Does Living and Working in a Hot Environment Induce Clinically Relevant Changes in Immune Function and Voluntary Force Production Capacity?

Wade KNEZ¹*, Olivier GIRARD¹, Sebastien RACINAIS¹, Andrew WALSH¹, Nadia GAOUA² and Justin GRANTHAM²

¹Aspetar, Orthopaedic and Sports Medicine Hospital, Athlete Health and Performance, Qatar ²Aspire Zone Foundation, Qatar

> Received April 8, 2012 and accepted February 6, 2014 Published online in J-STAGE February 28, 2014

Abstract: This study investigated the effect of living (summer vs. winter) and working (morning vs. afternoon) in a hot environment on markers of immune function and forearm strength. Thirty-one healthy male gas field employees were screened before (between 05:30 and 07:00) and after their working day (between 15:30 and 17:00) during both seasons. Body core temperature and physical activity were recorded throughout the working days. The hot condition (i.e. summer) led a higher ($p\leq0.05$) average body core temperature (~37.2 vs. ~37.4 °C) but reduced physical activity (-14.8%) during the work-shift. Our data showed an increase ($p\leq0.05$) in lymphocyte and monocyte counts in the summer. Additionally, work-shift resulted in significant ($p\leq0.001$) changes in leukocytes, lymphocytes and monocytes independently of the environment. Handgrip (p=0.069) and pinch (p=0.077) forces tended to be reduced from pre-to post-work, while only force produced during handgrip manoeuvres was significantly reduced ($p\leq0.05$) during the hot compared to the temperate season. No interactions were observed between the environment and work-shift for any marker of immune function or forearm strength. In summary, working and living in hot conditions impact on markers of immune function and work capacity; however by self-regulating energy expenditure, immune markers remained in a healthy reference range.

Key words: Biomarkers, Thermal stress, Work environments, Fatigue, Workload

Introduction

Human adaptation to varying degrees of heat stress received great attention after the Second World War. Whereas most of the early studies focused on the impact to soldiers¹), various experimentations were also performed to investigate heat acclimation in workers such as mine labourers^{2, 3}).

*To whom correspondence should be addressed.

E-mail: wade.knez@aspetar.com

However, these studies concentrated on performance and work capacity but immune function received little attention. Since this time, experimental studies have demonstrated that both physical⁴⁾ and psychological stress⁵⁾ influence immune function. Almost exclusively this research has been conducted in a laboratory-based environment in response to running on a treadmill or cycling on a stationary bicycle rather than in response to field-based activity. Regardless, the data shows that the extent of the impact on immune function is dependent on the magnitude of the stress experienced by the individual⁶⁾. For example, a passively-

^{©2014} National Institute of Occupational Safety and Health

induced increase in body core temperature has been shown to increase circulating leukocyte numbers⁷). Furthermore, exercise-induced alterations in immune function, are dependent on the intensity and duration of the activity itself in addition to changes in body core temperature^{6, 8–10}).

Relatively few studies have investigated the combined effect of working with the addition of heat stress on immune function. Consensus amongst the limited data shows that there is minimal impact on leukocyte numbers in response to an exercise-induced body core temperature increase of $\leq 1 \circ C^{11-13}$. Due to ethical restrictions in laboratory-based research, investigations are limited in the extent and duration of the heat exposure investigated; however, field situations such as those observed in the mining and fire fighting industries are not subject to these limitations. Therefore, such workers might experience a greater immune response than described in the literature. This is very important to our knowledge-base as physical activity induced changes in immune function have been proposed to contribute to the development of heat stroke¹⁴).

Working in the oil and gas industry requires, among others, well-developed qualities of forearm force in order to successfully perform specific tasks including the use of tools. Decrease in force during isometric contractions of both upper and lower extremities after either passive^{15, 16)} or active¹⁷⁾ heating is evident at a body core temperature of >38.5°C, even if such a decrease seems to be dependent of the muscle considered¹⁸⁾. Although workers in a hot environment are likely to reach such body core temperatures in the afternoon¹⁹, it is still unclear if working outdoors (8–12) h/day) during the summer months will induce a larger alteration in force production capacity compared to a neutral test setting. Indeed, it has recently been reported that working in a hot environment can lead to a higher average body core temperature than resting in neutral or hot environment but not than continuously exercising in neutral environment¹⁹). This study investigated the combined effect of working and living in a hot environment on markers of immune function and forearm strength.

