ACOEM GUIDANCE STATEMENT

Prevention of Occupational Heat-Related Illnesses


ACOEM Work Group on Occupational Heat-Related Illness

High ambient temperatures and strenuous physical activity put workers at risk for a variety of heat-related illnesses and injuries. Through primary prevention, secondary prevention, and treatment, OEM health providers can protect workers from the adverse effects of heat. This statement by the American College of Occupational and Environmental Medicine provides guidance for OEM providers who serve workers and employers in industries where heat exposure occurs.

H igh ambient temperatures and strenuous physical activity put workers at risk for a variety of heat-related illnesses and injuries (HRIs). According to the United States (US) Bureau of Labor Statistics (BLS), each year thousands of American workers experience serious heat-related illnesses, many of which result in fatality.1 Heat exposure directly caused more than 350 US worker deaths between 2000 and 2011.2,3 More recently, dozens of workers continue to die each year.1,4

Earth’s climate has become warmer in recent decades. Globally, the 10 hottest years on record have all occurred since 2005,4 with the most recent year, 2020, being tied with 2016 as the year with the warmest global average surface temperature.5,6 In the contiguous US, 2020 was the fifth warmest year on record, with the five warmest years all occurring since 2012.7 Climate scientists project that these warming trends will continue into the future.8

From the American College of Occupational and Environmental Medicine, Elk Grove, Illinois.

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Therefore, it is likely that US workers will be at risk of HRIs more often and in more parts of the country.9

Through primary prevention, secondary prevention, and treatment, occupational and environmental medicine (OEM) health care providers can protect workers from the adverse effects of heat. This statement by the American College of Occupational and Environmental Medicine (ACOEM) contains guidance for OEM providers who serve workers and employers in industries where heat exposure occurs. Table 1 provides a brief overview of the recommendations. More details are contained in the following sections. A prior ACOEM guidance statement about climate change contained general heat-related recommendations10; reviewing that article will be of benefit in understanding this document.

Description of Occupational Heat-Related Illnesses

Five diagnoses comprise most lists of “heat-related illnesses.” They are heat stroke, heat exhaustion, heat cramps, heat syncope, and heat rash:

1. Heat stroke is the most severe heat-related illness. Heat stroke is defined by a core body temperature ≥104°F (40°C) combined with central nervous system (CNS) dysfunction. CNS manifestations of heat stroke ranged from confusion and slurred speech to coma, seizures, and death. Heat stroke is a life-threatening emergency with a case fatality rate above 80% in workers.11 Manifestations of heat stroke in other organ systems include rhabdomyolysis, acute kidney injury (AKI), arrhythmias, liver damage, acute lung injury (which ranges from mild hypoxemia to acute respiratory distress syndrome), and disseminated intravascular coagulation (DIC).12

2. Heat exhaustion is an illness in which the body’s response to heat strain produces symptoms that can include nausea, fatigue, headache, dizziness, or excessive sweating. Symptoms of heat exhaustion are non-specific and can mimic other ailments such as viral illness. Diagnosis is aided by obtaining a thorough history to rule out other causes and to document antecedent exposure to heat stress. Heat exhaustion is distinguished from heat stroke by the lack of severe CNS abnormalities and the lack of extreme hyperthermia. There is no objective temperature threshold for the diagnosis of heat exhaustion. Heat exhaustion can progress quickly to heat stroke if the heat exposure continues.

3. Heat cramps are muscle spasms or pain following physical activity in hot environments. Heat cramps often affect the muscles of workers’ legs, upper extremities, back, or abdomen.

4. Heat syncope is an episode of orthostatic fainting due to transient reduction of cerebral blood flow during heat exposure. Heat-related changes in vascular resistance and blood distribution can cause the pooling of blood in the lower extremities, leading to syncope.

5. Heat rash (miliaria rubra) is a skin disorder associated with perspiration and blocked sweat ducts. Symptoms can include erythema, pruritis, vesicles, and papules.13

In addition to the above five diagnoses, heat-exposed workers can develop other acute or chronic disorders including the following:

- Dehydration due to loss of body fluid from sweating, without adequate replacement, is a common result of heat stress. Dehydration upsets the thermoregulatory mechanisms that regulate body temperature, increasing the risk of heat stroke. Symptoms and signs of dehydration may include thirst, fatigue, weakness, dizziness, tachycardia, hypotension, oliguria, and decreased skin turgor. Laboratory findings may include elevated blood urea nitrogen (BUN) and creatinine, high or low serum potassium and sodium levels, acid-base imbalance, and urine-specific gravity that is either high or normal. Low urine sodium may be a diagnostic indicator of dehydration.14

