HIPA

Heat illness prevention and awareness training
Protecting firefighters from heat related illness

Female firefighters
thermoregulation and health



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Contents

Glossary of terms

Acclimation	Process by which an organism adjusts to an artificial change in environment e.g. heat chamber
Acclimatisation	Process by which an organism adjusts to a change in the natural environment e.g. upon arrival at altitude
Circadian rhythm	Biological process that changes on a roughly 24 hour regular cycle
Cutaneous	Relating to the skin
Dehydration	Process of losing water from the body
Heat dissipation	Type of heat transfer, process of becoming cooler
Heat tolerance	Ability to respond appropriately to thermal challenges placed on the body
Menopause	When a women's menstruation ceases. Typically identified by one year without menstruation.
Menses	Menstruation or period. Part of the menstrual cycle.
Menstrual cycle	Process of ovulation and menstruation in women
Neural	Relating to nerves or the nervous system
Peri-menopause	The transition period into the menopause, where ovaries begin to make less oestrogen and menstrual cycles may become irregular.
Progesterone	Female steroid hormone released by the corpus luteum
Progestin	Synthetic hormone which has similar effect to progesterone
Thermoregulation	Regulate one's own body temperature
Uncompensable	Exposure to heat at which thermoregulatory processes cannot aid cooling effectively and subsequently the body will continue to warm
Vasoconstriction	Narrowing of blood vessels which increases blood pressure
Vasodilation	Widening of blood vessels which reduces blood pressure
Vasomotor	Relating to widening/constriction of blood vessels

Purpose

This information pack is an extension of the Heat Illness Prevention and Awareness (HIPA) training pack, which contains detailed information on thermoregulation, the physiological consequences of firefighting and how to prepare for or recover from severe heat exposure.

The purpose of this document is to educate all members of the fire service on the similarities and differences in thermoregulation and heat tolerance between sexes. Importantly it attempts to explain the female specific issues with thermoregulation during certain menstrual cycle phases and into menopause. At present less than 50% of women firefighters are aware that their menstrual cycle effects their thermoregulation and daily core temperature response, especially to severe heat exposure and exercise. Yet a quarter stated that their menstrual cycle or menopause effected their work (Watkins et al, 2019). It is important to have an understanding of these process and how these may or may not effect female staff.

We realise this may be a taboo subject for many male firefighters, but it is important that they too are aware of all colleagues' welfare, particularly for those managing staff.

Introduction

The body usually maintains a set core temperature (37 ± 0.5°C), which is controlled by the thermoregulatory centre within the hypothalamus (in the brain) (Casa et al., 2015). Numerous systems in the body, such as the cardiovascular system, skin, and central nervous system, work together to try and maintain this core temperature. This process is referred to as thermoregulation. Thermoregulation is challenged by extreme environments, including heat, which can result in decreased aerobic performance (Cheuvront et al., 2010) and put individuals at risk of experiencing a heat illness. Heat illness can range in severity from mild (heat cramps) to fatal (heat stroke).

The most effective heat loss mechanism in humans is the evaporation of sweat (Brotherhood, 2008). The increase in sweating when exercising in the heat is brought about by increased core temperature and skin temperature from the environmental conditions (Brotherhood, 2008). In temperatures above approximately 35°C, skin temperature is typically lower than air temperature, therefore cooling can only occur through evaporation of sweat (Eglin, 2007). When the environment is humid water vapour in the air can prevent a positive gradient between the air and skin, therefore reducing evaporative heat loss. Beneath personal protective equipment (PPE) there is minimal air movement, meaning evaporative heat loss is limited (Havenith, 2003). PPE layers can also create microenvironments where temperatures have been measured up to 60°C (Eglin, 2007). When exercising in the heat, with the added weight of PPE, and reduction in evaporative heat dissipation, core temperatures can exceed 40°C leading to exertional heat illnesses (Casa et al., 2015).

Sex is a predisposing factor for heat related illness with research indicating that females are less tolerant of exposure to hot environments than males. However, 39% of female firefighters' report having had a heat related illness (Watkins et al, 2019), similar to that reported across all US firefighters. Of these US heat illnesses, one third resulted in hospitalisation (Bach, Maley, Minett & Stewart, 2018). It is acknowledged that females are more challenged under heat stress compared to males, due to alterations in sweat response (Gagnon and Kenny, 2011). Sweating starts later in females, as a higher core temperature and skin temperature is needed to stimulate sweating (Bar-Or, 1998). Females also have a smaller number of sweat glands activated and the distribution of sweat is also different between sexes (see Figure 1). This means that the sweat response is not as widely dispersed over the body.

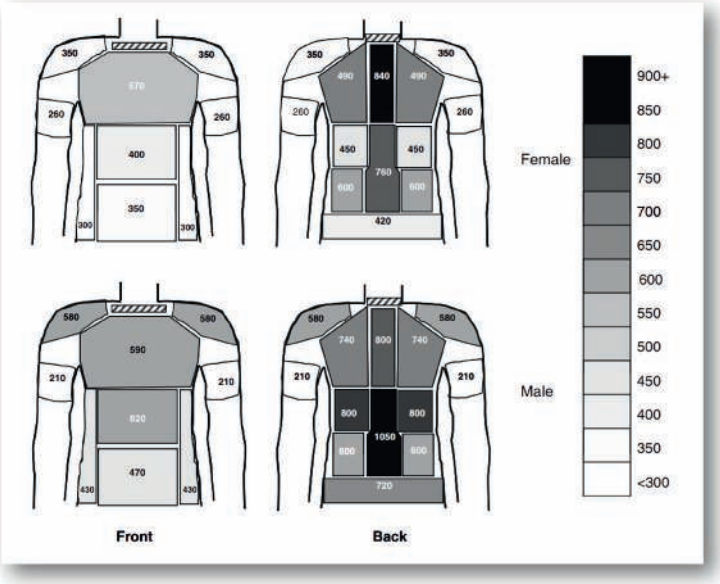

Figure 1. *Differences in sweat distribution on the front and back torso areas between male and female runners. Sweat rate rounded to the nearest 10g (Havenith et al., 2008).*

Females also experience fluctuations in hormonal releases until menopause and these cause regular cyclic changes in a number of physiological parameters, which may alter heat tolerance (Charkoudian and Stachendeld, 2016). Additionally, female body composition is different to males, with females typically having a greater body fat percentage than males of a similar body mass. This means generally women will have a lower body surface area to body mass ratio, resulting in a faster rise in core temperature as a result of poorer skin surface heat dissipation. Musculoskeletal injuries of the upper and lower limbs and back were reported in 9-23% of female firefighters. This is similar to that of male firefighters suggesting there is no difference in risk (Watkins et al, 2019). The same work identified limited access to female specific PPE in some regions of the UK and world. It is important for all staff to get appropriately fitting kit to reduce the risk of injury and physiological burden during firefighting activities.

Menstrual cycle effects on thermoregulation

Thermoregulation and core temperature are altered by the menstrual cycle. The menstrual cycle lasts approximately 28 days and is made up of various phases, throughout which hormones are released in coordination with the female reproductive system (Wadikar and Bhandarkar, 2017). Typically, the follicular phase occurs from day 1 to day 14 and is followed by ovulation. The luteal phase then occurs from ovulation through to the end of the cycle (typically day 28) when menses and the follicular phase begins again (Figure 2, Reed and Carr, 2000). The exact duration of each phase can vary between women.

