuries sustained by runners during a popular marathon. *Br J Sports Med* 1983;17:10-5.
 92. Nicholl JP, Williams BT. Medical problems before and after a popular marathon. *Br Med J (Clin Res Ed)* 1982;285:1465-6.
 93. Nicholl JP, Williams BT. Popular marathons: forecasting casualties. *Br Med J (Clin Res Ed)* 1982;285:1464-5.
 94. Roberts WO. A 12-year profile of medical injury and illness for the Twin Cities marathon. *Med Sci Sports Exerc* 2000;32:1549-55.
 95. Ridley SA, Rogers PN, Wright IH. Glasgow marathons 1982-1987: a review of medical problems. *Scott Med J* 1990;35:9-11.
 96. Adner MM, Scarale J, Casey J. The Boston marathon medical care team: ten years of experience. *Phys Sportsmed* 1988;16:99-106.
 97. Hölmich P, Darre E, Jahnsen F, Hartvig-Jensen T. The elite marathon runner: problems during and after competition. *Br J Sports Med* 1988;22:19-21.
 98. Darre E, Hölmich P, Jahnsen F, Jensen TH. Medical service and registration of injuries in the 1986 Wonderful Copenhagen marathon. *Ugeskr Laeger* 1987;149:811-3.
 99. Jakobsen BW, Kroner K, Schmidt SA, Jensen J. Running injuries sustained in a marathon race: registration of the

- occurrence and types of injuries in the 1986 Aarhus marathon. *Ugeskr Laeger* 1989;151:2189-92.
100. Crouse B, Beattie K. Marathon medical services: strategies to reduce runner morbidity. *Med Sci Sports Exerc* 1996;28:1093-6.
 101. Satterthwaite P, Larmer P, Gardiner J, Norton R. Incidence of injuries and other health problems in the Auckland Citibank marathon, 1993. *Br J Sports Med* 1996;30:324-6.
 102. Satterthwaite P, Norton R, Larmer R, Robinson E. Risk factors for injuries and other health problems sustained in a marathon. *Br J Sports Med* 1999;33:22-6.
 103. Caselli MA, Longobardi SJ. Lower extremity injuries at the New York City marathon. *J Am Podiatr Med Assoc* 1997;87:34-7.
 104. Sainsbury R. Medical experience of the Great North Run Fordham. *Br J Sports Med* 1984;18:265.