In addition to being a result of heat exposure, dehydration can be a preexisting factor that increases the risk of other HRIs. Pre-existing causes of
TABLE 1. Overview of Recommendations to Protect Workers from Heat-Related Illness

<table>
<thead>
<tr>
<th>Item</th>
<th>Recommendations</th>
</tr>
</thead>
<tbody>
<tr>
<td>Medical surveillance</td>
<td>Workers who will be exposed to heat stress should receive pre-placement and periodic medical examinations. These examinations should include past medical history and occupational history, assessment of personal risk factors, physical examination, and measurement of core temperature. Clinicians should recommend temporary or permanent work restrictions when indicated.</td>
</tr>
<tr>
<td>RTW after heat-related illness</td>
<td>Before clearing affected workers for RTW, ensure they are asymptomatic and that any abnormal biomarkers (eg, serum creatinine) have returned to normal. Verify that the employer has taken protective actions to prevent a recurrence.</td>
</tr>
<tr>
<td>Physiologic monitoring</td>
<td>For some occupations with high levels of heat stress, real-time monitoring of physiologic parameters like heart rate or body temperature may be appropriate. Physiologic monitoring programs should be designed and overseen by a qualified clinician.</td>
</tr>
<tr>
<td>Measurement of heat stress</td>
<td>Heat stress includes both metabolic heat and external environmental heat. Assessments should account for the worker’s physical activity level and the level of external heat. WBGT is the preferred environmental heat metric. Alternatives to WBGT include dry bulb temperature and heat index.</td>
</tr>
<tr>
<td>Design of employer programs to prevent heat-related illness</td>
<td>Programs should be based on the hierarchy of controls with engineering controls (eg, air conditioning) preferred when possible. Other important components include acclimatization protocols for new workers, work/rest cycles or self-pacing, heat stress hazard assessments, provision of hydration fluids, training about heat-related signs and symptoms, and an emergency response plan.</td>
</tr>
<tr>
<td>Protection of unacclimatized workers</td>
<td>Workers who are unacclimatized to heat stress, particularly newly hired workers, are at high risk of severe heat-related illness. Physiologic acclimatization develops gradually over the course of several days of exposure to heat stress. Unacclimatized workers should receive protections such as more frequent rest breaks and shorter duration of work in the heat. They should never work alone, and any signs or symptoms of heat-related illness should prompt immediate evaluation and first aid.</td>
</tr>
</tbody>
</table>

Dehydration include medications (eg, diuretics), alcohol, caffeine, acute illness (eg, gastroenteritis causing vomiting and diarrhea), chronic medical problems (eg, kidney disease or poorly controlled diabetes), and exposure to heat stress in the previous few days.12,14

- **Rhabdomyolysis** is the rapid breakdown and necrosis of skeletal muscle. It may occur in HRI, particularly when exposed to a hot environment is combined with prolonged, heavy, or unaccustomed physical exertion. For example, newly hired firefighters, soldiers, and police officers have been hospitalized with rhabdomyolysis after strenuous physical training sessions. Rhabdomyolysis can lead to arrhythmias, AKI, and DIC. The classic symptoms of rhabdomyolysis are muscle pain, weakness, and dark urine (due to excretion of myoglobin). Other symptoms include fever, fatigue, weakness, nausea, and vomiting, but affected individuals may be asymptomatic. The diagnosis of rhabdomyolysis requires an elevated creatine kinase (CK), also known as creatine phosphokinase (CPK). In general, the higher the CK levels, the more severe the rhabdomyolysis.12,14,16 Rhabdomyolysis can occur after strenuous physical exertion even in the absence of elevated ambient temperatures. Other non-heat-related causes of rhabdomyolysis include medications, drugs of abuse, alcohol, toxins, infections, poorly controlled chronic medical conditions (eg, diabetic ketoacidosis), crush injuries, and electrical injuries.16

- **Acute kidney injury**, previously termed an acute renal failure, is an abrupt loss of kidney excretory function that may accompany HRI. Heat-related AKI is caused by one or more of the following mechanisms: pre-renal hypovolemia from dehydration, acute tubular injury because of rhabdomyolysis or heat-related tubulointerstitial nephritis. The signs and symptoms of AKI depend on the severity of the HRI. Typical laboratory findings include elevated BUN and creatine, electrolyte abnormalities, and oliguria.11,16,17