Hormonal changes are associated with the different phases of the cycle (Grucza et al., 1993; (Marsh and Jenkins, 2002, Figure 2), which could alter thermoregulation (Kaciuba-Uscilko and Grucza, 2001). The gonadrotropin-releasing hormone is produced by the hypothalamus and controls the release of the follicle stimulating hormone and luteinising hormone from the anterior pituitary gland. These are the hormones that are responsible for regulating the release of oestrogen and progesterone. Oestrogen, which is mainly released from ovarian cells (known as follicles), increases towards a peak before ovulation and remains slightly elevated during the luteal phase (Marsh and Jenkins, 2002). Progesterone levels are low in the follicular phase and increase throughout the luteal phase (Marsh and Jenkins, 2002). Alone, oestrogen causes reduced body temperatures and changes in sweating and skin blood flow (Charkoudian, 2003). Progesterone causes increases in vasoconstriction, reductions in vasodilation and alters thermoregulatory control throughout the menstrual cycle (Chakoudian, 2003). The progesterone increase in the luteal phase leads to increased resting core temperature (Marsh and Jenkins, 2002; Charkoudian and Stachendeld, 2016). This higher set-point for resting core temperature in the luteal phase may be due to the altered firing of neurons in the preoptic region of the anterior hypothalamus caused by the higher progesterone levels (Marsh and Jenkins, 2002).

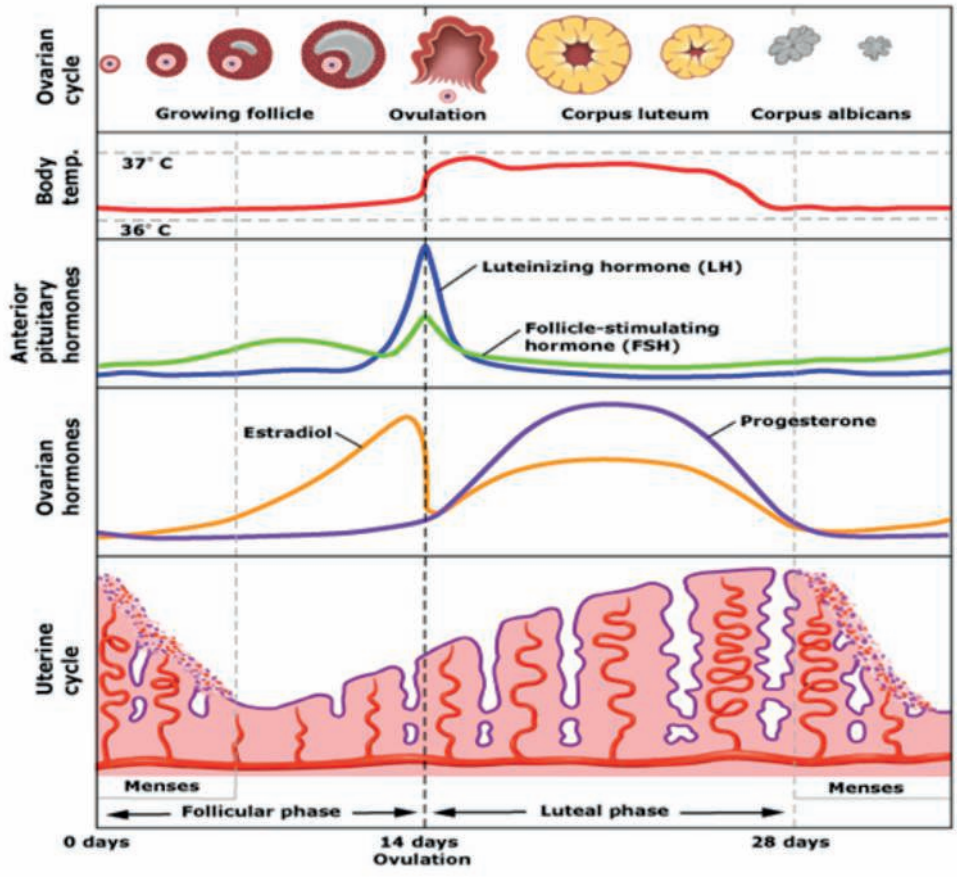

Figure 2: *Menstrual cycle hormonal changes, adapted from Carr & Wilson (1987).*

Menstrual cycle effects on thermoregulation (continued)

During exercise in heat stress environments increased skin temperatures (Lee et al., 2014) and heart rate (Lei et al., 2017) have been reported in the luteal phase. The sweating threshold temperature is also greater in the luteal phase, meaning the start of sweating is delayed and will occur at a higher temperature (Charkoudian and Stachendeld, 2016). Sweat rate itself is lower in the luteal phase than the follicular phase (Lee et al., 2014). The alteration in sweat threshold and rate may be a consequence of lower concentrations of progesterone acting on warm-sensitive neurons in the preoptic region of the anterior hypothalamus in the brain (Kolka and Stephenson, 1989).

The greater resting core temperature in the luteal phase may also persist into exercise performance and could lead to increased subjective feelings of exertion and a reduced performance (Sims and Heather, 2018). Core temperature increases were 0.3 - 0.5°C greater during days 19 - 22 of the cycle (mid-luteal phase), when exposed to temperatures above 32°C with 30-60% relative humidity, compared with days 2 - 5 (early-follicular phase) (Tengalia et al, 1999). Hayashi et al (2012) also demonstrated higher core and skin temperatures in the luteal compared to follicular phase, during 45 minutes of hot, moderate intensity exercise (Figure 3). During a heat tolerance test core temperatures have also been reported to be 0.19 ± 0.31°C greater in the luteal phase (Yanovich et al 2019). Ladies have also been reported to fatigue quicker during exercise in the heat in the luteal phase than in the follicular phase (Janse de Jong et al., 2012).

