Wilderness dermatology: mountain exposures

Samantha Schneider MD[1], Cecilia Blair Levandowski, BS,[2] Cory Manly MD, [3] Robert Dellavalle MD, PhD, MSPH,[4] and Cory A. Dunnick MD[4]

Affiliations: [1]Department of Dermatology, Henry Ford Hospital, Detroit, Michigan, [2]University of Colorado Denver, Denver, Colorado, [3]Department of Internal Medicine, Walter Reed National Military Medical Center, Bethesda, Maryland, [4]Department of Dermatology, University of Colorado School of Medicine, Aurora, Colorado

Corresponding Author: Cory A. Dunnick MD, University of Colorado, Department of Dermatology, 1665 Aurora Ct, 3rd Floor, Anschutz Cancer Pavilion, Aurora, CO 80045, Email: cory.dunnick@ucdenver.edu

Abstract

Exploring the mountains is a highly rewarding past time; however, certain high-altitude exposures can lead to dermatologic manifestations. In this review article, the authors will describe cold, solar, and severe weather that one may experience when spending time outdoors. Factors such as increased ultraviolet radiation, temperature extremes, and low partial pressure of oxygen, along with human physiologic parameters also contribute to disease severity and presentation. This review article will address the diagnosis, treatment, and prevention of high-altitude dermatology exposures.

Keywords: frostbite, chilblains, frostnip, cold urticaria, lightning, cold panniculitis, actinic prurigo, prolonged exposure dermatosis, high altitude, dermatology

Introduction

When engaging in mountainous, high altitude activities, there are different exposures that can lead to dermatologic manifestations. Engaging in outdoor activities at higher altitude implies colder weather exposures as air temperature decreases approximately 2°C for every increase in 310m (1000 feet), (**Figure 1**), [1]. Some of the more common culprits at high altitude that can lead to dermatologic – among other – consequences include low humidity, high-velocity wind, excessive ultraviolet (UV) light exposure, and extreme cold temperatures [1, 2]. In this review article, we will be examining dermatologic injuries due to cold, solar, and severe weather exposures including their diagnosis, treatment, and prevention.

Body of Article

Cold Exposures

There are several skin pathologies related to cold exposure that will be described in the following sections (see **Table 1**).

Frostbite

Frostbite is an important cold exposure, particularly when traveling at altitude as the risk of frostbite increases significantly above approximately 5182m [3]. Hypothermia and frostbite explain 3-5% of all injuries in mountaineers and about 20% of injuries in Nordic skiers [4]. When examining the injuries to climbers on Denali, McIntosh et al. found that the most common injury was frostbite (18.1%), [5]. Frostbite occurs when the temperature in the skin and deeper tissues reaches sub-freezing (0°C) temperatures [1, 3, 4, 6]. At sub-zero temperatures, icicles develop in the tissues ultimately causing cellular injury and death [2, 7-9]. In milder temperatures, frostbite can take time to develop; however, when the wind chill is around -27°C or colder, frostbite can form in as little as 30 minutes and sports such as running and skiing can increase the wind chill significantly [1]. There are four overlapping phases in the pathogenesis of frostbite, which will be reviewed in **Table 2** [6, 8, 10].

Frostbite most commonly occurs on exposed skin (i.e. hands, fingers, pinna of the ear, tip of the nose, and feet), [2-4, 6, 7]. Important environmental risk factors for the development of frostbite include high humidity, low temperatures, wind chill, high altitude, and prolonged exposure to the elements. Other risk factors include wearing tight constrictive clothing, immobility, nicotine use, medical diseases (such as