- **Chronic kidney disease of unknown etiology (CKDu)**, first described in El Salvador sugarcane workers, has now been detected in agricultural areas in various equatorial regions.18 In CKDu, chronic or end-stage kidney disease primarily affects young and middle-aged male workers. Postulated mechanisms of CKDu include infections, exposure to pesticides or heavy metals, or exposure to occupational heat stress. Evidence that supports the link between heat stress and CKDu includes rodent studies,19 and human studies that found repeated heat stress and dehydration were associated with kidney injury.20,21 Interventions to reduce heat stress, such as work-rest cycles, increased fluids, and other administrative controls appear protective.17,18,22–24

- **Other kidney disorders** associated with heat stress and dehydration include kidney stones and urinary tract infections.17

- **Skin burns**, in addition to heat rash, can sometimes be severe and accompanied by systemic symptom complexes associated with a deranged thermoregulation process, such as fever, chills, nausea, and other signs of altered mental status like dizziness and confusion. In more severe cases, depending on the prevailing environment, CNS dysfunction like ataxia, nuchal rigidity, and coma can occur.23

- **Traumatic injuries and decreased productivity** as a result of heat stress may reduce worker productivity and increase the risk of traumatic injuries. In a recent meta-analysis, there was a positive association between ambient temperature and risk of occupational injury, but the association was not statistically significant in all subgroup analyses.26 Further research is needed to understand the impact of heat stress on worker productivity and occupational injuries.27

**Heat Balance and Modes of Heat Exchange**

Maintenance of core body temperature within a safe range depends upon heat exchange between the worker and the environment. Workers’ bodies generate metabolic heat constantly. Those who do frequent heavy physical activity (eg, carrying loads, digging, climbing stairs, or ladders) are at increased risk of heat-related illness because of their higher metabolic heat generation, compared to workers who perform the light activity. Evaporative cooling, convection, and radiation are the
primary routes by which workers dissipate excess metabolic heat to the environment. 

- Evaporative cooling refers to heat removal from the skin during the phase transition from liquid to water vapor when sweat evaporates.
- Convection is heat transfer via air movement, such as a breeze.
- Radiation is the transfer of heat via electromagnetic waves, namely infrared radiation emitted by workers’ skin.

The rate of heat transfer depends upon several variables including the ambient temperature, humidity, sunlight, and wind speed. All modes of cooling are less efficient at higher temperature and humidity levels. In some circumstances, certain cooling pathways are completely ineffective, and workers gain heat from their surroundings. For instance, when the ambient temperature is above the average skin temperature of 95°F (35°C), convection increases the body’s total heat load. 

Clothing

In some occupations, clothing is an important factor that affects workers’ heat balance and contributes to heat stress. Bulky or non-breathable protective equipment can hinder heat dissipation from the body. Some vapor-impermeable clothing materials dramatically reduce the efficiency of sweating and evaporative cooling. Examples of occlusive clothing that contributes to workers’ heat stress include firefighters’ turnout gear, chemical protective suits worn by hazmat workers, aluminized overgarments worn by foundry workers to prevent thermal burns, thermal protection gear worn by welders and glassmakers, or even occlusive dressings for wounds.

When the job does not require specialized gear to protect against other hazards, clothing should be chosen to minimize heat stress if possible. Ideally, outdoor workers should wear clothing that is loose-fitting, light-colored, lightweight, and long-sleeved when environmental temperatures exceed body temperatures. Workers should be discouraged from wearing dark or non-breathable clothing.

Measurement of Heat Stress

As used by industrial hygienists, the term “heat stress” encompasses both environmental and metabolic heat produced by physical activity. Both components contribute to workers’ overall thermal load. The rate of metabolic heat generation (Watts) can be estimated from tables that map job tasks to physical activity categories. For example, walking is considered “moderate” physical activity, and using a shovel to dig a hole is “heavy” activity. The exact rate of metabolic heat generation depends upon individual factors such as age, body habitus, work pace, and the number and duration of rest breaks. Therefore, the standard tables provide rough estimates only. When heat stress is near or above screening thresholds, that is, TLV, a more detailed job task analysis by a qualified expert can provide a better estimate of metabolic heat.

Environmental heat—the thermal load imparted by one’s surroundings—depends upon several factors including ambient temperature, relative humidity, radiant heat, and air movement. Various metrics or devices are available to measure environmental heat in workplace settings. At US workplaces, three environmental heat metrics that are commonly used are: 1) ambient temperature; 2) heat index; and 3) wet bulb globe temperature (WBGT).