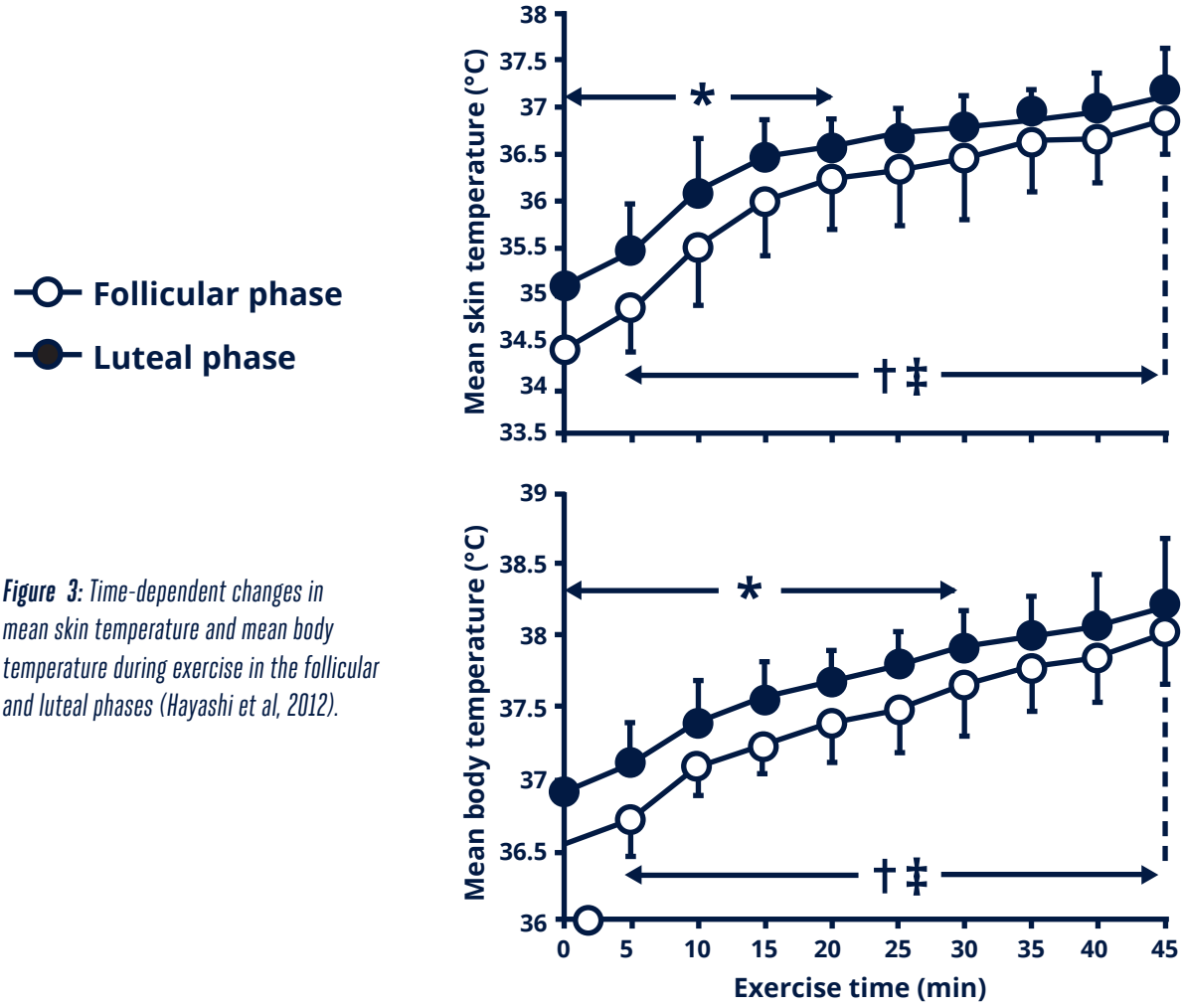

Figure 3: *Time-dependent changes in mean skin temperature and mean body temperature during exercise in the follicular and luteal phases (Hayashi et al, 2012).*

Menstrual cycle effects on thermoregulation (continued)

In contrast, Notely et al (2018) found no differences in exercise duration or core temperature between the mid-luteal, early-follicular and late-follicular phases when exercise at different severities in the heat (Figure 4), suggesting that the menstrual cycle may have no consequence on thermoregulation. Similarly, Kuwahara et al (2005) demonstrated that trained individuals (VO_2max 49 ± 1ml.kg^{-1}.min^{-1}) showed no difference in core temperature between menstrual phases, while untrained individuals (VO_2max 36 ± 1ml.kg^{-1}.min^{-1}) demonstrated significantly raised core temperatures in the mid-luteal phase similar to other work. This was also found to be the case Lei et al (2017).

Whilst research supports the occurrence of some thermoregulatory differences between the different phases on the menstrual cycle, there is conflicting evidence on the impact that this has on the response to exercise in the heat. It is suggested that individuals who maintain a high cardiovascular fitness level may see a less pronounced or possibly no difference in heat tolerance between phases.

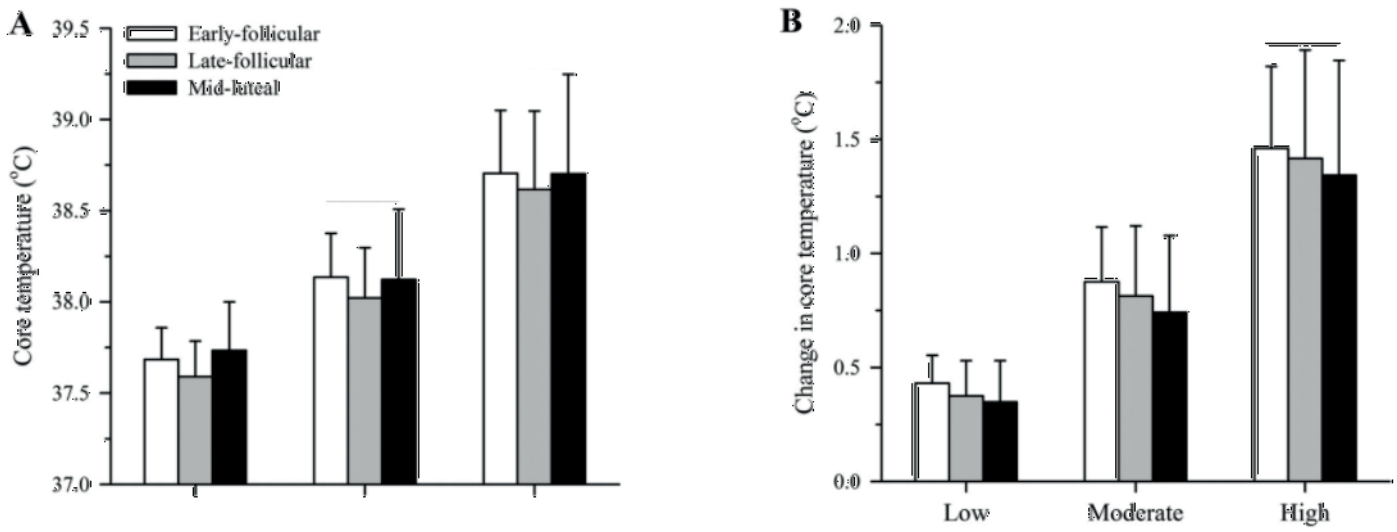

Figure 4: *Core temperature (A) and core temperature change (B) in young, non-endurance trained women (n 12) within the early-follicular (open bars), late-follicular (grey bars), and mid-luteal (closed bars) phases of the menstrual cycle during three 30-min bouts of cycling at increasing fixed heat loads of ~250 (Low), ~340 (Moderate), and ~430 W (High) {equivalent to low intensity [40% peak oxygen uptake (VO_2peak)], moderate-intensity (55% VO_2peak), and high-intensity (70% VO_2peak) exercise, respectively} in hot, dry conditions (40°C, 15% relative humidity). Data (means SD) are averages obtained during the final 5 min of each exercise bout. †Significantly different from Low. ‡Significantly different from Moderate (Notely et al, 2018).*

Contraceptives and thermoregulation

Oral contraceptive pills include oestrogen and/or progesterone which prevent ovulation from occurring. As the natural fluctuations of these hormones may be involved in altered thermoregulation, taking synthetic levels of the hormones may also result in small changes in thermoregulation.

Females who use oral contraceptives have been found to have a 0.2°C lower resting core temperature during the time they are taking their pill in comparison to the no pill week (Tengalia et al, 1999). Following exercise, no difference has been noted in core temperature responses between users and non-users (Tengalia et al, 1999, Figure 5). It is suggested that during the luteal phase, women taking oral contraceptives may have a greater capacity for heat tolerance than non-users, due to the change in resting core temperature (Cheung, 2000). Sometimes in research authors will refer to menstrual stages while taking the oral contraceptives as quasi-EF and quasi-ML, due to lack of hormone fluctuations. New unpublished data from the University of Brighton (Figure 6) also shows core and skin temperatures to be slightly lower in individuals using oral contraceptives.

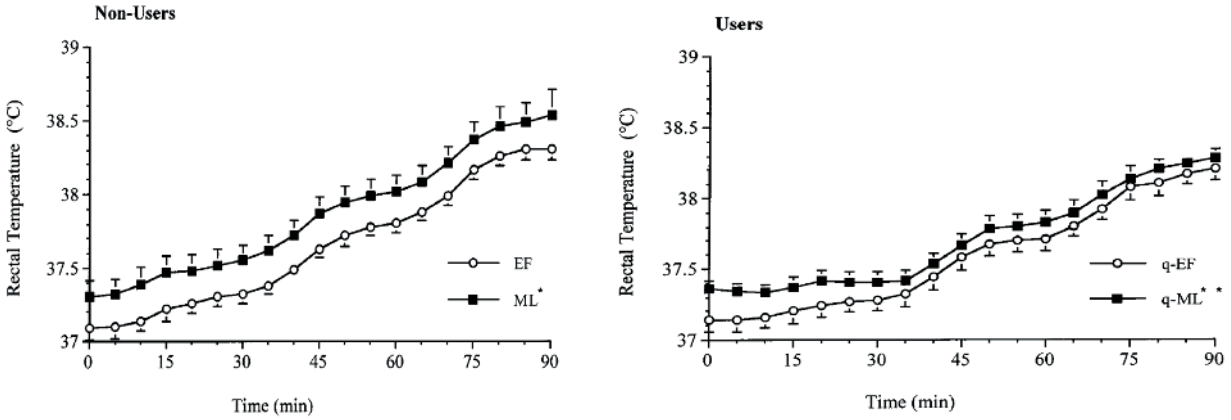

Figure 5: *Rectal temperature in users (n=9 to 75 min, n=8 to 90 min) and non-users (n=9 to 85 min, n=7 to 90 min) of oral contraceptives for the EF (open circle) and ML (closed square) phases of the menstrual cycle. A quasi-EF (open circle) and quasi-ML (closed square) phase was assumed for the users. Values are the mean (SE) (Tengalia et al. 1999).*

However, for long term users of oral contraceptives a higher core temperature, and skin temperature has been found after approximately 22 minutes of exercise in the heat and humidity (Minahan et al., 2017). While Lei et al. (2017) and Tenaglia et al. (1999) found no difference in sweat rates or skin temperatures between those on oral contraceptives and those in the mid-luteal phase.