Participants and Methods

A group of 31 non-acclimated Asian healthy male workers $(30 \pm 6 \text{ yr}; 66 \pm 7 \text{ kg}; 165 \pm 6 \text{ cm})$ from oil and gas industry took part in this study after providing their written informed consent. All participants had their immune function and forearm strength assessed (constant temperature of 22–24°C) before (between 05:30 and 07:00) and after a regular work-shift (between 15:30 and 17:00) during a hot

(August; outside temperature: \sim 37°C (range 29–47°C)) and a temperate (January; outside temperature: \sim 17°C (range 10–26°C)) season.

Body core temperature was continuously monitored during the day (1 value per minute, precision 0.01°C) via an ingestible radio-telemetric thermistor (VitalSense[®] recording system, Mini Mitter, Respironics, Herrsching, Germany). Physical activity was continuously monitored by a tri-axial accelerometer (Actical, Respironics Inc., Germany) located on the hip (right side).

Blood, sampled from the antecubital vein, was drawn before and after the work-shift into EDTA and serum vacutainers and analysed to assess changes in the immune cell counts of leukocytes, neutrophils, lymphocytes, monocytes and eosinophils. Analysis was completed and measured on the CELL-DYN 3700 SL analyser (Abbott Diagnostics, Chicago, USA) and adjusted for blood volume changes using the method of Dill and Costill (1974)²⁰⁾.

Maximal handgrip and pinch forces of the dominant forearm were measured using transducers (Captels, St Mathieu de Treviers, France). Participants were vigorously encouraged to perform maximally during two voluntary efforts (4s in duration with 30s of rest between each trial), which were averaged for further analysis.

Participants were also tested during a specific work task consisting of screwing two plates together with nuts and bolts of mixed sizes fitting in holes of mixed sizes randomly positioned on the plates. Participants were instructed to carry out the task as fast as possible and the outcome measure was time.

Statistical analysis

All data was coded and analysed using SPSS (v 18.0). Two-way repeated-measures analysis of variance (ANOVA) were conducted to investigate the main effects of the season (hot vs. temperate) and working day (morning vs. afternoon) along with possible interaction effects. Prior to ANOVA tests, data were screened for normality, homogeneity of variance and presence of outliers. Multiple comparisons were made with the Bonferroni post hoc test. A *p*-value ≤ 0.05 was considered as the cut-off for statistical significance. Cohen's *d* was used to represent effect size with ~0.2 to 0.3 to denote a "small" effect, ~0.5 a "medium" effect and ≥ 0.8 a "large" effect.

Results

Body core temperature, physical activity and blood pressure The average body core temperature during the work-

Parameters	Hot environment			Neutral environment		Reference
	Pre-work	Post-work	ANOVA	Pre-work	Post-work	Range
Leukocytes (cells \times 10 ⁹ .1 ⁻¹)	7.27 ± 1.65	8.17 ± 1.55	W†	7.05 ± 1.59	8.02 ± 1.59	4.0-10.5
Neutrophils (cells $\times 10^9.1^{-1}$)	3.61 ± 1.50	3.89 ± 1.42	N/S	4.01 ± 1.28	3.96 ± 1.09	1.4-6.6
Lymphocytes (cells \times 10 ⁹ .1 ⁻¹)	2.21 ± 0.70	2.76 ± 0.76	E*, W†	1.98 ± 0.49	2.51 ± 0.71	1.1-3.5
Monocytes (cells $\times 10^{9}.1^{-1}$)	0.60 ± 0.13	0.66 ± 0.14	E*, W†	0.54 ± 0.15	0.61 ± 0.17	0.3-0.8
Eosinophils (cells \times 10 ⁹ .1 ⁻¹)	0.55 ± 0.45	0.54 ± 0.40	N/S	0.44 ± 0.34	0.47 ± 0.35	< 0.81

Table 1. Immune cell counts corresponding to changes in environment and work-shift (mean ± SD)

*p≤0.05; †p≤0.001. E=Environmental main effect, W=Working-shift main effect.

