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# Heat illnesses: a hot topic in the setting of global climate change

## Background

Heat illnesses affect a large number of people every year and are becoming an increasing cause of pathology as climate change results in increasing global temperatures.

## Objective

This article will review the physiological responses to heat, as well as the pathophysiological processes that result in heat illnesses. The emphasis will be on providing general practitioners (GPs) with an understanding of how to prevent heat illness in their patients and how to predict who is most at risk.

## Discussion

Heat illnesses may be thought of as minor or major illnesses, any of which may present to the GP. Consideration must be given to identifying those who need more critical intervention and on when to transfer for higher-level of care.

## Keywords

climate change; emergencies; environmental medicine; general practice; heat stress disorders; sunburn



Heat illnesses encompass a range of specific disease states and afflict a large number of people every year. At their most minor, heat illnesses are no more than a nuisance, but at their most severe, they may be life-threatening. With the effects of climate change over time, the frequency and magnitude of heat waves is increasing.<sup>1</sup> As a result, heat illnesses and other unrelated illnesses that may be affected by heat are being seen in ever increasing numbers.<sup>2-5</sup> This article will review the spectrum of heat illnesses, specifically focusing on the recognition and management of minor and major illnesses.

## Homeostatic physiological responses to heat

Under normal circumstances, and in a vast range of temperatures, a panoply of physiological processes allow for humans to maintain a near constant internal environment that allows for maximal efficiency of cellular processes. When conditions become extreme, however, either as a result of extreme external heat or from the overproduction of internal heat, homeostatic mechanisms may be overwhelmed and eventually fail, resulting in the heat illnesses described below.

To better understand the illnesses that result when homeostatic mechanisms fail, it is instructive to first review those responses in detail:<sup>6</sup>

- The primary means by which body temperature is regulated is through the skin. Heat is lost to the surrounding air in increasing efficiency by conduction, radiation, evaporation and convection:
  - Conduction is the process by which heat is transmitted directly from one object to another by direct contact.
  - Radiation is the process by which heat is shed by the emission of infrared energy.
  - Evaporation occurs when water on the surface of a warm object evaporates off that surface taking with it energy in the form of heat.



- Convection occurs when there is movement of a cooler substance past a warmer one enhancing the transfer of heat. Convection dramatically improves heat loss by evaporation as well.
- To enhance heat loss through the skin, sweat glands excrete a concentrated salt solution with a high boiling point and enhance heat transfer as it evaporates.
- Blood vessels within the skin dilate and cardiac output increases to enhance maximal blood flow to the skin for cooling before returning to the core.

When a person moves to a hot environment or, alternatively, when the environment becomes hot, several physiological responses occur in an attempt to enhance the homeostatic responses outlined above. Collectively, these responses are referred to as acclimatisation and take 10–14 days to complete (Table 1).<sup>6</sup>

Even when homeostatic responses are functioning properly, behavioural issues can hinder adaptation to increased environmental heat. For example, the failure to dress properly or drink adequately may be seen in patients with mental illness and can lead to heat illness even though homeostatic responses are unaffected. Alternatively, disease states or medications may impair homeostatic responses, impairing the ability to shed heat and putting an individual at risk (Table 2).<sup>7</sup>

Finally, environmental conditions can contribute to a failure of homeostatic responses, or a person may overwhelm their physiology by simply generating more heat than can be effectively removed. Environmental conditions associated with increased likelihood of heat illness include increasing ambient temperature and higher humidity, whereas patient-related issues include obesity or exertion, especially in settings of high temperature.

## Role of the general practitioner

The general practitioner (GP) has an important role in mitigating the morbidity and mortality related to heat illnesses. Principal among these roles is the ability to recognise which patients are most vulnerable to such affliction so that they may be advised as to how best to prepare and to be on the alert for specific signs and symptoms. Because heat tends to affect specific types of people in specific areas, it is fairly easy to predict when and who may be at risk and to act proactively.<sup>8</sup> For example, elderly patients, particularly those with poorer socioeconomic status or those taking multiple medications, are at increased risk of succumbing to heat illness.

**Table 1. Physiological responses in heat acclimatisation**

- Increased sweat production
- More dilute sweat
- Sweating at lower temperatures
- Increased blood volume
- Decreased heart rate
- Decreased glycogen consumption
- Increased renal salt conservation

The GP may have an important role in educating these patients on how to behave in times of significant heat stress (eg increasing fluid intake, controlling environmental factors as best as possible, seeking out public, air-conditioned spaces) and what signs to watch for in terms of when to seek help.<sup>9</sup> Advice should be provided regarding appropriate fluid intake; for example, an electrolyte solution may be necessary to avoid hyponatraemia in the setting of prolonged exertion and/or humidity. The second, equally important role of the GP is to distinguish between minor illnesses for which patients may receive treatment as outpatients, and the major illnesses, which often require transfer to hospital for in-patient admission.