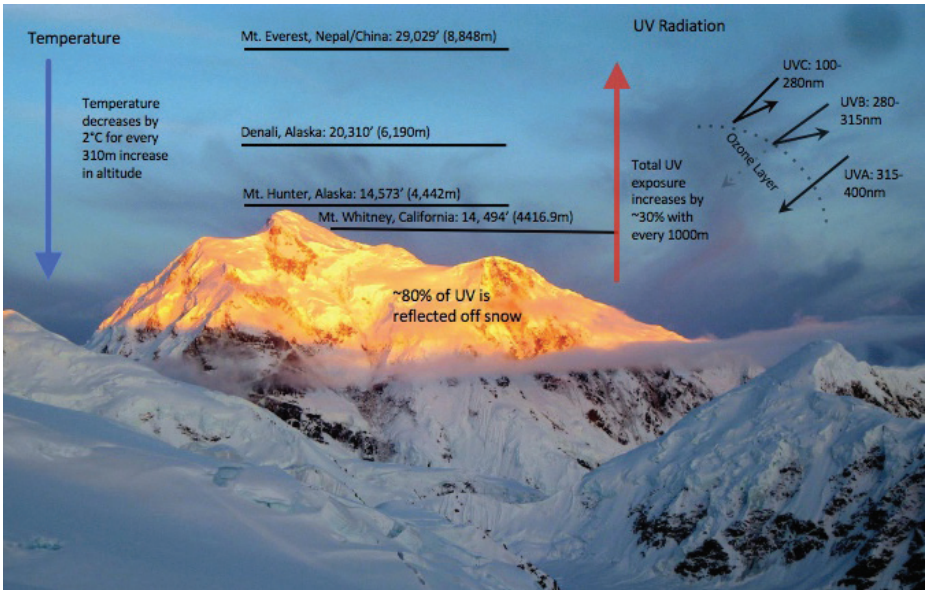

Figure 1. *High Altitude Effects of Temperature and UV Radiation. Mount Hunter (14,573' or 4,442m) is the beautiful mountain featured in this image. It is located near Denali in Alaska. Mount Whitney (14,494' or 4416.9m) has been included as a reference for the highest peak in the contiguous United States. Additionally, Denali is the highest peak in the United States at 20,310' or 6,190m and Mt. Everest is the highest peak in the world at 29,029' or 8.848m. As one ascends higher in altitude, the temperature drops by 2C for every 310m increase in altitude, which makes the peak of Everest about 28.6C colder than Mt. Whitney. With respect to ultraviolet radiation, there are three main types of UVR including UVA (315-400nm), UVB (280-315nm) and UVC (100-280nm). The ozone layer filters out all UVC and approximately 80-90% of UVB. Most UVA penetrates the ozone layer to reach the earth's surface. As one ventures higher in altitude, the total UV exposure (UVA and UVB) increases by as much as 30% for every 1000m. Someone on the summit of Mt. Everest would receive about 133% more UVR exposure compared to someone on the summit of Mt. Whitney. Aside from simply the altitude, the landscape can greatly increase the amount of reflected UVR. About 80% of UVR is reflected off snow and UV also reflects off certain rocks and sand. Protecting oneself from the varied elements at altitude is of utmost importance.*

peripheral vascular disease, diabetes mellitus, or vasoconstrictive disorders such as Raynaud disease), and use of vasoconstrictor medications (such as clonidine), [3, 7, 8, 11]. Women also tend to be more commonly affected than men [3].

The risk of injury related to frostbite is closely related to one's preparedness, any preexisting medical conditions, the body's physiologic response to the environment (notably impaired thermoregulation and increased heat loss), and any previous cold injury [4]. When preparing to spend time out in the elements at altitude, one should ensure good hydration and nutrition, dress in non-restrictive layers to cover all skin and scalp, optimize prior medical conditions, avoid wet extremities, and use chemical warmers [4, 5, 7, 8, 10, 11]. There are two classifications schemes to describe the severity of the frostbite: the classic four degrees of frostbite classification and the field classification system (**Table 3**). Patients' clinical presentations vary greatly across the spectrum from superficial to deep frostbite. With first-degree frostbite, patients have mild edema with white-yellow slightly raised firm plaques (**Figure 2**A). Second-degree frostbite consists of clear or milky fluid-filled vesicles surrounded by erythema and edema (**Figure 2**B). In third-degree frostbite, patients have deeper hemorrhagic blisters as well as blue-grey skin discoloration (**Figure 2**C), which suggests that the injury has extended into the reticular dermis beneath the dermal vascular plexus. Fourth-degree frostbite represents full-thickness damage that affects the skin as well as muscles, tendons, and bone (**Figure 2**D). Ultimately, these patients experience significant tissue loss commonly requiring surgical intervention including amputation.

Once a patient has confirmed frostbite, it is critical that they seek shelter from the elements and receive medical attention. In the field, treatment consists primarily of rapid re-warming via a water bath of 40-42°C. However, rapid re-warming should only be attempted if the tissue is not at risk of refreezing [10]. Repeated cycles of thawing and refreezing greatly increase the extent of the injury [7, 8, 9, 12]. If, in the field, a water bath is unavailable, one can warm the affected area with body heat from an adjacent person [10]. Upon initial re-warming, patients may have significant numbness followed by a burning, aching sharp pain [3]. In the field, the wound should be dressed and protected from the elements. Additionally, the patient should receive pain control and be transferred to a medical facility for more advanced therapies [7, 9, 10, 13].

	Characteristic	Symptom	Prognosis
Frostbite	Freezing injury whereby intra-epidermal icicles facilitate cellular damage and death	Ranges in severity from erythema to vesicles to eschar formation	Ranges from reversible damage to requiring surgical intervention
Frostnip	Non-freezing injury associated with severe vasoconstriction	Icicle formation on skin's surface causing temporary numbness and pallor	No long term consequences
Trench Foot	Non-freezing injury to feet ranging from erythema to bullae and gangrene	Paresthesias and numbness with edema, bullae, and possible gangrene	Worse, if gangrene
Chilblains (Perniosis)	Non-freezing injury characterized by painful or pruritic erythematous papules	Painful or pruritic erythematous papules	Resolves over several weeks
Cold Panniculitis	Non-freezing injury of tender erythematous areas of induration	Tender erythematous induration	Resolves over several weeks
Raynaud's	Vasoconstrictive disorder	Skin discoloration ranging from white to blue (cyanosis) to red (rewarming)	Long-term disease. Prevention can avoid future attacks
Cold Urticaria	Physical urticaria in setting of cold	Erythematous pruritic hives with or without angioedema	Patients may need anti-histamines or rarely epinephrine autoinjector (for anaphylaxis)

Table 1. *Cold Exposures. When traveling at altitude, there is increased exposure to cold weather. As such, patients may develop several different cold induced dermatologic conditions ranging from frost bite to chilblains to cold urticaria. Most these diseases do not have long term consequences; however, frostbite may have a poor prognosis at times even requiring amputation.*

At a medical center, patients can receive additional treatments such as tissue plasminogen activator (TPA) or prostaglandins [8, 10, 12]. Several studies have demonstrated that giving TPA within 24 hours of re-warming greatly decreases the risk of amputation [10, 14]. An additional medical therapy includes iloprost, which is a prostaglandin E1 analogue. Iloprost has been shown to decrease the rate of digital amputation in severe cases of frostbite [5, 10, 13, 15]. However, iloprost is not currently available in the United States. Tissue viability can be determined rapidly via MRA or bone scintigraphy to help with prognosis and possible plans for surgical intervention. After the tissue has thawed, additional therapies include hydrotherapy to increase circulation, remove superficial bacteria, and debride devitalized tissue and hyperbaric oxygen [7, 10]. Following recovery from frostbite, patients may have long-term sequelae including decreased sensation, cold sensitivity, hypopigmentation, hyperhidrosis, and autonomic dysfunction [8].