Table 2. Forearm strength and job specific skill values corresponding to changes in environment and workshift (mean ± SD)

Parameters	Hot environment		Neutral en	ANOVA	
	Morning	Afternoon	Morning	Afternoon	ANUVA
Handgrip force (kg)	38.91 ± 7.79	38.14 ± 7.24	42.42 ± 7.57	40.91 ± 7.92	E*
Pinch force (kg)	7.51 ± 2.35	7.00 ± 1.71	7.89 ± 2.03	7.60 ± 2.28	
Specific task (s)	168.55 ± 67.68	138.20 ± 38.87	151.48 ± 48.20	132.30 ± 41.21	W#

* p<0.05; #p<0.01. E=Environmental main effect, W=Working-shift main effect.

shift was significantly ($p \le 0.01$) higher in the hot season (37.4 ± 0.20°C) in comparison to a temperate environment (37.2 ± 0.2°C). The normalised (acceleration) physical activity was significantly lower (-14.8%) during the shift in hot than in temperate environment (98 ± 6 vs. 120 ± 7×10^{-3} g, p < 0.01).

Immune function

Table 1 displays immune cell counts corresponding to changes in environment and work-shift. Compared to a temperate environment, living in a hot environment resulted in an increase in lymphocytes $(1.98 \pm 0.49 \text{ vs.} 2.21 \pm 0.70; p \le 0.025)$ and monocytes $(0.54 \pm 0.15 \text{ vs.} 0.60 \pm 0.13; p = 0.035)$. Analysis showed that a work-shift had a significant impact on leukocytes $(7.16 \pm 1.61 \text{ vs.} 8.10 \pm 1.56; p = 0.042)$, lymphocytes $(2.09 \pm 0.61 \text{ vs.} 2.63 \pm 0.74; p \le 0.001)$ and monocytes $(0.57 \pm 0.14 \text{ vs.} 0.64 \pm 0.16; p \le 0.001)$. Data analysis showed no interaction in the combined impact of the change in the environment and the effect of work-shift in any of the markers of immune function investigated.

Forearm strength

Handgrip force was lower during the hot than the temperate season ($p \le 0.05$). Handgrip force (p=0.069) and pinch forces (p=0.077) tended to decrease from pre-to post-work-shifts. Whilst, the speed of the job specific skill of screwing two metal plates together improved ($p \le 0.01$) from pre-to post-work-shift (Table 2). Testing of a specific

task showed that participants were faster in screwing two metal plates together in the afternoon as compared to the morning ($p \le 0.005$, Table 2). There was no significant (p > 0.05) interaction between environment and work-shift for any strength variable or for the specific task.

Discussion

This project was the first to determine the impact of both living and working in a hot environment on markers of immune function and voluntary force production capacity. Compared to a temperate condition, working in a hot environment led to a moderate (d=0.50) increase in average body core temperature ($37.4 \pm 0.20^{\circ}$ C vs. $37.2 \pm 0.20^{\circ}$ C), but with a corresponding decrease in energy expenditure (-14.80%). However, the effects of the workshift on immune markers and forearm force were independent of the environmental conditions.

Effects of environment

The present study showed that despite the large differences in environmental temperature between the hot and the neutral environment, living in these hot conditions (average summer temperature of 37°C, with diurnal temperature up to 47°C) had very little impact on immune cell counts. Data showed that lymphocytes and monocytes increased in the hot environment compared to the neutral environment but this change was minimal and remained within a healthy range. This finding supports previous research that reported the circannual impact on both lymphocytes and monocytes is minor and remains stable across the year²¹). Furthermore, the moderate impact on immune cell counts, in response to the hot environment provides support for previous literature showing minimal impact on immune cell number if body core temperature does not increase by 1 °C^{11–13}). It is clinically relevant to observe that such prolonged heat exposure did not markedly alter the immune function.

Interestingly, handgrip but not pinch force was reduced with heat exposure. Some decrement in maximal voluntary force has recently been observed from the early stage of an increase in central temperature during thumb abduction in a laboratory setting¹⁵⁾. However, the effects of hyperthermia are different for different muscle groups (e.g. knee extensors vs. plantar flexes)¹⁸⁾. The current data suggests that voluntary muscle force production can be affected during the hot environment (i.e summer months); however this is dependent upon the muscle group considered. Interestingly, this decrement was present after a day of work in a hot environment, even when participants were tested in a controlled and neutral environment. Future study needs to investigate the time course of occurrence and decay of such alteration along with the underlying mechanisms (e.g., motivational drop or physiological limitation).