## Minor heat illnesses

Minor heat illnesses are commonly encountered and generally do not require more than supportive, symptomatic care.

### Sunburn

Prolonged exposure to ultraviolet radiation (UVR) from the sun results in sunburn. In general, the severity of sunburn is related to the intensity of the UVR and the duration of the exposure.<sup>10</sup> Sunburns are characterised by the appearance of intense redness, the sensation of burning and pain 4–6 hours after sun exposure. Recurrent sunburn in childhood and teenage years, especially when accompanied by blistering, is associated with a significant increase in the risks of basal cell carcinoma and malignant melanoma.<sup>10,11</sup>

Treatment for sunburn is almost always symptomatic. Blisters should be left intact. Topical creams and anaesthetics may be helpful, especially those containing vitamin E or aloe vera.<sup>12</sup>

**Table 2. Conditions that increase risks of heat illnesses**

Condition	Rationale
Cardiac disease	Poor cardiac output to the skin: <ul style="list-style-type: none"> <li>• decreases efficiency of heat shedding</li> </ul>
Renal impairment	Inability to retain sodium or water leads to: <ul style="list-style-type: none"> <li>• dehydration</li> <li>• decreased sweat production</li> <li>• perfusion of skin</li> </ul>
Endocrinopathies	<ul style="list-style-type: none"> <li>• Decreased sweat production</li> <li>• Poor regulation of heat production and shedding</li> </ul>
Medications	Anticholinergic medications: <ul style="list-style-type: none"> <li>• decrease sweat production</li> </ul> Diuretics: <ul style="list-style-type: none"> <li>• worsen dehydration</li> </ul> Beta-blockers: <ul style="list-style-type: none"> <li>• impair cardiac function</li> </ul>
Skin conditions	Impairment of efficient cooling



None of the non-steroidal anti-inflammatory drugs (NSAIDs), corticosteroids or antihistamines, either alone or in combination, has been shown to be helpful.

Prevention of sunburn remains the best course of action. This is achieved by ensuring adequate protection from UVR through any of the following measures:

- remaining indoors when the sun is strongest
- generous and frequent application of sunscreens
- use of UVR protective clothing.<sup>12</sup>

Rarely, significant sunburns may require more aggressive treatment in hospital. This would be true if the total body surface area burned was very large or if the severity of the burn was very grave. The issue for these patients is that the integrity of the skin is compromised and fluid loss may become severe, especially in young children. Although there are no established guidelines related to severe sunburn, it would be prudent for the GP to refer a patient with more than 5% total body surface area of second degree (blistered) sunburn for further evaluation for possible hospital admission.<sup>13</sup>

### **Miliaria rubra (also known as ‘prickly heat’ or ‘heat rash’)**

Sweat production is increased and begins at lower temperatures soon after moving to a warmer environment. This can result in sweat glands becoming occluded and then being unable to function efficiently. Occasionally, the glands become infected. The resulting papulovesicular eruption is pruritic and follows areas of skin that are covered by clothing (*Figure 1*). The rash itself is benign and self-limited. The aim of management is to keep affected areas dry and open to the air if at all possible. The condition generally lasts 7–10 days.<sup>6,14</sup>

### **Heat oedema**

The expansion of plasma volume with accompanying cutaneous vasodilation can result in dependent oedema in the extremities, most commonly in the hands and feet. This often results in jewellery or shoes no longer fitting properly. The condition is completely benign and resolves spontaneously within a few days. No treatment is needed aside from reassuring the patient.<sup>6,14</sup>

### **Heat syncope**

Vasodilation and pooling of fluid in the extremities may, in combination, lead to episodes of syncope that are essentially vasovagal in nature. Although treatment is not needed for this, aside from rest and fluids, the treating physician must be diligent in considering other possibilities such as primary cardiac issues or significant dehydration. These are more likely if the syncope occurs after the acclimatisation period or with physical exertion in the heat.<sup>6</sup>

### **Heat cramps**

Heat cramps are almost always seen in the setting of prolonged exertion in a hot environment and tend to affect the larger muscle groups such as the hamstrings, quadriceps and gastrocnemius.<sup>6</sup>



**Figure 1. Miliaria rubra (prickly heat rash)**  
Reproduced with permission from DermnetNZ from Miliaria.  
Available at <http://dermnetnz.org/hair-nails-sweat/miliaria.html>

Although long thought to be related to dehydration and electrolyte losses, there has never been any scientific evidence to support this assertion.<sup>15</sup> Regardless, management is still based on fluid and electrolyte repletion along with rest, stretching and massage. Heat cramps are benign and self-limited.

### **Major heat illnesses**

Minor heat illnesses represent no more than a mere nuisance for most patients, whereas major heat illnesses are a different story altogether. Unrecognised or untreated, major heat illnesses may cause significant morbidity and even death.