Frostnip

Frostnip is a non-freezing injury that is associated with severe vasoconstriction. It is commonly thought to be a precursor to frostbite as it occurs in a similar distribution (cheeks, ear, nose). Icicles form on the skin's surface giving the appearance of frost. However, there are no icicles within the skin and thus no long-term consequences of frostnip. Patients present with numbness and pallor on exposed skin that resolves quickly following rewarming [10].

Trench Foot

Trench foot (i.e. immersion foot) is another non-freezing cold injury. Trench foot occurs when the feet are exposed to cold (temperatures >0°C) and wet environments for prolonged periods of time. Initially, with stage one, patients develop erythema, edema, and tenderness. Over the next day or two, patients develop stage two, which consists of paresthesias and numbness with marked edema and occasional bullae. Lastly, patients can progress to stage three, which consists of gangrene [3, 4, 7, 8]. It is recommended that those at risk for or with a history of trench foot change their shoes and socks frequently in cold, wet conditions. Additionally, one may try prophylactic treatment with anti-perspirants that include aluminum hydroxide to decrease perspiration on the feet [3].

Chilblains

Chilblains (i.e. perniosis) is another non-freezing cold injury. It is defined as a superficial skin injury that, like trench foot, occurs in cold wet environments above freezing [3, 4, 7, 8, 12, 16]. After being in the

cold wet conditions for about 1.5 hours, patients susceptible to chilblains develop painful or pruritic erythematous papules that resolve in several weeks [2-4, 7, 12, 16]. The most commonly affected locations include tips of toes, fingers, nose, and the ear's pinna [2, 7, 16]. There are two types of chilblains: primary and secondary. Primary is idiopathic chilblains, whereas secondary chilblains occurs as a result of another medical condition such as myelocytic leukemia, cold agglutinins, cryoglobulinemia, anorexia nervosa, or systemic lupus erythematosis [16]. A study examined the prevalence of chilblains in Ladakh - a high altitude region of India. The authors found that about 5.75% of dermatology clinic visits during the study period were for chilblains and interestingly only a single case occurred in a local resident, which was secondary to systemic lupus erythematosus. This study suggested that there may be a protective benefit for those living locally at high altitude. The pathogenesis is still being elucidated but it is thought to arise from an abnormal vascular response to cold environments [16]. Aside from environmental conditions, other risk factors include patients with a low body mass index, genetic predisposition, inadequate clothing, inadequate shelter, dehydration, fatigue, and previous cold weather injuries [2, 16]. Once a patient develops chilblains, the treatment is primarily symptomatic including rest, warmth, and topical anti-pruritic agents. Patients can try to prevent chilblains by using emollients and wearing layers of clothing. Fortunately, there are no lasting effects [3, 12, 16].

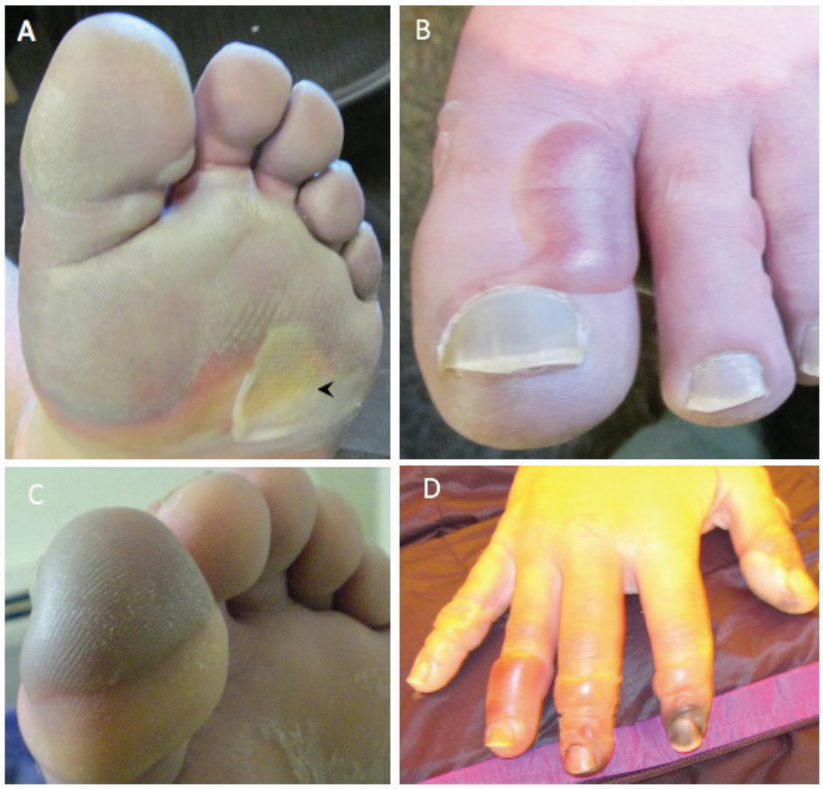

Figure 2. *Clinical Frostbite. Patient's clinical presentations vary greatly across the spectrum from superficial to deep frostbite. A) First degree or superficial frostbite is illustrated clinically with a yellow plaque on the patient's dorsal foot (identified with an arrowhead). The other areas of this patient's foot likely represent at least third degree or deep frostbite given the blue-grey discoloration. B) Second-degree frostbite consists of clear or milky fluid-filled vesicles surrounded by erythema and edema. C) This patient most likely had third degree or deep frostbite given the blue-grey skin discoloration. D) Fourth-degree frostbite represents full-thickness damage that affects the skin as well as muscles, tendons, and bone, which is best evaluated after rewarming. Given the eschar formation on this patient's index finger it may present full-thickness damage; however, it could also represent third degree frostbite.*

Cold Panniculitis

Cold panniculitis is the last non-freezing cold weather injury. Patients develop tender erythematous areas of induration upon exposure to cold [7]. Typically, the lesions appear within a few days of cold exposure and resolve spontaneously over the next several weeks. Cold panniculitis tends to be more common in children. As with cold urticaria (see below), the ice cube challenge test can help diagnose susceptible individuals. Those with a positive test will develop an erythematous plaque about 12-18 hours after exposure to the ice cube [7].