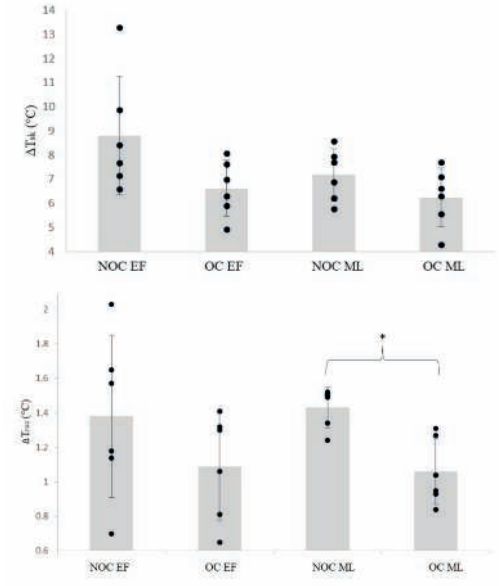

Figure 6: *Change in core and skin temperatures during a 40 min heat tolerance test on female firefighters at Early-follicular (EF), Mid-luteal (ML) and with no oral contraceptive (NOC) and with contraceptives (OC).*
Tsk = Skin Temperature, Tcore = Rectal core temperature.
Black dots show individuals data.
Bars show standard deviation.
**denotes significant difference between the two trials.*

Summary table of relevant research

Table 1. *Non-exhaustive summary of studies on female thermoregulation and heat tolerance*

Study	Participants	Protocol	Groups	Main findings
CHARKOUDIAN & STACHENFELD, 2016 REVIEW	No oral contraceptives.	Light clothes.	Follicular v Luteal	• Baseline Tcore - Luteal ⟩ Follicular • Tcore Threshold for sweating and vasodilation - Luteal ⟩ Follicular
GRUCZA ET AL., 1993	n = 20. Oral contraceptives and no oral contraceptives. Mean age 21.3 ± 0.6 yrs and 22 ± 1.7 yrs.	20 min rest + 45 min cycle at 50% VO_2 peak. 24°C, 50% RH. Light clothes.	Follicular (5-8) v Luteal (18-24) and Oral contraceptives v No oral contraceptives	• Oral Contraceptive - Tcore baseline - Luteal ⟩ Follicular - ΔTcore no difference Luteal v Follicular - ΔTsk no difference Luteal v Follicular - Sweat rate Luteal ⟩ Follicular • No Oral Contraceptive - Tcore baseline Luteal ⟩ Follicular - Δ Tcore Follicular ⟩ Luteal - Tsk baseline no difference - ΔTsk - Follicular ⟩ Luteal
JANSE DE JONG ET AL., 2012	n = 12. No oral contraceptives. Mean age 23.7 ± 4.1 yrs.	60 min cycle at 60% VO_2 peak + incremental test to exhaustion. 20°C, 45% RH and 32°C, 60% RH. Light clothes.	Hot v Temperate and Early-follicular (3-6) v Mid-luteal (19-25)	Tcore baseline & HR baseline - Luteal ⟩ Follicular • Temperate - No difference in performance Luteal v Follicular - Tcore submax exercise Luteal ⟩ Follicular • Hot - Time to fatigue Follicular ⟩ Luteal - Tcore submax exercise Luteal ⟩ Follicular - ΔTcore no difference - Final Tcore no difference - HR & RPE submax exercise - Luteal ⟩ Follicular - HR at exhaustion no difference
KOLKA & STEPHENSON 1989	n = 7. No oral contraceptives. Mean age 26.6 ± 3.6 yrs.	180 min passive exposure + ~ 9 min cycle at 80% VO_2 peak in 50°C, 14% RH and 20 min passive exposure + 35 min cycle at 85% VO_2 peak in 35°C, 25% RH. Light clothes.	Follicular (4-7) v Luteal (19-22)	• T-oes baseline Luteal ⟩ Follicular • T-oes threshold for sweating Luteal ⟩ Follicular • Tsk at 50°C Luteal ⟩ Follicular
KOLKA & STEPHENSON 1997	n = 5. No oral contraceptives. Mean age 27.6 ± 4.4 yrs	30 min cycle at 80% VO_2 peak. 35°C, 22% RH. Light clothes.	Early-follicular (3-6) v Mid-luteal (19-22)	• T-oes at rest and during exercise - Luteal ⟩ Follicular • Tsk at rest - Luteal ⟩ Follicular • HR baseline no difference
KUWAHARA ET AL., 2005	n = 17. No oral contraceptives. Trained and untrained. Mean ages 22.1 ± 0.3 yrs and 20.0 ± 0.4 yrs.	30 min cycle at 50% VO_2 peak. 25°C, 45% RH. Light clothes.	Trained v Untrained and Mid follicular (6-9) v Mid-luteal (19-22)	– T-oes baseline and exercising during Luteal phase - Untrained ⟩ Trained - HR and Tsk no difference - Untrained v Trained • Trained Only - T-oes, ΔT-oes and sweat response no difference Luteal v Follicular • Untrained Only - Baseline T-oes - Luteal ⟩ Follicular - Toes during exercise and ΔT-oes - Luteal ⟩ Follicular - Sweat Rate - Follicular ⟩ Luteal
LEE ET AL., 2014	n = 8. No oral contraceptives. Inactive. Mean age 25.0 ± 1.9 yrs.	40 mins passive heat exposure. 41°C, 21% RH. Light clothes.	Follicular (6-9) v Luteal (21-24)	• Tsk baseline & through heat exposure Luteal ⟩ Follicular • Sweat Rate - Luteal ⟩ Follicular

Summary table of relevant research (continued)