### **Heat exhaustion**

When an individual is exposed to heat for a prolonged period of time and homeostatic mechanisms begin to fail, heat exhaustion sets in. Signs and symptoms of heat exhaustion are varied and include tachypnoea, tachycardia, weakness, nausea, vomiting, profuse sweating, syncope, myalgias, flushing of the skin, headache and fatigue. Core temperature is also elevated in this setting but never above 40°C and mental status always remains normal.<sup>6,16,17</sup> The temperature and mental status distinctions are critical to note as they distinguish heat exhaustion from the much more serious heat stroke (*Table 3*).<sup>16,17</sup>

Patients who experience heat exhaustion are invariably depleted of water and electrolytes to varying degrees. The aim of management, therefore, is to cool the patient and restore volume and electrolytes by intravenous and oral routes.<sup>16</sup>

If a GP is confronted with a patient with suspected heat exhaustion, preparations should be made for transfer to hospital for more definitive management. A core temperature (as opposed to peripheral) should be obtained to help establish the diagnosis and differentiate it from heat stroke (see below). Temporising measures that can be initiated before transfer include administration of appropriate oral rehydration solutions

**Table 3. Distinguishing between heat exhaustion and heat stroke**

	Heat exhaustion	Heat stroke
<b>Core temperature</b>	May be normal, always <40°C	>40°C
<b>CNS status</b>	Normal	Always altered or depressed May present with seizures
<b>Hepatocellular dysfunction</b>	Absent	Present
<b>Coagulopathy</b>	Absent	May be present

(eg gastrolyte, hydrolyte), if possible, as well as enhancing cooling by placing the patient in as cool an environment as possible and disrobing them to as much of an extent as is achievable given the setting, while taking care to avoid shivering.

### Heat stroke

Unlike any of the other illnesses described in this article, heat stroke is a life-threatening emergency. Once cooling mechanisms fail, core temperature rises, eventually eclipsing the critical level beyond which significant cellular injury begins to occur. In humans this is 41.6–42°C. The pathological changes seen are believed to be initiated by direct toxicity mediated by the systemic inflammatory response syndrome (SIRS).<sup>18</sup> The elevated core temperature also activates the clotting cascade, leading to disseminated intravascular coagulation (DIC).<sup>19</sup> The two main organ systems affected by heat-triggered SIRS and DIC are the brain, particularly the cerebellum, and the liver. For this reason, diagnosis of heat stroke requires the presence of an elevated core temperature of >40°C, altered central nervous system (CNS) status and evidence of hepatocellular dysfunction manifested by elevated liver function tests.<sup>16</sup> CNS dysfunction may manifest as vertigo, confusion, ataxia and, eventually, coma. Renal and splanchnic shunting of blood to the skin in an effort to lower core temperature may further worsen liver and kidney injury.<sup>16</sup> Other organ systems may also be affected, including the gut (bowel ischaemia) and heart (severe cardiomyopathy in the absence of known coronary artery disease).<sup>20</sup>

Heat stroke may arise in one of two distinct settings:<sup>16</sup>

- Exertional heat stroke (EHS): seen in athletes who exert themselves in a hot environment, or occupational settings where patients are exposed to high temperatures and/or are required to wear insulating gear in warm environments (eg firefighters).
- Classic heat stroke (CHS): seen in individuals who have 'impaired physiologic mechanisms for heat dissipation, stemming from comorbid metabolic or cardiac conditions, or who lack the means to escape a hot environment, for economic, psychiatric, or social reasons, including substance abuse, or because of physical challenges'.<sup>16</sup>

Of the two types, CHS is much more concerning as the underlying comorbidities often lead to delays in presentation and higher risks of mortality. The GP is often not properly equipped to manage these critically ill patients and so the emphasis should be on rapid identification and prompt transfer. Once identification has been made, and while transfer is being arranged, any measure that can be taken

to begin cooling the patient and possibly rehydrating them (with oral rehydration solution) should be taken. Once in hospital, initial management of the patient with heat stroke is aimed at stabilisation of the airway, breathing, circulation and cooling (A, B, Cs).

The prognosis for heat stroke depends on several factors:<sup>21</sup>

- the presence of underlying comorbidities that can augment the risk (*Table 2*)
- evidence of significant end-organ failure: seizures, elevated serum creatinine, abnormal coagulation profile and abnormal liver function tests all indicate multi-system organ failure and portend a poor outcome
- the response to treatment: patients who regain consciousness after their core temperature has been lowered below 40°C have a good prognosis, whereas those who do not uniformly do poorly.

### Summary

Heat illnesses are a significant cause of morbidity and mortality. The frequency and adverse impact of these illnesses are increasing as the climate warms over time. The GP can expect to be called on to manage minor heat illnesses and must be familiar with the signs, symptoms and initial phases in the management of major illnesses. Early recognition and implementation of cooling are critical in effecting good outcomes in these cases.

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