Raynaud Disease

Raynaud disease is a vasoconstrictive disorder primarily found in women. There are two types of Raynaud: primary and secondary. Primary Raynaud (also known as Raynaud disease) affects young women and is not associated with any other medical conditions. It is common, affecting 3-5% of the population. Secondary Raynaud, on the other hand, is known as Raynaud phenomenon and is secondary to an underlying disease such as systemic sclerosis (most common), systemic lupus erythematosus, dermatomyositis, cryoglobulinemia, and other diseases [17]. In both cases, cold is a major precipitant of an attack.

	Stage of Frostbite Pathogenesis	Characteristics
A	Pre-Freeze Phase	Vasoconstriction leads to mild ischemia
B	Freeze-Thaw Cycle	Ice crystal formation induces cell membrane lysis and apoptosis
C	Vascular Stasis	Vessels constrict and dilate ("Hunting" reaction) causing both vascular leakage and stasis
D	Late Ischemic	Inflammatory mediators (i.e. thromboxane A2, prostaglandins, bradykinins, histamine) destroy microcirculation leading to cell death

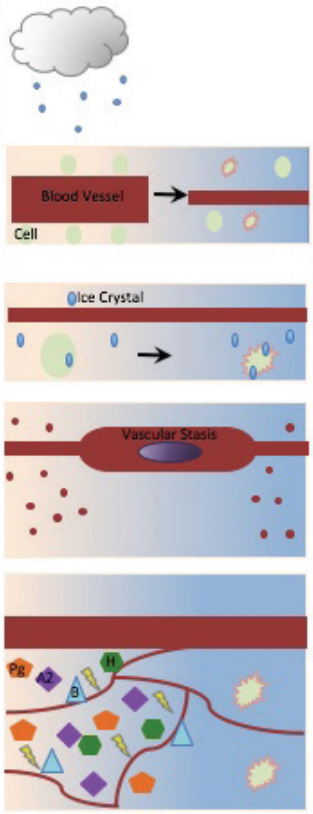

Table 2. *Pathogenesis of Frostbite. There are four overlapping phases in the pathogenesis of frostbite. A. The pre-freeze phase is where the tissue decreases in temperature causing vasoconstriction and ischemia, which manifests as hyperesthesia and paresthesia. This is visualized in the panel A whereby vasoconstriction causes ischemia to some adjacent cells. B. During the freeze-thaw cycle, ice crystals form either extracellularly with slower freezing or intracellularly with rapid injury. These icicles lead to electrolyte and cellular fluid shifts ultimately causing cell membrane lysis and apoptosis. C. Vascular stasis, stage three, consists of blood vessel oscillations between constriction and dilatation, which is also known as the hunting reaction. These oscillations result in both leaky blood vessels and coagulation. D. The last phase is termed late ischemic, which initiates a cellular cascade that increases inflammation by release of thromboxane A2, prostaglandin F2-alpha, bradykinins and histamine. These inflammatory mediators play a role in destruction of the microcirculation, which similarly leads to cellular death.*

In the setting of a cold environment, patients with Raynaud conditions have an uncharacteristically strong vasoconstrictive reaction, which greatly decreases blood flow to the fingers. As such, patients develop skin discoloration that starts white, then turns blue (if cyanosis develops) and resolves with a red discoloration upon rewarming [3, 17]. These skin changes are associated with tingling, swelling, and painful throbbing [3]. Prevention is key in avoiding physical manifestations of Raynaud conditions, particularly by wearing warm clothing such as gloves.

Cold Urticaria

Cold urticaria is a type of physical urticaria that results from exposure to cold environments. It is thought to be a type of allergic response to cold that results in erythematous pruritic hives with or without angioedema within minutes of exposure to cold environments (**Figure 3**), [3, 4, 8, 18, 19, 20]. The most common stimuli include aquatic activities, cold environments, ingestion of cold foods or liquids, and even handling cold objects [19]. As with chilblains, there are both primary and secondary types of cold urticaria. Most patients have primary or essential cold urticaria (>90%), [3, 7, 18, 20]. Other secondary (acquired) causes include cryoglobulinemia, cold agglutinins, or paroxysmal hemoglobinuria [7, 18-20]. A third, rare, type of cold urticaria is called delayed cold-induced urticaria, which occurs 9-18 hours after a cold exposure [7, 18]. Finally, there is a rare familial cold urticaria where patients develop a non-urticarial rash, fever, arthralgias, and conjunctivitis within a few hours of cold exposure [3, 7, 18-20]. Aside from the skin manifestations, patients can also have systemic symptoms including fatigue, headache, dyspnea, and tachycardia [7, 19, 20]. Rarely, patients even experience anaphylaxis [4, 19, 20].

Cold urticaria affects men and women equally with most patients presenting in young adulthood (second or third decade of life). It can be diagnosed by the ice cube challenge test [3, 7, 8, 18-20]. In this diagnostic test, the professional applies an ice cube to the skin for around 10-20 minutes. The test is positive if a wheal appears in that period of time.

