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PHYSIOLOGICAL EFFECTS AFTER EXPOSURE TO HEAT: A BRIEF LITERATURE REVIEW

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ABSTRACT

Many employees are exposed to heat stress during their work. Although the direct effects of heat are well reported, the long term physiological effects occurring after heat exposure are hardly described. The present manuscript addresses these issues in the form of a brief literature review. Repeated heat exposure results in heat acclimatization. These physiological adaptations decay gradually afterwards, re-increasing the vulnerability to heat injuries. Repeated heat exposure may lead to kidney damage (related to dehydration) and reduced efficiency of the reproductive system. A history of heat stroke may increase the sensitivity to heat illness. The increased susceptibility possibly indicates an impaired thermoregulatory system resulting from a heat stroke, or a genetic predisposition prior to the first heat stroke.

Keywords: heat, strain, stress, physiology, health, temperature

FIZIOLOŠKI UČINKI PO VROČINSKI IZPOSTAVLJENOSTI: KRATEK PREGLED LITERATURE

IZVLEČEK

Mnogi delavci so pri svojem delu izpostavljeni vročinskemu stresu. Čeprav so neposredni učinki vročine dobro preučeni, so dolgoročne fiziološke spremembe le redko opisane. Članek opisuje tovrstno tematiko v obliki kratkega pregleda literature. Ponavljajoče se izpostavljanje vročini vodi v vročinsko aklimatizacijo, te fiziološke prilagoditve pa začnejo postopoma upadati ter povečujejo občutljivost za vročinske poškodbe. Ponavljajoča se izpostavljenost vročini lahko vodi v ledvične okvare (povezane z dehidracijo) in zmanjšano učinkovitostjo reproduktivnega sistema. Predhodno prebolela vročinska kap (toplotni udar) poveča občutljivost za vročinske obolevnosti. Večja dovzetnost najverjetneje nakazuje na neučinkovitost termoregulacijskega sistema, ki je posledica vročinske kapi ali genske predispozicije pred prvo vročinsko kapjo.

Ključne besede: vročina, napor, stres, fiziologija, zdravje, temperatura

INTRODUCTION

A core temperature in a narrow band around 37 °C is essential for optimal human functioning. This thermal equilibrium can be achieved if heat gain equals heat loss. In the absence of thermal extremes, core temperature is maintained through constriction and dilatation of blood vessels in the skin. In the heat, and particularly during exercise, sweat evaporation contributes substantially to the heat balance. If sweat evaporation is compromised, for instance due to wearing protective clothing, the core temperature increases. High core temperatures result in reduced performance, both physiological (Galloway & Maughan, 1997; Gonzalez-Alonso et al., 1999; Parkin, Carey, Zhao & Febbraio, 1999) and psychological, e.g., on vigilance and complex dual tasks (Ramsey, 1995; Pilcher, Nadler & Busch, 2002; Hancock, Ross & Szalma, 2007). For instance, the physiological strain endured by fire fighters is described elsewhere (Romet & Frim, 1987), in addition, it is established that warm environments can reduced driving performance (Wyon, Wyon & Norin, 1996). If heat strain results in an increased core temperature (hyperthermia) it can lead to mortality and morbidity under healthy, well trained individuals, through exertional heat stroke (Coris, Ramirez & Van Durme, 2004).

Heat strain is common in many occupations as recognized by the American National Institute for Occupational Safety and Health (NIOSH). Due to the rising environmental temperature the occurrence of heat strain is likely to increase in at least some occupations. In fact, the Intergovernmental Panel on Climate Change (IPCC, 2007)

predicts a global temperature rise of 1.1 °C to 6.4 °C within this century, mainly due to the greenhouse effect. Besides global warming can also urbanization play a role in increases in temperature to which people are exposed. For instance, people working in urban environments are exposed to higher levels of heat stress compared to workers in rural areas. This phenomenon is caused by so called urban heat islands, and can account for an increased ambient temperature as large as 8 °C for a city size of about half a million inhabitants compared to a rural area (Roodenburg, 1983).