- Ambient temperature, also known as dry bulb temperature, can be measured alone and used for environmental heat assessment. For example, California’s heat stress standard for outdoor workplaces requires employers to undertake additional preventive measures when the ambient temperature reaches or exceeds 80°F. 
- Heat Index, developed by the National Weather Service, combines ambient temperature and relative humidity into a single number that represents how hot the conditions feel to humans. Humid conditions feel hotter because increased humidity inhibits evaporative cooling of sweat. Many US workers and supervisors rely upon Heat Index reports and forecasts provided by local weather bureaus.
- WBGT was developed by the US military in the 1950s as part of an effort to prevent heat-related illnesses in soldiers and trainees. WBGT devices combine readings from three temperature sensors (dry bulb, wet bulb, and globe) into a single value that accounts for temperature, humidity, radiant heat, and air movement. Military and civilian guidance documents help responsible parties interpret WBGT readings, in the context of workers’ physical activity levels, to establish proper amounts of rest breaks and water consumption. 

Personal Risk Factors

Clinicians should be aware of personal risk factors that place some workers at higher risk of heat-related illness. Knowledge of these personal risk factors allows individualized preventive recommendations at routine encounters such as pre-placement examinations, periodic examinations, and primary care visits. We recommend pre-placement and periodic medical surveillance examinations for all workers who are exposed to occupational heat stress (“Guidance for OEM Providers Who See Workers in Clinic” section). Assessment of personal risk factors is also vital during post-incident investigations of occupational heat-related illnesses, to inform decisions about return-to-work for affected workers and to advise employers about mitigation strategies.

Acclimatization and New Workers

Lack of acclimatization to heat stress appears to be the most important personal risk factor for occupational HRIs and fatalities. The ACGIH defines acclimatization as “a gradual physiological adaptation that improves an individual’s ability to tolerate heat stress.” During the first 7 to 14 days of exposure to heat stress, physiologic adaptations include the earlier onset of sweating, increased sweat production, lower sodium levels in the sweat, higher plasma volume, and improved circulation. Acclimatized individuals also typically have increased skin blood flow, lower core body temperatures, and lower heart rates, compared to unacclimatized individuals performing the same work. Studies have shown that as little as two 1-hour periods per day of working in the heat can cause beneficial physiologic changes. Conversely, if a worker’s heat stress exposure ceases for as few as 7 to 14 days, he or she can become de-acclimatized, and a gradual reintroduction to heat stress should begin again upon return to work (RTW).

Although there is no universal definition of “new worker,” it is generally accepted that the risk of HRI is highest during the first 2 to 4 weeks of a worker’s heat stress exposure. For this document, “new workers” refers not only to new hires but also to other workers who lack acclimatization, including temporary or contingent workers, workers who have transferred to a new job task at the same employer, and workers who have returned to work after time off. New workers are at a very high risk of heat-related fatality. Epidemiology studies conducted by the US military in the mid-20th century revealed that over 50% of fatal heat stroke cases involved recruits who had spent 4 weeks or less in hot training environments. The US OSHA performed several structured analyses of HRIs and found that between 2011 and 2016, 45% (10 of 22) of civilian heat-related fatalities
occurred on the worker’s very first day of hire or the first day back at work after an extended absence. An even higher fraction, 73% (16 of 22) of cases, died during their first week on the job. Similarly, in an Australian study of heat-related fatalities, 50% (6 of 12) occurred within the first 4 days on the job. A Canadian study of 785 emergency department visits for HRI and 612 lost-time workers’ compensation claims for HRI between 2004 and 2011, found that workers employed less than 1 month were almost twice as likely to have a lost-time claim for HRI.

Studies of athletes confirm the benefits of heat stress acclimatization. An international sports medicine panel reviewed the literature on training and competing in the heat and concluded that heat acclimatization is essential for athletes competing in warm to hot environmental conditions. A recent meta-analysis of 96 studies on heat adaptation and physiologic response concluded that short acclimatization periods (<7 days) provide some benefit, but acclimatization programs of 14 days or longer maximize the physiologic benefit of acclimatization.

Medical Conditions, Comorbidities, and Medications

Personal factors such as pre-existing medical conditions, obesity, certain medications, age, pregnancy, and lower levels of physical fitness, may increase the risk of HRI (Table 2). As part of an employer’s heat illness prevention program, pre-placement and periodic medical monitoring enable OEM providers to screen for these risk factors and educate workers or recommend interventions to reduce the risk.