Work-day

Irrespective of whether the participants worked in a neutral or hot environment, an increase leukocyte, lymphocyte and monocyte²²⁾ counts were observed from pre- to postwork shift, but no change was observed in neutrophils. The lack of any significant changes in circulating neutrophils may be due to modest change in body core temperature. Indeed, it has been suggested that both lymphocytes and monocytes are more sensitive to changes in temperature when exercising in the heat than neutrophils²³⁾. Notwithstanding these changes, all of the counts remained within a normal healthy range. It is therefore reasonable to suggest that the modest increases observed in body core temperature (≤ 1 °C) may explain the mild changes in immune cell counts^{12, 13)}.

Previous research in physical activity and environmental stress has been conducted at a fixed work-load; physical activity at a fixed intensity >50% of VO₂peak and has resulted in an average body core temperature in excess of 38.5 °C and an increase in leukocyte count^{11, 24, 25}). However, when participants' final rectal temperature is modest (37.6 °C), little impact on leukocyte count is observed²⁵). In the current project there was no fixed working intensity but rather self-paced as attested by the significant decrement in activity (-13%, $p \le 0.01$). This supports previous research conducted on athletes in a hot controlled environment that has shown that when given the opportunity individuals will adjust their work-rate to maintain thermal homeostasis^{26–28}). It appears that in the present study the participants may have self-regulated their physical activity and thereby enabling them to complete their tasks whilst experiencing modest changes in body core temperature and minimal impact on immune function.

The fact that total leukocytes, lymphocytes and monocytes increased regardless of the environmental conditions gives rise to the possibility that these changes were simply due to a circadian effect. These results provide support for previous research²²⁾ observing a nadir in leukocytes, lymphocytes and monocytes in the morning (~09:00) significantly increasing throughout the day to a peak at approximately ~22:30–23:50 ($p \le 0.0001$, $p \le 0.0001$, p=0.0009 consecutively).

Although not significant, it is also interesting to observe that both handgrip and pinch forces decreased (-2.4% and -4.5% respectively) between morning and afternoon test sessions. None of these alterations were dependent of having performed the work-shift in a hot or temperate environment suggesting that changes in forearm strength may reflect the acute fatigue due to the day of work without any additional alteration specifically triggered by working in hot environment. Despite the reduction in forearm force, performance during the specific task was not impaired, indeed it improved. This shows that maximal voluntary force production capacity is probably not a good indicator for performance of specific tasks in this population.

Conclusion

The present study showed that both living and working in a hot environment impact markers of immune function and forearm strength; however in the absence of hyperthermia these results remained within a healthy range and therefore have limited biological significance. This provides support for previous laboratory-based research; when participants are provided with the opportunity, they will self-regulate their work output and in a field environment this restricts the change in body core temperature below a threshold response resulting in immune cell counts not being clinically significant.