Most research is conducted in the area of direct effects of heat on health and performance. However, less is known about the long term effect on heat exposure on health. This is relevant since many employees are exposed to heat for prolonged periods in their life, such as workers in the metal melting industry, road construction workers, and military personnel serving in jungle and desert climates for several months. On a national level, governments are introducing new laws making employers more responsible for the health of their employees, in order to minimize the negative effect of work on the general health. The goal of these changes is to reduce sick-leave and thereby increasing productivity. However, the stimulus of governments did not result in increased research activity in the field of health effects after exposure to heat stress; this manuscript will focuses on these effects. Physiological effects during exposure to heat stress are covered elsewhere (see e.g., Sawka & Young, 2005; Cheung, 2010).

PHYSIOLOGICAL EFFECTS AFTER EXPOSURE TO HEAT

Decay of acclimatization

Exposure to heat for a sufficient period results in physiological adaptations known as acclimatization (Nielsen, 1994). Moreover, Nielsen and co-workers conducted a series of extensive studies (Nielsen et al., 1993; Nielsen, Strange, Christensen, Warberg & Saltin, 1997) in which they monitored the acclimation effects to dry and humid heat. In these studies respectively 8 and 12 participants exercised till exhaustion at 50% of the maximum oxygen uptake (VO_2max), in a laboratory kept at 41 °C, 12% relative humidity (RH) and 35 °C, 87% RH. Both studies find an increased sweat rate, increased plasma volume, decreased heart rate and increased time to exhaustion, as a result of acclimation. Understanding the decay of acclimatization will allow for improved reintegration schemes or prescribed heat strain exposure for employees irregularly exposed to warm environments.

It is often thought that most of these physiological adaptations will disappear in the course of one to three weeks (Armstrong & Maresh, 1991; Pandolf, 1998; Astrand, Rodahl, Dahl & Stromme, 2003). However, recently 16 participants underwent a 10 days acclimatization protocol during the UK winter months. The participants were divided into two even groups who carried out an exercise protocol in a warm environment after 12 and 26, for each group separate (Weller, Linnane, Jonkman & Daanen, 2007). The results indicate that many of the advantageous of the adaptation remained after 12 and

even 26 day after completing the acclimatization protocol. Thus, contrary to general belief, heat acclimation decay may take more than a month (Weller et al., 2007; Daanen, Jonkman, Linnane & Weller, in press). Although acclimatization effects are similar for conditions with comparable levels of heat stress (Griefahn, 1997), some studies indicate that acclimatization to dry heat in athletes will persist longer than acclimatization to humid heat (Pandolf, 1998). Acclimatization will also persist longer in physically active individuals (Pandolf, 1998). The latter being intuitive because physical activity is likely to coincide with elevated levels of the core temperature. Since hyperthermia is the trigger for the acclimatization process (Nielsen, 1998) the acclimatization decay will be slower when high levels of physical activity are maintained.

History of heat stroke and thermal physiological function

An elevated core temperature might lead to heat illness (Wexler, 2002), of which heat stroke is the most severe. Heat stroke is life-threatening and trained medical personal should supervise treatment (Wexler, 2002). For health individuals, a core temperature around 40 °C is usually associated with an increased risk for heat stroke (Wexler, 22; Coris et al., 2004). Two types of heat stroke can be differentiated: (i) classic (or non exertional) heat stroke resulting from a high environmental temperatures; (ii) exertional heat stroke which is the result of excessive heat produced due to high metabolic demands. The mechanisms of heat stroke are described elsewhere (Gisolfi & Mora, 2000). Understanding the relationship between the history of heat stroke and the thermal physiological functioning, allows for improved screening of suitable individuals for jobs were heat exposure is inevitable.