Medical conditions such as cardiovascular disease, diabetes, pulmonary disease, kidney disease, skin disorders, and infections increase the risk of HRI and heat-related deaths. Much of the evidence comes from observational studies of elderly individuals during heat waves. There is also some evidence from working populations. For example, in a review of 64 work-related HRIs investigated by OSHA from 2011 to 2016, 38% of patients were obese, 26% had hypertension, 10% had diabetes, and 17% had cardiac disease. These conditions may increase the severity of HRI, as workers who died of HRI were significantly more likely to have one or more of these comorbidities, compared to workers with non-fatal HRI.

Older workers may have impaired thermoregulatory mechanisms. (In this context, the definition of “older” is imprecise, as different references state that the risk of HRI increases after age 60 or age 75 or another threshold age.) These age-related changes include slower sweat gland response to heat, lower plasma volume, decreased cardiac output, reduced blood flow to the skin, and diminished thirst drive. Working for shorter intervals and taking longer breaks may be protective for older workers who are at risk for HRI because of these natural effects of aging.

Pregnant women have higher body temperatures and need more fluids to cool their core temperature. A recent systematic review of adverse effects of environmental heat on pregnant women found evidence of neonatal stress, shortened gestation, lower birth weights, and increased stillbirths. The presence of the fetus during pregnancy increases the mother’s metabolic heat load, increasing the risk of HRI as well. Obesity has been identified as a risk factor for heat stress. Obese individuals are 3.5 times as likely to suffer an HRI. Although the causes of heat intolerance in obesity are not completely understood, mechanisms likely include an increase in the normal load for any given task, increased cardiac strain, impaired heat-sensing abilities, or increased internal heat retention due to the insulation of the adipose layer.

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Prescription or over-the-counter medications, alcohol, or illicit drugs may impair thermoregulatory mechanisms. Implicated medications include antidepressants and other psychotropic medications, diuretics, and anticholinergics (Table 3). Illicit drugs that increase the risk of HRI include cocaine and amphetamines such as methamphetamine. In particular, amphetamines, whether taken illegally or legally (such as for treatment of attention deficit hyperactivity disorder), can increase endogenous heat production. Amphetamine use appears to be strongly associated with severe HRI in workers. An OSHA case series found evidence of amphetamine use in 26.5% of workers with severe hyperthermia.

OEM clinicians evaluating workers with potential exposure to occupational heat stress should review prescription, over-the-counter, and recreational drug use with their patients. Clinicians should also review acute and chronic medical conditions and assess whether the medical conditions are well-controlled. OEM providers should educate workers about symptoms of heat stress, the importance of adequate fluid intake, and medical conditions and medications that can increase heat strain. Providing specific guidance to workers with comorbidities can reduce the risk of HRI. For some patients, clinicians may need to recommend temporary or permanent work restrictions.

Religious and Cultural Factors

Numerous religions and cultures observe holidays that call for reduced intake of calories and/or fluids (ie, fasting). In many cases, physiologic changes during the acute phase of the fasting period may impair the body’s normal thermoregulatory process until the body adapts to the reduced caloric state. Dietary changes and dehydration may predispose fasting workers to heat-related injuries. More research needs to occur to better define the risk associated with specific characteristics of various fasting periods and types of fasts. OEM providers should be aware of major fasting events to help guide employers toward increased surveillance and acclimatization of employees during temporary changes in diet.

GUIDANCE FOR HELPING EMPLOYERS DEVELOP A PROGRAM TO PREVENT HEAT-RELATED ILLNESSES

OEM clinicians may be asked to help employers design and implement HRI prevention programs. This section provides guidance about important components to include in such programs. Additional information is available from OSHA and ACGIH. Clinicians should also ensure that the programs comply with applicable

<table>
<thead>
<tr>
<th>TABLE 2. Personal Risk Factors for Heat-Related Illness</th>
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<tbody>
<tr>
<td><strong>Acute Infection or Illness</strong></td>
</tr>
<tr>
<td>Deconditioned status</td>
</tr>
<tr>
<td>Mental illness</td>
</tr>
<tr>
<td>Pregnancy</td>
</tr>
<tr>
<td>Skin disease</td>
</tr>
<tr>
<td>Kidney disease</td>
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<tr>
<td><strong>Cardiovascular Disease</strong></td>
</tr>
<tr>
<td>Diabetes mellitus</td>
</tr>
<tr>
<td>Neurological disease</td>
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<tr>
<td>Previous history of heat-related illness</td>
</tr>
<tr>
<td>Hypothyroidism</td>
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<tr>
<td>Parkinson disease</td>
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</table>

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TABLE 3. Partial List of Medications/Illicit Drugs that May Increase Risk of Heat-Related Illness