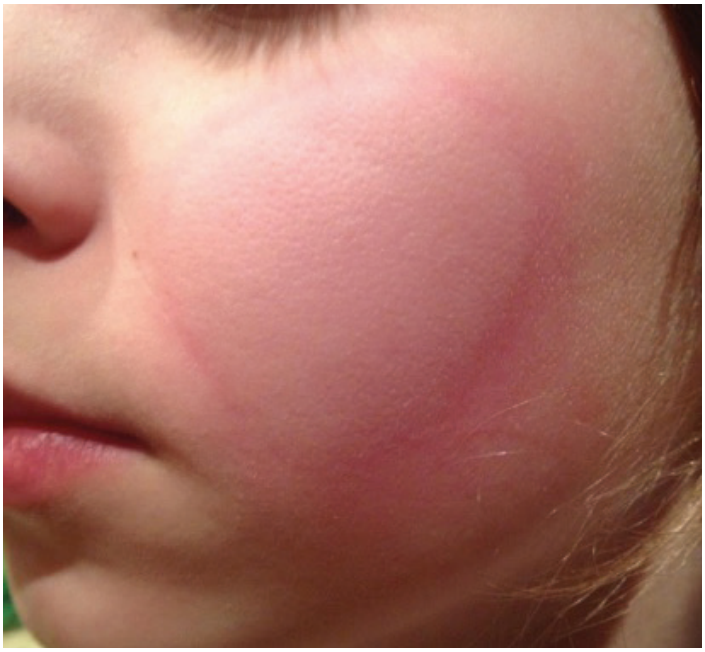

Figure 3. *Cold Urticaria. Cold urticaria is a physical urticaria that develops upon exposure to cold environments. Patients develop develop an urticarial eruption as demonstrated by the large wheal present on this patient's left cheek. Some patients may develop systemic symptoms such as bronchospasm, malaise, or fatigue, which may necessitate the use of an epinephrine autoinjector. Most patients respond well to anti-histamines prior to cold exposures.*

However, approximately 20% of patients with cold urticaria have a negative ice cube challenge test. Patients with cold urticaria can be prophylactically treated with antihistamine medications. More potent antihistamines such as cyproheptadine, doxepin, ketotifen, or cimetidine tend to have better success [4, 7, 8, 18-20]. Additionally, patients with severe cold urticaria should have an epinephrine auto-injector in the case of, albeit rare, anaphylaxis.

Solar Exposures

Solar radiation is made up of three different components: approximately 50% visible light, 40% infrared light, and 9% ultraviolet radiation [21]. Ultraviolet radiation (UVR) is the damaging culprit when discussing solar dermatologic exposures. There are three types of UVR light classified by the wavelengths that they emit: UVA (320-400nm), UVB (280-315nm), and UVC (200-290nm), [2, 21, 22]. The most important types of UVR with respect to human exposures are UVA and UVB, as UVC is filtered by the ozone layer [21]. Most of the UVR that reaches the earth's surface is in the form of UVA, which damages the skin through oxidative stress. Although only about 5% of UVB reaches the earth's surface, it is the most damaging type of UVR, as DNA is its major target (**Figure 1**), [21]. UVB exposure ultimately causes cyclobutane pyrmidine dimers and pyrimidone (6-4) pyrimidone photoproducts, which leads to errors in DNA repair creating the potential for oncogenesis [21, 23, 24]. Solar exposures can lead to several dermatologic manifestations (**Table 4**).

Skin Cancer

Skin cancer is the most common form of cancer in the United States with approximately 2 million new diagnoses each year [6, 25]. There are many great reviews on both melanoma and non-melanoma skin cancer - for more detailed information see Tripp et al. [26], Mulliken et al. [27], and Kallini et al. [28]. Sunburns from UVR exposure tend to be the precursor to all skin cancers. A study of 283 male mountain guides in Germany, Switzerland, and Austria found that mountain guides were at greater risk of developing both pre-malignant lesions as well as skin cancer, demonstrating an association between high occupational UVR exposure and increased prevalence of both precancerous skin lesions and skin cancer. A second study of 62 mountain and ski guides in Europe found similar results with 43.5% of participants developing NMSC or pre-cursor lesions [29]. In all types of skin cancer, the most modifiable risk factor is UVR from sun exposure. Certain environmental factors can increase the amount of UVR including simply being at higher altitudes, as well as clear skies, and reflective surfaces including snow, sand, and water [30]. Additionally, it has been estimated that the daily total of UVR increases by ~32% (UVB increases by 17-22% and UVA by 11%) for every 1000m above sea level during the winter [30]. A study of Alpine skiers and snowboarders in the western United States in the late 1990s found that skin cancer was positively associated with chair-lift elevation, sun sensitivity, and prior sunburn while skiing [30].

Actinic Prurigo

Actinic prurigo is a sunlight-induced pruritic eruption that can occur in all races, though it is particularly common in Native Americans. Other risk factors include living at higher altitudes. It often develops in childhood with good resolution, but in some cases, it can persist into adulthood. Girls tend to be more

Four Degrees of Frostbite			Field Classification System	
	Skin Findings	Symptoms		Characteristics
First Degree	Mild edema with white-yellow slightly raised firm plaques	Reversible erythema and numbness	Superficial Frostbite	No or minimal anticipated tissue loss
Second Degree	Clear or milky fluid filled vesicles with surrounding erythema & edema	Eschar formation		
Third Degree	Hemorrhagic blisters with blue-gray skin discoloration	Deep burning pain upon rewarming Thick eschar formation	Deep Frostbite	Tissue loss is anticipated
Fourth Degree	Full-thickness damage	Commonly need surgical intervention		

Table 3. *Frostbite Classification. There are two classifications schemes to describe the severity of the frostbite: the classic four degrees of frostbite classification and the field classification system. The four degrees of frostbite classification system is based on acute physical findings after warming as well as imaging [2, 7, 11, 13, 14]. The four degrees of frostbite classification system does not directly dictate immediate treatment - as treatment is rapid rewarming in all patients - or prognosis – as prognosis depends upon tissue demarcation after 3-4 weeks of tissue healing. As such, a second classification scheme was developed for simpler stratification of patients in the field [7, 10, 11, 14]. With superficial frostbite, patients have no or minimal anticipated tissue loss. This is thought to correlate with first or second-degree frostbite. And, with deep frostbite, patients have anticipated tissue loss due to their injury, which correlates with third and fourth-degree frostbite.*

affected than boys. Actinic prurigo is described as intensely pruritic, crusted papules that arise in sun-exposed areas particularly on the face and distal limbs. It is thought to possibly be a persistent variant of polymorphous light eruption [31]. Photoprotection can be helpful in preventing a flare. Treatment is with topical corticosteroids, topical tacrolimus, narrow-band UVB, PUVA and resistant disease should be treated with oral thalidomide [31].