Several studies have focused on the effect of a history of exertional heat stroke on the thermal physiological response to heat. One study found such effect (Shapiro et al., 1979), whereas other studies do not confirm these results (Armstrong, De Luca & Hubbard, 1990; Royburt, Epstein, Solomon & Shemer, 1993). Shapiro et al. (1979) examined 9 young men whom suffered from heat stroke 2–5 years earlier and 10 young men without a history of heat illness. They found significant differences in heart rate and rectal temperature between the two groups during exercise (40 and 80 W for 45 and 20 min, respectively) at room temperature (23 °C, 50% RH) and high ambient temperature (40 °C, 50% RH). However, VO_2 was significantly lower in patients who had a history of heat stroke during exercise in a warm environment, this was left unexplained. In addition, no significant difference was found for sweat rate. It was concluded that since the former heat stroke patients generated the same amount of external power (and even had a lower VO_2), their significantly higher body core temperature might indicated an impaired transport of heat from the core to periphery.

Other studies were carried out under comparable conditions (Armstrong et al., 1990; Royburt et al., 1993), and did not find a difference between former heat stroke patients and matched control participants in hear rate and body core temperature during exercise. It is difficult to indicate the basis for this discrepancy; it could be due to differences in anthropometrics, or response time and treatments to which the heat

stroke patients were exposed. The latter are indicated as relevant for the efficiency of recovery (Lew, Lee, Date & Melnik, 2002). It therefore remains unclear if former heat stroke patients are more sensitive to heat after some recovery time. However, the former heat stroke patients used in the above mentioned studies suffered from exertional heat stroke. Interestingly, history of heat stroke is often indicated as a risk factor for heat illness (Gisolfi & Mora, 2000; Coris et al., 2004). Thus, it is unclear if heat stroke causes a degradation of the thermoregulatory system, it is equally likely that former heat stroke patients had a degraded thermoregulatory system beforehand.

Exertional heat stroke is more relevant to a working population than classical heat stroke. However, the latter is not irrelevant due to trend of increasing the retirement age as a response to the recent reduction in economic prosperity combined with the increasing life expectancy. From research done during the 1995 heat wave in Chicago, which resulted in 600 excess deaths, only 52% of the former classic heat stroke patients survived the first year (Dematte et al., 1998). None of these surviving patients (n = 58) recovered completely within a year. The authors concluded that near fatal classic heat stroke is associated with multi-organ dysfunction. However, it is difficult to compare patients suffering from exertional and classic heat stroke, since the former affects healthy exercising individuals and the latter affects the young, the elderly and the sick. Thus, patients suffering from classic heat stroke are less favoured, they have a 50% change not to survive the year following their stroke, and will most likely not recover completely within a year.

Reduced efficiency of the reproductive system

Different organs in the human body exhibit different sensitivities towards heat. For instance, whole body effects of muscle impairment at a given high temperature are likely less severe than brain tissue impairment at the same temperature (Gisolfi & Mora, 2000). Although increased temperature of the reproductive system is unlikely life threatening to the exposed individual, it is likely to affect new life due to its effect on spermatogenesis. In fact, long term difficulties with desired and successful reproduction are likely to be an important factor for mental wellbeing.

Rachootin and Olsen (1983) found that exposure to heat was associated with an increased risk of delayed conception, with a factor 1.8. It was further found that spermatozoa have a greater likelihood for morphological abnormalities. This finding was explained through a deviation away from the optimum testicular temperature ranging from 34–35 °C. Intuitively, these authors identified that warm environment to which the workers were exposed as the cause for the increased testicular temperature. Also other studies confirm this (Baird & Wilcox, 1986; Figa-Talamanca et al., 1992; Mur, Wild, Rapp, Vautrin & Coulon, 1998). However, if assessed, no effect on the female reproductive system was found in the before mentioned studies.