<table>
<thead>
<tr>
<th>Drug or Drug Class</th>
<th>Mechanism of Action</th>
</tr>
</thead>
<tbody>
<tr>
<td>Alcohol</td>
<td>Diuresis, cognitive impairment</td>
</tr>
<tr>
<td>Amphetamines (eg, methamphetamine, ecstasy [MDMA])</td>
<td>Increased endogenous heat production, cognitive impairment</td>
</tr>
<tr>
<td>ACE inhibitors ARB</td>
<td>Salt depletion</td>
</tr>
<tr>
<td>Anticholinergics (eg, benztpine, cyclobenzaprine)</td>
<td>Impaired heat dissipation (sweating)</td>
</tr>
<tr>
<td>Anticonvulsants (eg, topiramate)</td>
<td>Impaired heat dissipation</td>
</tr>
<tr>
<td>Antihistamines (eg, diphenhydramine)</td>
<td>Antimuscarinic, impaired heat dissipation</td>
</tr>
<tr>
<td>Antipsychotics (eg, haloperidol and phenothiazine)</td>
<td>Impaired heat dissipation</td>
</tr>
<tr>
<td>Benzodiazepines</td>
<td>Sedation and cognitive impairment could reduce judgment or perception of heat</td>
</tr>
<tr>
<td>Beta-blockers</td>
<td>Impaired cutaneous vasodililation</td>
</tr>
<tr>
<td>Cocaine</td>
<td>Increased endogenous heat production, impaired heat dissipation, cognitive impairment</td>
</tr>
<tr>
<td>Diuretics</td>
<td>Dehydration, salt depletion</td>
</tr>
<tr>
<td>Opioids</td>
<td>Sedation and cognitive impairment could reduce judgment or perception of heat</td>
</tr>
<tr>
<td>Tricyclic antidepressants</td>
<td>Impaired sweating, altered central thermoregulation</td>
</tr>
</tbody>
</table>

ACE, angiotensin converting enzyme; ARB, and angiotensin receptor blockers.

federal or state regulations. For instance, California, Minnesota, and Washington have state regulations regarding occupational heat stress.

In general, HRI prevention programs should be based upon the hierarchy of controls. At some indoor workplaces, it may be possible to use elimination or substitution to ensure that heat stress is not hazardous (eg, by removing heat-producing equipment and substituting cooler alternatives). At most other workplaces, heat stress is controlled by engineering controls, administrative controls, and personal protective equipment (PPE).

Engineering Controls

Engineering controls are devices or processes that reduce heat stress. Some engineering controls reduce environmental heat parameters such as temperature and/or humidity. Examples include air conditioning, ventilation, and fans. At outdoor work sites, shade structures (eg, canopies) can block solar radiant heat. Fans or misting devices can increase convective and evaporative cooling rates in some circumstances. Engineering controls can also reduce workers’ metabolic heat generation if they decrease workload. For instance, replacing manual lifts with a mechanical device (eg, forklift) can reduce the physical activity level of warehouse workers.

Protect Unacclimatized Workers

As mentioned above, new workers are at high risk of HRI. A primary reason appears to be those new workers, and long-term workers who return from an extended absence, may lack acclimatization to heat stress. All heat stress prevention programs should include an acclimatization protocol that allows unacclimatized workers to develop acclimatization gradually and safely. These should be codified in employer safety plan practices.

There are various methods to acclimatize workers gradually. The duration of heat stress exposure can be increased incrementally over a period of 7 to 14 days. For example, a new employee could work for 2 hours in the heat on his first day, followed by a 3-hour heat exposure on the second day. Alternatively, new workers can be given longer and more frequent rest breaks, compared to acclimatized existing workers. If an unacclimatized worker will work a full shift in the heat, she should be given hourly breaks in a cool location (eg, an air-conditioned room or a shaded area) to allow dissipation of excess body heat acquired during the periods of work.

It is also important to ensure that unacclimatized workers, particularly those who are new to the job, be monitored closely for early detection of the heat-related signs and symptoms. Monitoring can be performed either by supervisors or by coworkers via a “buddy” system. The supervisor or “buddy” in the field must be able to recognize the warning signs of impending or severe heat illness, including mental confusion, incoordination, or sustained vomiting. The supervisor’s responsibility is then to activate the employer’s emergency response plan as spelled out in the employer’s written heat illness prevention plan. The emergency response plan may often include calling the onsite emergency response plan or 911, as directed in their training. New workers should never work alone when heat stress is present. Any symptoms that could be heat-related should be investigated promptly, preferably by a knowledgeable healthcare professional, to ensure that heat exhaustion does not proceed to heat stroke. Finally, employers should ensure that unacclimatized workers consume an adequate (but not excessive) quantity of fluids.