Seborrheic Dermatitis

Seborrheic dermatitis is a mild chronic inflammatory skin condition that affects areas of the skin with large sebum production including the scalp, ears, face, central chest, and intertriginous areas [32]. The pathogenesis of seborrheic dermatitis is still under research. However, increased sebum production, in conjunction with the yeast Malassezia furfur (Pityrosporum ovale) have both been implicated. There are two main types of seborrheic dermatitis: infantile and adult. For the purposes of this review, we will focus on the adult type.

Adult seborrheic dermatitis is thought to be the most common type of eczema affecting 2-5% of the population [32, 33]. It typically develops around the fourth to seventh decade of life and there is a higher incidence in men. Clinically, seborrheic dermatitis is described as sharply demarcated pink-yellow to red-brown patches with flaky, greasy scales in a seborrheic distribution[32].

UVR is known to aggravate existing seborrheic dermatitis [31]. As such, seborrheic dermatitis occurred in 8% of patients receiving psoralen-UVA treatment, which was thought to be secondary to UV-induced immunosuppression [33]. Additionally, a study in the late 1990s, examined the prevalence of seborrheic dermatitis in mountain guides in Austria, Switzerland, and Germany [33]. It was found that 16.3% of mountain guides in the study had evidence of dermatologist diagnosed seborrheic dermatitis with equivalent incidence in all three countries. As with the increased incidence of seborrheic dermatitis with PUVA, the increased incidence in mountain guides is thought to be related to UV-induced immunosuppression given their chronic occupational exposure. Other possibilities include poor hygiene and the fact that sunscreens may have altered their facial lipid compositions.

Severe Weather Exposures

Lightning

Lightning strikes remain the second most common cause of deaths related to storms and inclement

		Characteristics	Symptoms	Prognosis
Skin Cancer	Melanoma	Pigmented skin cancer	May be asymptomatic	Improves with earlier diagnosis and treatment
	NMSC	Commonly pink to red exophytic growths with or without bleeding	May be asymptomatic or pruritic	Generally good prognosis for BCC and improved prognosis for SCC with early diagnosis and treatment
Polymorphous Light Eruption		Most common photodermatosis	Varies phenotypically but generally consists of pruritic, erythematous papules on sun-exposed areas	May have recurrent disease. Treatment is primarily preventative.
Actinic Prurigo		Sunlight induced pruritic eruption	Pruritic, crusted papules in sun-exposed areas	Often resolves before adulthood. Photoprotection is useful.
Solar Urticaria		Physical urticaria in response to UVR or visible light exposure	Transient pruritic wheals in sun-exposed distribution	Resolves over several hours after sun exposure. Patients may need treatment with anti-histamines.
Seborrheic Dermatitis		Mild chronic inflammatory skin condition that may worsen with sun exposure	Sharply demarcated pink-yellow to red-brown patches with flaky, greasy scales	May persist but often responds to treatments

Table 4. *Solar Exposures. At altitude, there is increased exposure to ultraviolet radiation putting outdoor enthusiasts at risk for multiple solar exposures. These range from an increased incidence of skin cancer (as identified in studies of mountaineers) to polymorphous light eruption to solar urticaria. As with cold exposures, most these diseases do not have long term consequences; however, skin cancer can be a very serious and potentially devastating diagnosis.*

weather in the United States (after flash flooding), [34]. Lightning strikes affect as many as 400 people annually in the United States resulting in approximately 40 deaths [34, 35]. Those most commonly affected are males (>80%) between the ages of 20-45 [35].

Lightning can be both positively or negatively charged as well as have direct or alternating current [35]. A single bolt of lightning contains as much as 30,000-110,000A [36]. However, the energy is only applied for a few milliseconds creating little opportunity for transfer to the body [35]. There are several different types of lightning strikes: direct strike, contact injury (when lightning strikes an adjacent object that is touching the patient), side splash (when the current jumps from a nearby object to the patient), and ground current (when the lightning travels through the ground to strike the patient), [35, 36]. Ground current is the most common mechanism of a lightning strike.

Prevention is key when it comes to lightning exposure. Examining weather patterns is critical. Signs that a storm is approaching include cumulonimbus clouds, increasing wind, darkening skies, and the presence of thunder. Signs of an imminent lightning strike include a blue haze around objects (known as St. Elmo's fire), static electricity over skin or hair, ozone smell, or a nearby crackling sound [35]. In the city, the recommendation is to seek shelter indoors. In the wilderness, however, one should avoid being in high-risk areas such as ridgelines and summits, as well as avoid tall isolated objects (i.e. trees), [35]. Additionally, one can attempt to insulate oneself from the ground by sitting on a pack (after removing any metal), a dry coiled rope, or a rolled foam sleeping pad [35]. Groups should spread out to avoid mass casualties [37].