Study	Participants	Protocol	Groups	Main findings
LEI ET AL., 2017	n = 10. No oral contraceptives. Well trained. Mean age 34 ± 9 yrs.	12 mins fixed intensity + 30 mins self-selected intensity cycle. 29°C, 81% RH. Light clothes.	Early-follicular (3-6) v Mid-luteal (18-21)	• Tsk, local Sweat Rate, exercise performance, TS, RPE no difference • Tcore at rest & exercising Luteal ⟩ Follicular • ΔTcore Follicular ⟩ Luteal
MARSH & JENKINS, 2002 REVIEW	No oral contraceptives.	Light clothes.	Follicular v Luteal	• Tcore baseline & exercising in ambient & hot - Luteal ⟩ Follicular • Sweat Rate - Luteal ⟩ Follicular • Tsk baseline and exercising - Luteal ≥ Follicular • HR, RPE baseline & exercising no difference
MINAHAN ET AL., 2017	n = 16. Oral contraceptives and no oral contraceptives. Recreationally active. Mean age 22 ± 4 yrs.	52.5 mins passive exposure + 90, 135 & 180% lactate threshold. 22°C and 35°C. RH not given. Light clothes.	Oral contraceptives (active pill phase) v No oral contraceptives (2-6)	•T-oes baseline - Oral Contraceptives ⟩ No Oral Contraceptives • Final Toes - no difference • HR - no difference • Final RPE - Oral Contraceptive ⟩ No Oral Contraceptives • Skin blood flow elevated for longer in Oral Contraceptives v No Oral Contraceptives
NOTLEY ET AL., 2018	n = 12. No oral contraceptives. Active. Mean age 21 ± 3 yrs.	30 min at 40, 55 & 70% VO_2 peak + 15 mins rest. 40°C, 15% RH. Light clothes.	Early-follicular (~4) v Late follicular (~10) v Mid-luteal (~22)	• Toes baseline Luteal ⟩ Follicular • ΔT-oes, Tsk & HR no difference
PIVARNIK ET AL., 1992	n = 9. No oral contraceptives. Active. Mean age 27.2 ± 3.7 yrs.	60 min cycle at 65% VO_2 peak. 22°C, 60% RH. Light clothes.	Mid follicular (~7) v Mid-luteal (~21)	•Tcore baseline & final Luteal ⟩ Follic lar • HR & final RPE Luteal ⟩ Follicular • Sweat Rate & Tsk no difference
STEPHENSON & KOLKA, 1985	n = 4. No oral contraceptives. Mean age 29.5 ± 4 yrs.	30 min cycle at 60% VO_2 peak. 35°C, 1.73kPa H20 vapour pressure. Light clothes.	Follicular (4-7) v Luteal (18-24)	• Toes & HR baseline - Luteal ⟩ Follicular • HR exercising no difference • T-oes threshold for sweating & vasodilation - Luteal ⟩ Follicular
SUNDERLAND & NEVILL, 2003	n = 15. Oral contraceptives and no oral contraceptives. Well trained. Mean age 20.3 ± 0.4 yrs.	High intensity intermittent running to exhaustion. 31°C, 23% RH. Light clothes.	Mid follicular (~7) v Mid-luteal (~21) and Oral contraceptives v No oral contraceptives	•No Oral Contraceptive - Tcore, HR, SR, RPE no difference Luteal v Follicular • Oral Contraceptive – Tcore - Luteal ⟩ Follicular - HR, SR, RPE no difference Luteal v Follicular
TENAGLIA ET AL., 1999	n = 18. Oral contraceptives and no oral contraceptives. Recreationally active. Mean ages 23.3 ± 1.9 yrs and 23.4 ± 0.7 yrs.	75 - 225 minutes intermittent light exercise. 40°C, 30% RH. NBC clothing.	Oral contraceptives v No oral contraceptives and Early-follicular (2-5) v Mid-luteal (19-22)	• Oral Contraceptive & No Oral Contraceptive - Tcore baseline & exercising - Luteal ⟩ Follicular - HR, Sweat Rate no difference • No Oral Contraceptive - ΔTcore Follicular ⟩ Luteal - Final Tcore no difference - Tsk baseline & exercising Luteal ⟩ Follicular • Oral Contraceptive - ΔTcore & Tsk no difference

Bracketed numbers denote days of the menstrual cycle. HR = heart rate, RPE = rate of perceived exertion, TS = thermal sensation, Tcore = rectal core temperature, T-oes = oesophageal temperature, Tsk = mean skin temperature, NBC = nuclear, biological and chemical

Menopause and thermoregulation

The menopause occurs when the ovaries stop producing female reproductive cells and is inevitable in all women (Barlow et al., 2005). Menopause has been reported to occur around the age of 51 years, with 17% of female firefighters now reporting to either be peri-menopausal or menopausal (Freedman and Blacker, 2002; Watkins et al, 2019). Peri-menopause occurs as ovarian functions begins to decline with large variation of abrupt increases or decreases of oestrogen and a rise in follicle stimulating hormone, indicating a degree of feedback failure from ovarian hormones (Bachmann, 2005; Barlow et al., 2005). This instability of oestrogen is thought to effect females, physically via vasomotor symptoms (hot flushes, night sweats) and sleep disturbances, psychologically (mood disturbance, anxiety, irritability, memory, low libido) and somatically (fatigue, aches, pains) (Shaver and Paulsen, 1993; Nelson et al., 2005; Cohen et al., 2006). When oestrogen levels are depleted and consequently menstruation stops, the final menses has occurred (Barlow et al., 2005). Female firefighters that had gone through the menopause identified hot flushes, fatigue, mood swings, heavy/irregular bleeding and memory loss as the main five issues effecting their work during menopause (Watkins et al, 2019).

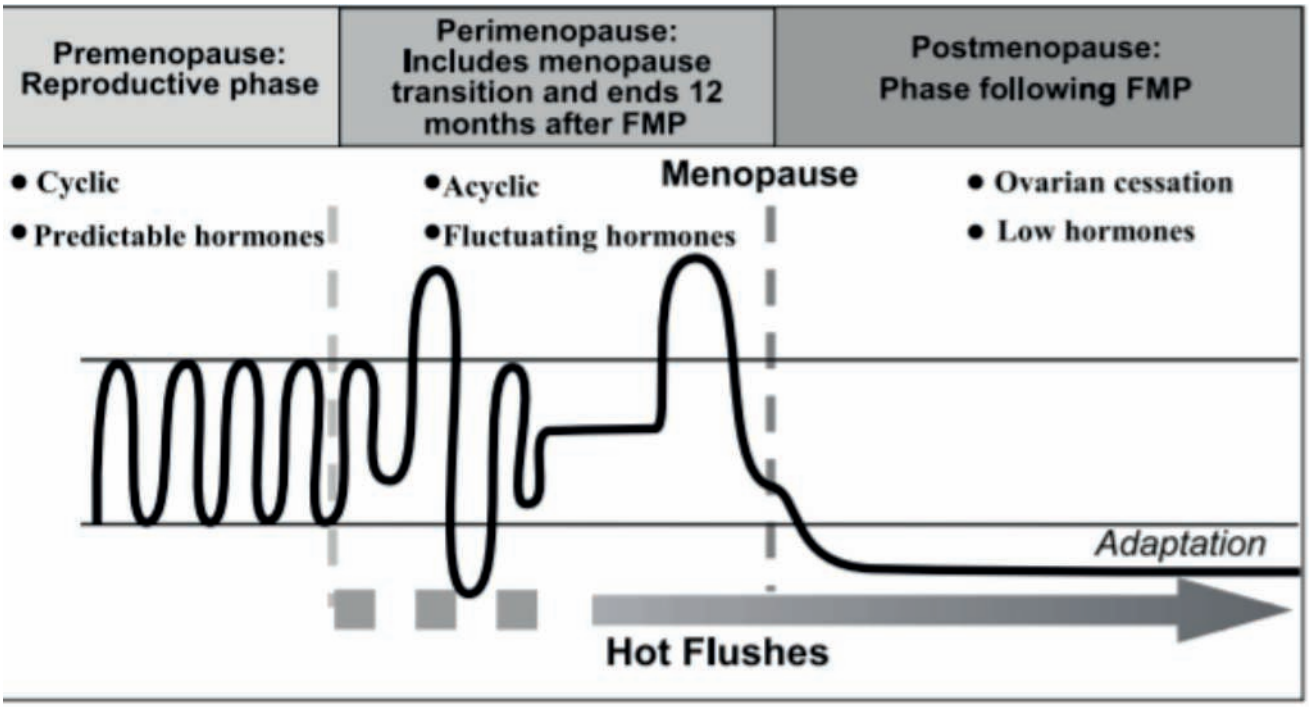

Figure 7: *Relationship between a woman's reproductive phases, oestrogen and the occurrence of hot flushes (Deecher and Dorries, 2007).*

Ladies who have a hysterectomy may also experience menopausal symptoms; a hysterectomy is when the uterus, and in some cases also the cervix and ovaries are surgically removed. Depending on the extent of the surgery, ladies may immediately experience the menopause (referred to as surgical menopause) or be likely to experience an earlier onset of menopause, regardless of age (Farquhar et al., 2005; Moorman et al 2011). Some research also suggests that symptoms, such as hot flushes, night sweats and mood changes, may be more severe or occur for longer durations of time in ladies who have had a hysterectomy (Wilson et al 2016).