Work/Rest Cycles or Self-Pacing

Heat stress can be mitigated by increasing the frequency and/or duration of rest breaks as heat stress rises. This concept is sometimes termed a “work/rest cycle.” In general, workers should rest for at least 15 minutes per hour when a heat stress hazard is present.14,15 Ideally, rest breaks should be mandatory, because workers might put themselves at risk of HRI by skipping optional breaks. Skipping breaks is a particular concern for newly hired workers who might not understand that their symptoms are heat-related or who might attempt to continue working while symptomatic.

Self-pacing is an alternative to mandatory rest breaks. Self-pacing means that workers are empowered to reduce their work pace to avoid unsafe heat strain in hot conditions. Several industries have successfully implemented self-pacing. To be successful, self-pacing requires a supportive corporate safety culture that allows workers to lower their pace with no negative repercussions.

Medical Surveillance/Monitoring

Medical monitoring evaluations are an important part of an HRI prevention program. The examinations should include obtaining the worker’s medical and occupational history with an emphasis on past, present, and anticipated future exposures to heat; prior history of heat-related illness; and personal risk factors for heat-related illness. “Guidance for OEM Providers Who See Workers in Clinic” section contains more information about clinical aspects of medical surveillance for heat-exposed workers.
Training
Employers should train employees and their supervisors (in their working languages) about heat stress safety. The training must include the identification of signs and symptoms of heat-related illness. Workers should be taught first aid for HRI, with an emphasis on calling 911 (or the appropriate on-site emergency response number) and providing immediate cooling if someone shows any signs of heat stroke (eg, confusion or slurred speech). Understanding proper hydration (ie, at least 8 oz. of fluids every 15–20 mins) is an essential part of this training. Topics like acclimatization, the consequences of risk factors such as drug use and obesity on one’s heat tolerance, and the role of metabolic heat in occupational HRI, should also be covered.14

Hazard Assessment
Monitoring the level of heat stress is the employer’s responsibility. OEM clinicians who consult for employers can direct them to consensus hazard assessment guidelines.1,14,17 Currently, the gold standard method for assessing heat stress at worksites is to measure WBGST and estimate workers’ metabolic rate (ie, workload).

Provide Adequate Hydration Fluids
The amount and type of fluids consumed are of particular importance for workers in hot environments. Employers should provide potable drinking water. The water should be cool and readily available near the job location. During prolonged periods (>2h) of sweating in hot conditions, employers should also provide electrolyte-containing beverages such as sports drinks.14 OEM clinicians should counsel workers to drink an adequate quantity of fluids to replace fluid losses from sweating, which can be substantial on hot days. Clinicians should also advise workers not to overconsume fluids, which can lead to exercise-associated hyponatremia. Pre- and post-shift weights are one way to monitor whether the worker consumed an appropriate quantity of fluids.

Emergency Response Plan
Workers and supervisors should be trained to recognize and provide first aid for HRIs. Heat stroke is a medical emergency. If there is evidence of heat stroke, such as abnormal behavior, confusion, or other signs of CNS dysfunction, first responders should notify emergency medical services by calling 911 or the appropriate on-site emergency response number. Concurrently, immediate cooling measures are necessary. An important early treatment of heat stroke is to apply ice and wet the worker’s skin using a cold-water bath, garden hose, or any other available source of cool water.14 Other symptoms of heat strain can be nonspecific and insidious in onset. Importantly, heat-related symptoms are often more subtle in older employees.14,22 Supervisors and coworkers must be able to recognize signs and symptoms of heat strain, provide first aid, remove the affected worker from further heat stress exposure, and obtain definitive medical care. Supervisors and coworkers should have a low threshold for contacting emergency medical services. To assure that ill workers receive first aid, supervisors should monitor their employees for heat strain during the workday.14 Alternatively, a buddy system, in which trained workers monitor each other, can be effective in some situations.

Personal Protective Equipment
If engineering and administrative controls do not adequately reduce heat stress, employers could consider PPE. For heat stress, PPE can include air-, ice-, or water-cooled garments such as ice vests or wetted overgarments.24 Some industries require overgarments that can increase workers’ heat stress, ie, fire retardant clothing in aluminum smelters, etc. Their use should be regulated to only being utilized in the immediate environment and will increase the need for shorter work-rest cycles.