In patients struck by lightning, the amperage the patient receives determines the severity of their injuries. A low amperage injury may lead to paresthesias whereas a high amperage injury can cause ventricular fibrillation and respiratory or cardiac arrest. After a lightning strike, an initial evaluation of the patient's cardiac and respiratory status is paramount, followed by initiation of cardiopulmonary resuscitation while awaiting emergency medical services. A pathognomonic skin finding found in lightning strike victims is the Lichtenberg figure. The Lichtenberg figure is a unique red colored fern-like pattern that is also known as arborescent burn of lightning, feathering, and ferning [35, 37, 38]. This ferning pattern does not follow any vascular or neurologic pattern [37]. The Lichtenberg figure is thought to be a physical manifestation of the lightning strike and not a true burn given that there are no changes to any tissue histologically. It typically appears within an hour of the lightning strike and resolves within 24-48 hours [35, 36, 38].

After a lightning strike, patients may also experience burns including linear burns, punctate burns, and full-thickness burns [36, 37]. Linear burns form as a patient's sweat is vaporized during the electrical current causing a partial-thickness burn (also known as a flashover). Punctate burns occur at the presumed site of exit of the lightning strike causing a circular burn. Larger full thickness burns may occur when a patient is in contact with an object that has been struck by lightning or by clothing melting secondary to the lightning strike [35]. Patients with burns should receive medical attention and be treated with routine burn care.

Prolonged Exposure Dermatosis

Prolonged exposure dermatosis (previously referred to as sun bumps) is a recently described phenomenon by Totten et al. in 2015 [39]. They performed an observational study of 74 participants in a ski touring class in Wyoming and found that 26% of participants had similar lesions, described as edematous pale papules and plaques with erosions and crusts on underlying erythema. The most commonly affected area in the study was the face (90% of affected persons, with the cheek and nose being the most common sites). The lesions occurred after approximately 8.7 days in the wilderness and resolved 10.6 days later. There was a similar incidence between men and women. The affected individuals did not have a history of polymorphous light eruption. Interestingly, Totten et al. did not find a correlation with sun exposure or lack of sun protection [39].

Conclusion

Spending time in nature, particularly in the mountains, is a wonderful and therapeutic adventure. There are many possible exposures that can result in dermatologic manifestations. If one knows the emergent warning signs and practices good prevention, exploring the great outdoors can be thrilling and safe.

References

1. Bergeron MF, Bahr R, Bartsch P, Bourdon L, Calbet JA, Carlsen KH, Castagna O, Gonzalez-Alonso J, Lundby C, Maughan RJ, Millet G, Mountjoy M, Racinais S, Rasmussen P, Singh DG, Subudhi AW, Young AJ, Soligard T, Engebretsen L. International Olympic Committee consensus statement on thermoregulatory and altitude challenges for high-level athletes. *Br J Sports Med*. 2012;46(11):770-9. [PMID: 22685119].
2. Singh G, Chatterjee M, Grewal R, Verma R. Incidence and care of environmental dermatoses in the high-altitude region of ladakh, India. *Indian J Dermatol*. 2013;58(2):107-12. [PMID: 23716798].
3. Castellani JW, Young AJ, Ducharme MB, Giesbrecht GG, Glickman E, Sallis RE, American College of Sports M. American College of Sports Medicine position stand: prevention of cold injuries during exercise. *Med Sci Sports Exerc*. 2006;38(11):2012-29. [PMID: 17095937].
4. Fudge JR, Bennett BL, Simanis JP, Roberts WO. Medical Evaluation for Exposure Extremes: Cold. *Wilderness Environ Med*. 2015;26(4 Suppl):S63-8. [PMID: 26617380].
5. McIntosh SE, Campbell A, Weber D, Dow J, Joy E, Grissom CK. Mountaineering medical events and trauma on Denali, 1992-2011. *High Alt Med Biol*. 2012;13(4):275-80. [PMID: 23270445].
6. Monseau AJ, Reed ZM, Langley KJ, Onks C. Sunburn, Thermal, and Chemical Injuries to the Skin. *Prim Care*. 2015;42(4):591-605. [PMID: 26612374].
7. Page EH, Shear NH. Temperature-dependent skin disorders. *J Am Acad Dermatol*. 1988;18(5 Pt 1):1003-19. [PMID: 3290279].
8. Sallis R, Chassay CM. Recognizing and treating common cold-induced injury in outdoor sports. *Med Sci Sports Exerc*. 1999;31(10):1367-73. [PMID: 10527306].
9. Sward DG, Bennett BL. Wilderness medicine. *World J Emerg Med*. 2014;5(1):5-15. [PMID: 25215140].
10. McIntosh SE, Hamonko M, Freer L, Grissom CK, Auerbach PS, Rodway GW, Cochran A, Giesbrecht G, McDevitt M, Imray CH, Johnson E, Dow J, Hackett PH, Wilderness Medical S. Wilderness Medical Society practice guidelines for the prevention and treatment of frostbite. *Wilderness Environ Med*. 2011;22(2):156-66. [PMID: 21664561].
11. Harirchi I, Arvin A, Vash JH, Zafarmand V. Frostbite: incidence and predisposing factors in mountaineers. *Br J Sports Med*. 2005;39(12):898-901; discussion [PMID: 16306495].
12. Murphy JV, Banwell PE, Roberts AH, McGrouther DA. Frostbite: pathogenesis and treatment. *J Trauma*. 2000;48(1):171-8. [PMID: 10647591].
13. McIntosh SE, Opacic M, Freer L, Grissom CK, Auerbach PS, Rodway GW, Cochran A, Giesbrecht GG, McDevitt M, Imray CH, Johnson