Menopause and thermoregulation (continued)

Symptoms such as hot flushes and night sweats are the most common menopausal symptoms and occur in around 80% of women (Deecher and Dorries, 2007). These flushes tend to be associated with an acute rise in skin temperature, peripheral vasodilation and an increase in heart rate (7–15 b.min-1) (Sturdee et al., 1978; Freedman, 2014). Hot flushes begin with significantly higher core temperature (36.82 ± 0.04°C) when compared to non-flush periods (36.70 ± 0.05°C) and it is recognised that hot flushes are preceded by a significant increase in core temperature (Freedman et al., 1995, Figure 8). When core temperature increases above the thermoregulatory neutral zone a heat dissipation response is triggered (Freedman and Blacker, 2002). In symptomatic women, the temperature limits narrow from 0.4°C to 0.0°C due to the shivering threshold increasing and the sweating threshold lowering (Freedman et al., 1995; Freedman, 2001). Therefore, if core temperature reaches the threshold, hot flushes (sweating and peripheral vasodilation) could occur until core temperature returns to the thermo-neutral zone (Freedman, 2014). This is supported further with hot flushes having a circadian rhythm, peaking at 1800 hours, which is on average where our core temperature peaks (Freedman et al., 1995; Waterhouse et al., 2005). However, a study by Jones et al (2019) demonstrated that only half of recorded hot flushes were preceded by a core temperature rise. Authors suggested that this may be a result of neural activation or fluctuations in the thermo-neutral zone. Although core temperature is seen to fluctuate during the menopause it has been identified that core temperature is significantly lower in post-menopausal women (0.25 ± 0.06 °C) when compared to pre-menopausal women.

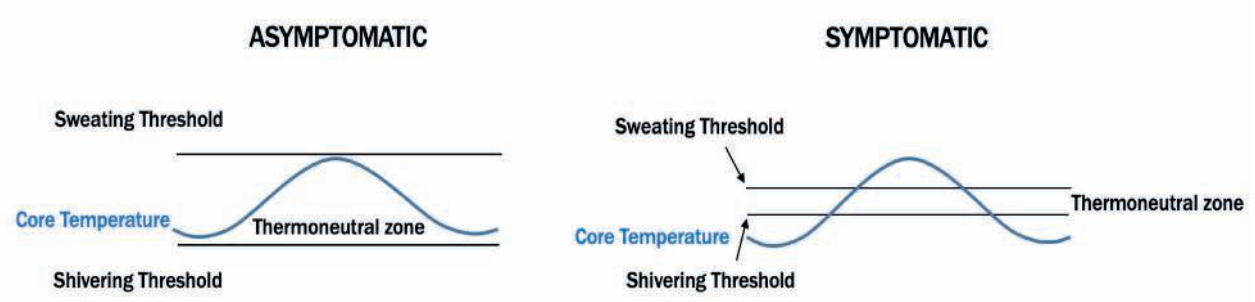

Figure 8: *Small core body temperature elevations acting within a reduced thermoneutral zone trigger hot flushes in symptomatic postmenopausal women (Freedman, 2005).*

Vasomotor symptoms have been seen to decrease by 85% when using hormone replacement therapy (HRT; oestrogen/progestogen) (Bcerug et al., 1998). It has been reported that heart rate and core temperature (~0.5°C) can be significantly reduced when using HRT (oestrogen) (Tankersley et al., 1992). However, this data has only been collected on a small number of participants (n=5) and therefore is hard to generalise to a wider population. HRT has also been noted to cause a significant increase in the core temperature sweating threshold (37.98 ± 0.09°C to 38.14 ± 0.09°C) along with reduced hot flushes over the 3 hour data collection period (1.4 ± 0.5 to 0.6 ± 0.6 hot flushes) due to the significant changes in hormone balance (Freedman and Blacker, 2002). Therefore, by increasing the core temperature sweating threshold the fluctuations in core temperature do not reach the threshold and in turn do no cause heat dissipation responses associated with hot flushes (Freedman and Blacker, 2002). There is some research that suggests HRT raises cancer risk (Beral, 2003; Brown et al. 2015), so individuals considering the use of HRT should consult with their GP.

Although no studies appear to have investigated severe heat exposure or firefighting tasks on menopausal women, it would be more than reasonable to suggest that firefighting in peri-menopause is likely to induce a hot flush as a result of quickly increasing core temperatures. However, there is no suggestion that this actually increases the risk of suffering with heat illness, but rather the perceptual sensation of feeling hot. Post menopause, there is nothing to suggest that heat tolerance is effected and it is likely that heat tolerance may well have adapted and improved. Subsequently, it may well be appropriate to consider the needs of peri-menopausal staff regarding available cooling methods immediately post firefighting.

Effects of ageing on bone and fitness

With increasing age, physiological changes including the loss of muscle and bone mass, contribute to a greater risk of falls and consequently fractures (Padilla Colón et al., 2018). Ageing correlates with a decline in bone mass and an increased risk of hip and spine fractures (Leite et al., 2010). There is also a decline in muscle and bone strength with ageing (Leite et al., 2010).

Osteoporosis is a condition of structural deterioration of bone tissue, resulting in decreased bone strength and high risk of fractures (DeFina et al., 2016, Figure 9). It is estimated to affect over 61 million men and women over 50 years old in the United States, by 2020 (DeFina et al., 2016). Osteoporosis has a higher prevalence in women compared to men due to decreasing oestrogen levels, resulting in a rapid loss of bone mineral density in the first 3-5 years after menopause (DeFina et al., 2016). This deficiency of oestrogen leads to bone loss from a year pre-menopause, through increasing bone break down (Snyman, 2014). On average there is a 10% decrease in bone mineral density throughout the menopausal transition. This risk of osteoporotic fracture doubles every ten years after the age of fifty (Snyman, 2014). Women with osteopenia (bone mass loss) are at greatest risk of fractures and thus should be medically assessed more regularly (Snyman, 2014).

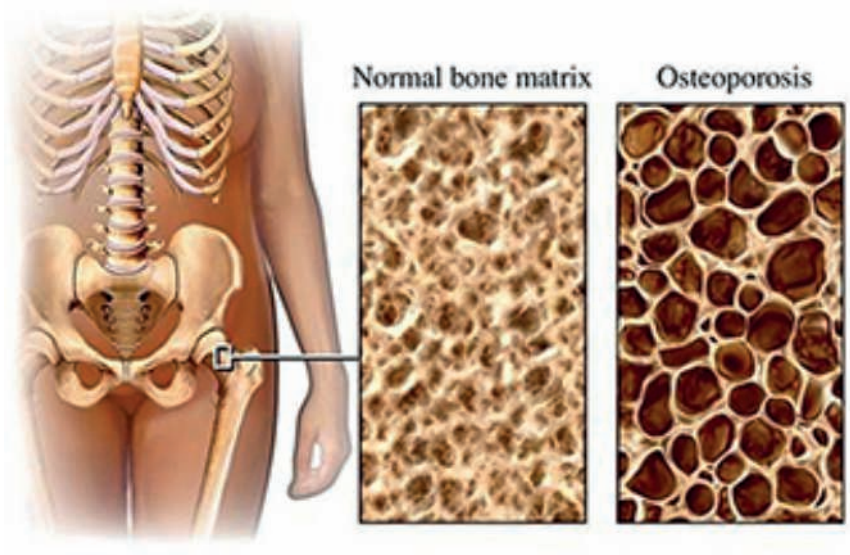

Figure 9: *Normal and osteoporosis bone matrix (National Kidney Foundation, 2019).*

Prevention of osteoporosis includes appropriate nutrition and healthy lifestyle choices throughout life (Synman, 2014). It has been shown that exercising for early post-menopausal women will reduce the decline in bone mineral density (Kemmler et al., 2016). Exercising regularly, even if exercises are adapted, is shown to be effective after menopause to combat osteoporosis (Kemmler et al., 2016). Weight bearing exercise is beneficial in both pre- and post-menopausal women to enhance bone health (Roush, 2011), with regular muscle-strengthening and weight-bearing exercise recommended for treatment and prevention of osteoporosis (DeFina et al., 2016). In post-menopausal women, these exercises are also shown to reduce bone mineral density loss (DeFina et al., 2016). Resistance training results in increased bone mass and helps to reduce alterations from osteopenia (Leite et al., 2010). Evidence suggests that preservation of bone mineral density from resistance exercises was effective at the lumbar spine and femoral neck (Zhao, 2015). Combinations of weight-bearing and resistance exercises have resulted in improved bone mineral density at the hip and spine in post-menopausal women (Zhao, 2015). However, for this population the optimal loading levels during resistance training are still under debate (Leite et al., 2010).