References

- Fox RH, Goldsmith R, Kidd DJ, Lewis HE (1963) Acclimatization to heat in man by controlled elevation of body temperature. J Physiol 166, 530–47.
- Peter J, Wyndham CH (1966) Activity of the human eccrine sweat gland during exercise in a hot humid environment before and after acclimatization. J Physiol 187, 583–94.
- Strydom NB, Wyndham CH, Williams CG, Morrison JF, Bredell GA, Benade AJ, Von Rahden M (1966) Acclimatization to humid heat and the role of physical conditioning. J Appl Physiol 21, 636–42.
- Jonsdottir IH (2000) Special feature for the Olympics: effects of exercise on the immune system: neuropeptides and their interaction with exercise and immune function. Immunol Cell Biol 78, 562–70.
- Khanfer R, Phillips AC, Carroll D, Lord JM (2010) Altered human neutrophil function in response to acute psychological stress. Psychosom Med 72, 636–40.
- Nieman DC, Miller AR, Henson DA, Warren BJ, Gusewitch G, Johnson RL, Davis JM, Butterworth DE, Nehlsen-Cannarella SL (1993) Effects of high- vs moderate-intensity exercise on natural killer cell activity. Med Sci Sports Exerc 25, 1126–34.
- Walsh NP, Whitham M (2006) Exercising in environmental extremes : a greater threat to immune function? Sports Med 36, 941–76.
- Gleeson M (2007) Immune function in sport and exercise. J Appl Physiol 1985 103, 693–9.
- Blannin AK, Robson PJ, Walsh NP, Clark AM, Glennon L, Gleeson M (1998) The effect of exercising to exhaustion at different intensities on saliva immunoglobulin A, protein and electrolyte secretion. Int J Sports Med 19, 547–52.
- Robson PJ, Blannin AK, Walsh NP, Castell LM, Gleeson M (1999) Effects of exercise intensity, duration and recovery on in vitro neutrophil function in male athletes. Int J Sports Med 20, 128–35.
- McFarlin BK, Mitchell JB (2003) Exercise in hot and cold environments: differential effects on leukocyte number and NK cell activity. Aviat Space Environ Med 74, 1231–6.
- 12) Peake J, Peiffer JJ, Abbiss CR, Nosaka K, Okutsu M, Laursen PB, Suzuki K (2008) Body temperature and its effect on leukocyte mobilization, cytokines and markers of neutrophil activation during and after exercise. Eur J Appl Physiol **102**, 391–401.
- Starkie RL, Hargreaves M, Rolland J, Febbraio MA (2005) Heat stress, cytokines, and the immune response to exercise. Brain Behav Immun 19, 404–12.
- 14) Lim CL, Mackinnon LT (2006) The roles of exerciseinduced immune system disturbances in the pathology of heat stroke : the dual pathway model of heat stroke. Sports

Med 36, 39-64.

- Gaoua N, Grantham J, El Massioui F, Girard O, Racinais S (2011) Cognitive decrements do not follow neuromuscular alterations during passive heat exposure. Int J Hyperthermia 27, 10–9.
- 16) Todd G, Butler JE, Taylor JL, Gandevia SC (2005) Hyperthermia: a failure of the motor cortex and the muscle. J Physiol 563, 621–31.
- Saboisky J, Marino FE, Kay D, Cannon J (2003) Exercise heat stress does not reduce central activation to nonexercised human skeletal muscle. Exp Physiol 88, 783–90.
- Racinais S, Girard O (2012) Neuromuscular failure is unlikely to explain the early exercise cessation in hot ambient conditions. Psychophysiology 49, 853–65.
- Racinais S, Fernandez J, Farooq A, Valciu SC, Hynes R (2012) Daily variation in body core temperature using radio-telemetry in aluminium industry shift-workers. J Therm Biol 37, 351–4.
- Dill DB, Costill DL (1974) Calculation of percentage changes in volumes of blood, plasma, and red cells in dehydration. J Appl Physiol 37, 247–8.
- 21) Abo T, Miller CA, Cloud GA, Blach CM (1985) Annual stability in the levels of lymphocyte subpopulations identified by monoclonal antibodies in blood of healthy individuals. J Clin Immunol 5, 13–20.
- 22) Sennels HP, Jørgensen HL, Hansen AL, Goetze JP, Fahrenkrug J (2011) Diurnal variation of hematology parameters in healthy young males: the Bispebjerg study of diurnal variations. Scand J Clin Lab Invest 71, 532–41.
- Peake J (2010) Heat, Athletes, and Immunity. Am J Lifestyle Med 4, 320–26.
- 24) Laing SJ, Blackwell J, Gwynne D, Walters R, Walsh NP (2005) Neutrophil degranulation response to 2 hours of exercise in a 30 degrees C environment. Aviat Space Environ Med 76, 1068–73.
- 25) Mitchell JB, Dugas JP, McFarlin BK, Nelson MJ (2002) Effect of exercise, heat stress, and hydration on immune cell number and function. Med Sci Sports Exerc 34, 1941–50.
- 26) Marino FE (2004) Anticipatory regulation and avoidance of catastrophe during exercise-induced hyperthermia. Comp Biochem Physiol B, Biochem Mol Biol **139**, 561–9.
- 27) Tatterson AJ, Hahn AG, Martin DT, Febbraio MA (2000) Effects of heat stress on physiological responses and exercise performance in elite cyclists. J Sci Med Sport 3, 186–93.
- 28) Tucker R, Rauch L, Harley YXR, Noakes TD (2004) Impaired exercise performance in the heat is associated with an anticipatory reduction in skeletal muscle recruitment. Pflugers Arch 448, 422–30.