GUIDANCE FOR OEM PROVIDERS WHO SEE WORKERS IN CLINIC

All workers who are exposed to heat stress should receive medical surveillance. This medical surveillance should include, at minimum, a pre-employment examination and periodic examinations. Currently, there are limited testing modalities that specifically assess an individual’s capacity to undertake manual labor in hot environments.53,54 It is therefore up to the OEM clinician to incorporate national and international heat-related guidance, their own clinical judgment, and the worker’s past medical history and past occupational history, to reach a conclusion about the worker’s fitness to work in hot environments.

The baseline and periodic examinations should include a health history questionnaire to identify personal risk factors for HRI (“Personal Risk Factors” section). A full review of the job description should be conducted to identify the number of hours per day and number of days per week of exposure to heat stress, the nature of the physical labor, and whether there are mitigating factors such as access to cool areas for rest breaks. Local climate patterns should also be considered. Measurements of serum creatinine and glomerular filtration rate should be performed to identify workers with kidney disease, which may increase susceptibility to rhabdomyolysis or heat-related AKI.

If the OEM clinician determines that the worker is at increased risk of HRI, the clinician should issue formal work restrictions and/or recommendations. For example, a farmworker with HRI risk factors could be restricted to a 75% work to 25% rest ratio each hour when heat stress levels are above a screening threshold, and the work/rest ratio could increase as heat levels increase.55 Clinicians should also consider issuing temporary work restrictions for new workers during their first 7 to 14 days of heat stress exposure when the risk of fatal HRI is highest. If the worker needs to be on work restrictions only during the hottest month(s), with allowance for modified work and/or reasonable accommodations as deemed appropriate, these avenues should be pursued accordingly.

RTW Issues
Workers who experience HRI may not RTW until completely recovered and measures to prevent a recurrence have been put into place. If restricted duty is available that allows a workers’ biomarkers to return to normal, this should be considered before a full-duty RTW occurs. OEM clinicians can assist with RTW decisions by assessing clinical recovery and evaluating workers’ likelihood of another HRI. US military return-to-activity guidelines utilize physician judgment and acclimatization protocols prior to returning to duty after an HRI.50,56 However, there is limited existing civilian clinical guidance about RTW after an episode of HRI.

At a minimum, clinicians should ensure that workers are asymptomatic and have normal biomarkers (eg, blood tests for kidney and liver function) before RTW. The amount of recovery time varies with the severity of the illness. Workers who experience mild or moderate HRI might be able to RTW the next day or in a few days, with appropriate precautions. Conversely, workers with heat stroke or other organ damage (eg, AKI) need more time to recover. Guidelines for athletes recommend a rest period of at least 7 to 21 days after heat stroke.57

Serum biomarkers such as creatinine, BUN, CK, aspartate aminotransferase, alanine aminotransferase, and lactate dehydrogenase may be elevated acutely in episodes of heat stroke.58 Biomarker abnormalities are also present in other types of HRI, such as heat-related AKI with an elevation of serum creatinine. OEM clinicians should ensure that elevated biomarker returns to normal before clearing a
monitoring protocols that are appropriate for the workforce and workers being assessed.

Physiological monitoring technologies have limitations that clinicians should consider carefully before use. Direct measurements of core temperature (eg, esophageal, intra-abdominal, or rectal) are the gold standard for body temperature assessment, but such techniques are logistically difficult in a work environment and are not readily accepted by workers. On the other hand, peripheral temperature measurements (eg, oral, tympanic, or urine) are easier to perform but are prone to falsely low readings that may underestimate core temperature. Non-invasive physiologic monitoring devices such as smartwatches, wearable hydration sensors, and other wearable heat strain monitors are commercially available. Research to determine the validity and utility of these technologies is ongoing, and there is not yet enough data to recommend any specific device. However, the potential for future ability to monitor workers in a high-heat environment is promising.

Clinician oversight is important to ensure that physiologic monitoring complies with applicable confidentiality and ethics issues. Physiologic monitoring should only be performed if the worker has been informed about the discomfort and the potential risks associated with the measurement technique and consents to such measurements. The supervising OEM clinician should ensure that the measurements present no unacceptable risks of harm or breach of confidentiality.

We acknowledge that there are many controversies regarding HRI as to prevention, monitoring, diagnosis, and treatment. What is presented in this guidance statement is a researched and reasonable approach, the aspects of which should be applicable to all industries and workers. Some of our recommendations are best practices that may be considered cost-prohibitive to small businesses, while some companies have the capability to and have developed more rigorous policies and procedures. We believe that the guidance presented herein will benefit most companies and their workers, and we recommend implementing the best practices for individual industries.

REFERENCES


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30. Organisation of the Army. TRADOC Regula-