Effects of ageing on bone and fitness (continued)

Bone loss resulting from age is characterised through calcium loss from bone (Tang et al., 2007). Vitamin D intake at sufficient levels is important to ensure bone health. Vitamin D plays a crucial role in muscle strength and balance but needs sufficient calcium intake for proper absorption (Roush, 2011). For women over 50 it is recommended to consume a minimum of 1200mg elemental calcium a day (National Institutes of Supplements, 2019) and this should be primarily from food sources (Roush, 2011). Foods containing vitamin D include milk, grain products and others (Moore et al., 2004). For lowering osteoporosis risk in the spine and hip, a balanced diet with appropriate vitamin D and calcium, and regular exercise should be maintained (Mishra et al., 2011). However, during menopause, increased intake of calcium does not completely offset bone loss (National Institutes of Supplements, 2019). Postmenopausal women can improve bone mineral density with appropriate vitamin D and calcium intake (Roush, 2011). Calcium with or without the combination of vitamin D supplementation can be used as preventative treatment of osteoporosis (Tang et al., 2007). However, Aggarwal, (2013) found that using only vitamin D without calcium had no effect on reducing the risk of fractures. Tang et al. (2007) state that with an average treatment time of 3-5 years, fracture risk of bone loss at the hip and spine were both reduced with calcium and/or vitamin D supplementation. Contradictory to this, Jackson et al., (2006) found that for postmenopausal women, taking vitamin D and calcium supplements did diminish bone loss but at no statistical significance. These changes could be attributable to the doses used. Among elderly women, those consuming 1.2g of elemental calcium and 800IU of vitamin D3 had a reduced risk of hip fractures (Chapuy et al., 1992). The wide variety in results from using vitamin D and calcium supplementation following menopause suggests that there are small and varied benefits on bone mineral density.

Bischoff-Ferrari et al. (2012) found that daily vitamin D doses of 800IU or higher may reduce fracture risk in those aged 65 and over. Other research found no benefit from doses lower than 400IU per day when consuming 1000mg or less of calcium (Sameer Aggarwal, 2013). There is also evidence of increased kidney stone risk with taking these supplements (Sameer Aggarwal, 2013). There is little information on the baseline level of vitamin D prior to taking supplements. However, desired levels of vitamin D are possible from a healthy lifestyle and supplements could lead to overdoses of vitamin D potentially leading to health concerns (Sameer Aggarwal, 2013).

There is some evidence that having a higher cardiorespiratory fitness level will reduce the risk of bone loss, thereby reducing the risk of osteopenia and osteoporosis in healthy women post-menopause (DeFina et al., 2016). Through regular resistance based exercise it is possible to reduce the risk of osteopenia, osteoporosis and fractures through reducing the rate of bone loss in middle aged adults and stimulating bone formation in younger adults (Haskell et al., 2007). Due to the physical activity firefighters complete, firefighters should achieve and maintain sufficient cardiorespiratory fitness levels (Durand et al., 2011).

The accelerated decline in bone mineral density following menopause is possibly combatted by increasing aerobic weight bearing exercises (Fleg, 2012). This exercise increases bone density. Proctor and Joyner (1997) found that per kilogram of appendicular muscle there are reductions in aerobic capacity contributing to the lowered VO_2max with age. Tanaka et al., (1997) found that endurance-trained compared to sedentary women, have higher rates of decline in VO_2max with age. In contradiction, Atomi and Miyashita, (1974) found active females of any age had higher VO_2max values than sedentary individuals however, there was still a decline with age.

VO2max is a measurement of maximal oxygen uptake and can be used to indicate cardiorespiratory fitness (da Rocha Aragão, 2015). With increasing age, there is a decline in VO2max (Betik and Hepple, 2008) of approximately 10% for each decade after the age of 30 (da Rocha Aragão, 2015). Declining VO_2max is related to reductions in maximal cardiac output and reduced skeletal muscle mass (Proctor and Joyner, 1997). Menopause associated cardiopulmonary changes are caused by lower oestrogen, and result in prevention of adequate oxygen flow required for the rising oxygen demand in exercise through the vascular structure (Abdulnour, 2016).
For postmenopausal women, there is a worsened arterial vasodilation response, and body composition changes resulting from age and menopause lead to lowered cardiorespiratory fitness (da Rocha Aragão, 2015). These changes include reduced skeletal mass and strength.

Effects of ageing on muscle

Menopause can also contribute to the decline in muscle mass over time (Figure 10). This decline in muscle mass is known as sarcopenia. Rolland et al (2007) demonstrated a 0.6% loss in muscle mass per year after menopause.

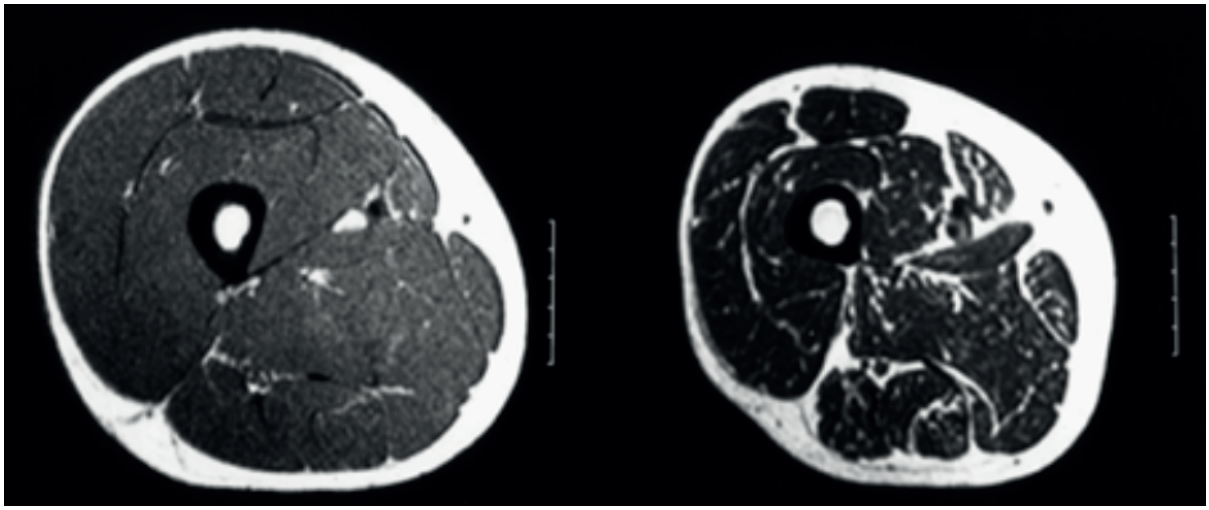

Figure 10: *Cross-sectional magnetic resonance images of the mid thighs from a 24 year old and 65 year old (right), subjects with the same body mass. Scale increments are 1cm.*

Over working age, women have been shown to lose 21% of muscle strength between the age of 25 and 55 (Asmussen et al, 1962). However, research demonstrates a pronounced reduction in strength after the 50th year of life (Doherty, 2001). Post menopause, Jubrias et al (1997) suggest isokinetic strength may reduce by approximately 10N per year (Figure 11). This drop in muscle mass and strength has been correlated with declining oestrogen levels (Philips et al. 1993).