EL, Dow J, Hackett PH, Wilderness Medical S. Wilderness Medical Society practice guidelines for the prevention and treatment of frostbite: 2014 update. *Wilderness Environ Med*. 2014;25(4 Suppl):S43-54. [PMID: 25498262].
14. Twomey JA, Peltier GL, Zera RT. An open-label study to evaluate the safety and efficacy of tissue plasminogen activator in treatment of severe frostbite. *J Trauma*. 2005;59(6):1350-4; discussion 4-5. [PMID: 16394908].
15. Cauchy E, Cheguillaume B, Chetaille E. A controlled trial of a prostacyclin and rt-PA in the treatment of severe frostbite. *N Engl J* Med. 2011;364(2):189-90. [PMID: 21226604].
16. Singh GK, Datta A, Grewal RS, Suresh MS, Vaishampayan SS. Pattern of chilblains in a high altitude region of Ladakh, India. *Med J Armed Forces India*. 2015;71(3):265-9. [PMID: 26286795].
17. Connolly M. Systemic Sclerosis (Scleredema) and Related Disorders. In: Bolognia J, Jorizzo J, Schaffer J, editors. Dermatology: Elselvier Health Sciences; 2012. p. 643-55.
18. Alangari AA, Twarog FJ, Shih MC, Schneider LC. Clinical features and anaphylaxis in children with cold urticaria. *Pediatrics.* 2004;113(4):e313-7. [PMID: 15060259].
19. Hochstadter EF, Ben-Shoshan M. Cold-induced urticaria: challenges in diagnosis and management. *BMJ Case Rep*. 2013;2013. [PMID: 23839613].
20. Neittaanmaki H. Cold urticaria. Clinical findings in 220 patients. *J Am Acad Dermatol.* 1985;13(4):636-44. [PMID: 4078052].
21. Baron ED, Suggs AK. Introduction to photobiology. *Dermatol Clin*. 2014;32(3):255-66, vii. [PMID: 24891049].
22. Herlihy E, Gies PH, Roy CR, Jones M. Personal dosimetry of solar UV radiation for different outdoor activities. *Photochem Photobiol.* 1994;60(3):288-94. [PMID: 7972383].
23. Gilchrest BA, Eller MS, Geller AC, Yaar M. The pathogenesis of melanoma induced by ultraviolet radiation. *N Engl J Med.* 1999;340(17):1341-8. [PMID: 10219070].
24. Lichte V, Dennenmoser B, Dietz K, Hafner HM, Schlagenhauff B, Garbe C, Fischer J, Moehrle M. Professional risk for skin cancer development in male mountain guides--a cross-sectional study. *J Eur Acad Dermatol Venereol.* 2010;24(7):797-804. [PMID: 20015058].
25. Moyer VA, Force USPST. Behavioral counseling to prevent skin cancer: U.S. Preventive Services Task Force recommendation statement. *Ann Intern Med*. 2012;157(1):59-65. [PMID: 22751761].
26. Tripp MK, Watson M, Balk SJ, Swetter SM, Gershenwald JE. State of the science on prevention and screening to reduce melanoma incidence and mortality: The time is now. *CA Cancer J Clin.* 2016. [PMID: 27232110].
27. Mulliken JS, Russak JE, Rigel DS. The effect of sunscreen on melanoma risk. *Dermatol Clin*. 2012;30(3):369-76. [PMID: 22800545].
28. Kallini JR, Hamed N, Khachemoune A. Squamous cell carcinoma of the skin: epidemiology, classification, management, and novel trends. *Int J Dermatol.* 2015;54(2):130-40. [PMID: 25428226].
29. Zink A, Koch E, Seifert F, Rotter M, Spinner CD, Biedermann T. Nonmelanoma skin cancer in mountain guides: high prevalence and lack of awareness warrant development of evidence-based prevention tools. *Swiss Med Wkly*. 2016;146:w14380. [PMID: 27922162].
30. Buller DB, Andersen PA, Walkosz B. Sun safety behaviours of alpine skiers and snowboarders in the western United States. *Cancer Prev Control.* 1998;2(3):133-9. [PMID: 10093624].
31. Lim H, Hawk J. Photodermatologic Disorders. In: Bolognia J, Jorizzo J, Schaffer J, editors. Dermatology: Elselvier Health Sciences; 2012. p. 1467-86.
32. Reider N, Fritsch P. Other Eczematous Eruptions. In: Bolognia J, Jorizzo J, Schaffer J, editors. Dermatology: Elselvier Health Scienes; 2012. p. 219-31.
33. Moehrle M, Dennenmoser B, Schlagenhauff B, Thomma S, Garbe C. High prevalence of seborrhoeic dermatitis on the face and scalp in mountain guides. *Dermatology.* 2000;201(2):146-7. [PMID: 11053918].
34. Spano SJ, Campagne D, Stroh G, Shalit M. A lightning multiple casualty incident in Sequoia and Kings Canyon National Parks. *Wilderness Environ Med.* 2015;26(1):43-53. [PMID: 25281586].
35. Davis C, Engeln A, Johnson EL, McIntosh SE, Zafren K, Islas AA, McStay C, Smith WR, Cushing T, Wilderness Medical S. Wilderness Medical Society practice guidelines for the prevention and treatment of lightning injuries: 2014 update. *Wilderness Environ Med.* 2014;25(4 Suppl):S86-95. [PMID: 25498265].
36. Mahajan AL, Rajan R, Regan PJ. Lichtenberg figures: cutaneous manifestation of Tel electrocution from lightning. *J Plast Reconstr Aesthet Surg.* 2008;61(1):111-3. [PMID: 17664090].
37. Nagesh IV, Bhatia P, Mohan S, Lamba NS, Sen S. A bolt from the blue: Lightning injuries. *Med J Armed Forces India*. 2015;71(Suppl 1):S134-7. [PMID: 26265809].
38. Cherington M, Olson S, Yarnell PR. Lightning and Lichtenberg figures. *Injury.* 2003;34(5):367-71. [PMID: 12719166].
39. Totten JE, Brock DM, Schimelpfenig TD, Hopkin JL, Colven RM. Prolonged Exposure Dermatosis: Reporting High Incidence of an Undiagnosed Facial Dermatosis on a Winter Wilderness Expedition. *Wilderness Environ Med.* 2015;26(4):525-30. [PMID: 26165579].