The effects on the reproductive system are mainly exhibited during exposure, with parameters such as spermatozoa quantity, morphology, and function, returning to normal levels soon after exposure. In fact, almost all human populations exhibit seasonal

variation in births (Bronson, 1995; Sharpe, 2000). This is partly explained by seasonal variation in the frequency of conception. Bronson (1995) also recognizes temperature as an important factor for these variations. In fact, 9 months after the summer birth rates are lower compared to 9 months after the winter season, this tends to indicate a recovery of from heat exposure.

Nephrolithiasis

While exposed to heat stress, the human body can produce excessive amounts of sweat, in an attempt to lose heat through sweat evaporation. In fact, sweat rates of 7 l per working day have been reported (Leithead & Lind, 1964). Sweat is produced from intercellular fluid but excreted with a reduced osmolarity. Thus, work in a warm environment intensifies the kidney function aimed at maintaining a stable blood osmolarity. Ingestion of fluids, which is essential for compensating for water loss through sweating, further affects the osmolarity of blood. This mechanism forms the basis for the relationship between exposure to heat and nephrolithiasis (the development of kidney stones) (Vander, Sherman & Luciano, 2001). Understanding the relationship between warm environments and nephrolithiasis is likely to contribute to the reduction of its occurrence.

Borghi et al. (1993) studied 236 employers of a glass plant in Italy exposed to heat (30 °C WBGT) and 165 employers of the same factory not exposed to heat (25 °C WBGT). Urine analysis revealed an increased uric acid (722 vs. 482 mg/l), decreased pH (5.3 vs. 5.6), increased specific gravity (1026 vs. 1021 kg/m) and higher incidence of nephrolithiasis (8.5 vs. 2.4%) for the workers exposed to heat. They concluded that the increased incidence for nephrolithiasis was caused by an insufficient fluid intake (dehydration). This is supported by an epidemiological survey that demonstrated a higher prevalence of nephrolithiasis in populations of hot climates (Parry & Lister, 1975). Another group studied workers exposed to heat and intense sunlight for at least 6 months per year and confirmed a higher incidence of kidney stones (Better, Melamud, Shabtai, Berenheim & Chaimowitz, 1978). A sufficient fluid and salt intake was suggested as a solution to this problem. However, from athletes it is known, that usually voluntarily insufficient fluids are consumed while exercising in a hot environment (Nielsen & Krog, 1994; Murray, 1995). This is likely also the case for employees working in hot environments.

There might also be a link between sunshine and nephrolithiasis. This link is formed through vitamin D, which is generated due to ultraviolet B radiation from sunlight and stimulates the absorption of calcium from the intestines (Holick, 2003). An increased vitamin D production can lead to an increased calcium concentration in the blood and thus to an increased calcium flux through the kidneys. An increased calcium flux through the kidneys coexists with an increased risk of nephrolithiasis (Shekarriz & Stoller, 2002). This hypothesis is postulated by Parry and Lister (1975). However, if vitamin D plays a substantial role in nephrolithiasis, it can easily be prevented by using sunscreen (Holick, 2003).

CONCLUSIONS

This brief literature review focused on physiological effects after exposure to heat. Although limited literature is available on this topic, there is scientific basis for the following events to occur after a multi-day exposure to heat: (i) decay of acclimatization acquired during heat exposure, (ii) reduction in the efficiency of the reproductive system (iii) increased risk for nephrolithiasis. It is undetermined if repeated heat exposure affects the risk for heat stroke.

OUTLOOK

More knowledge is needed to improve our understanding, allowing a better protection of employees. Such future work should not only focus on extending the knowledge on mechanisms that increase health risks, which was the focus in this brief review, but should also consider positive health effects of exposure to heat. For instance, from research on chickens it is known that exposure to heat at a young age increases resistance against heat injuries later in life (Arjona, Denbow & Weaver, 1990; Yahav et al., 1997; Zulkifli, Liew, Israf, Omar & Hair-Bejo, 2003). At present it is unclear if such mechanisms also exist in humans.

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