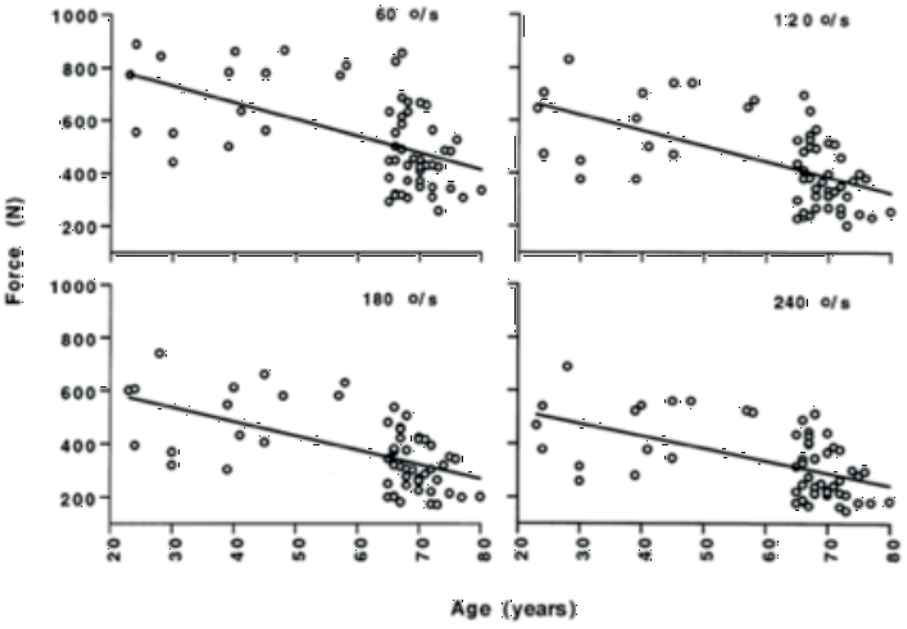

Figure 11: *Relationship between force and age. Data is shown at four angular velocities of isokinetic knee extension. The solid lines are linear regressions. All relationships are significant (from Jubrais et al, 1997).*

While some of this muscle and strength loss can be attributed to hormonal changes and inflammation, much of the decline is also a result of reduced physical activity and lower protein intake. Female firefighters should be strongly encouraged to maintain fitness and particularly strength-based activity to maintain muscle mass. Alongside activity there is a need for additional protein intake and potentially vitamin D.

Fertility and maternal health

Research from the United States suggests that the rate of miscarriages in female firefighters is twice as high as that recorded amongst the general population (Jahnke, 2018). Moreover, female firefighters experienced more preterm (<37 weeks) births (11.6 - 16.7%) than the general population (9.6%) (March of Dimes, 2015; Jahnke 2018). However, 23.9% of Fire departments in the United States did not have a policy regarding pregnancy, which is in contrast to the UK where all Fire and Rescue Services have their own individual policies. Furthermore, only one third of women restricted their duties in the first trimester (weeks 1 – 12 of pregnancy) and some women (10-16%) did not restrict their duties at all throughout pregnancy. It is currently unknown if the level of miscarriage and preterm births amongst UK female firefighters is similar to or greater than the UK general population. However, it is important to consider the possible consequences that Jahnke et al (2018) indicate may occur due to continued completion of firefighting tasks during pregnancy.

Numerous aspects of firefighting tasks have been associated with maternal and child health. Both working night shifts and daily heavy lifting (>20kg) have been reported to increase the risk of miscarriage by 1.21 and 1.31 times, respectively (Nilsson et al, 2014). The effect of night shifts may be due to changes in hormone fluctuations across the day that therefore cause menstrual cycle disturbances, but is likely also linked to associated reductions in physical activity and poor diet (Fernandez et al, 2016). There are also a wide range of toxins and chemicals, such as polybrominated diphenyl ether flame retardants, combustion related air pollutants, chemical solvents and phthalates, that have been linked to an increased risk of infertility, miscarriage and neurodevelopment disorders (Wang et al., 2016; Denicola, 2018).

In addition, many animal studies have indicated that heat stress and hyperthermia can have negative consequences on female fertility by altering hormone levels, reduced growth of female reproductive cells, decreased chance of fertilisation and higher levels of miscarriage (Boni, 2019). There is scant research on humans in this area, mainly due to the ethical issues surrounding the topic and difficulties in controlling confoundable factors. A study of ladies undergoing in vitro fertilisation cycles, identified that experiencing a fever (>38.5°C) resulted in a poor ovarian response, meaning reproductive cell growth was impaired. However, this study was observational in nature and only included 6 women, therefore clear generalizable conclusions cannot be drawn from the research (Awwad et al, 2012). Retrospective reviews of women who experienced a fever in the first trimester of pregnancy revealed no association between fever occurrence, intensity or duration and any congenital malformations diagnosed by three and a half years of age (Sass et al, 2017) or on miscarriage rates (Andersen et al, 2002). Alternatively, a review of 46 studies associated with fever and pregnancy reported that risk of neural tube defects, congenital heart defects, and oral clefts were increased by 1.5 to nearly 3-fold through exposure to maternal fever during the first trimester (Drier et al, 2014). The inconsistency in findings is likely a consequence of variation in how data is collected, the time point at which pregnant ladies are recruited, and the reliance on self-reported fevers and temperatures in some studies. It is generally accepted that a rise of ≥ 2°C is required before foetal health could be impacted, which is typically equivalent to a core body temperature of ≥39°C (Drier et al, 2014, Graham et al, 1998). Overall, it is possible that being a firefighter may impact fertility and maternal/child health, however it is unclear if this is related to heat exposure per se, or other aspects of the role.

Key considerations

The literature suggests that the response of women to exercise in hot environments varies with menstrual cycle phases and can be altered by contraceptive use, although the nature of these differences is inconclusive. There is little data on how women react to severe heat stress and only one study has been conducted on women while wearing PPE in a hot environment with exercise. There is work being undertaken at present but the current research identifies the following key points:

General

- Women may have a reduced heat tolerance to men, due to an altered later sweat response and slightly higher (in general) body fat percentage.
- Maintaining a high level of fitness is likely to help tolerate heat and hot activity irrespective of menstrual or menopause stage.

Menstrual Cycle

- There is a higher sweat rate in the early-follicular stage of the menstrual cycle, aiding heat loss.
- Core temperature is approximately 0.5ºC higher in the mid-luteal phase of the cycle, due to reduced sweat response. Subsequently, women during the mid-luteal phase may be a greater risk of heat illness.
- Those in their mid-luteal phase should be considerate of their possible increased risk of heat illness. Make sure methods to improve heat tolerance such as hydration and diet are well managed. If in a training period, consider pre-cooling methods as described in HIPA.

Menopause and Ageing

- Hot flushes during peri-menopause are caused by a small but sudden increase in core temperature.
- Peri-menopausal women may wish to have readily available methods for immediate cooling post exposure or during recovery phases.
- Peri-menopausal women should be able to remove outer tunics/ PPE when not required for protective purposes to aid heat dissipation.
- Hot flushes tend to be perceptual in nature and do not necessarily increase risk of heat illness.
- There is a lower resting core temperature post menopause, suggesting a similar or improved heat tolerance than pre-menopause.
- Bone mineral density and muscular strength may decline more quickly as a result of menopause. Lifestyle, diet and training should be encouraged to reduce the consequence of these aging processes.
- Staff should be supported with information on how to maintain fitness and strength with age.

Pregnancy

- Pregnant staff should consider shift working patterns to reduce night shifts.
- Heavy lifting should be avoided where possible.
- While the risk of contaminants is for everyone, these pose potentially greatest danger to the child.

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Protecting firefighters from heat related illness

Female firefighters
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