Heat stress, thermoregulation, and fluid balance in women

As with so many aspects of exercise related research, most investigations have focused on the male of the species. In 1940, however, Hardy and Du Bois1 undertook an investigation into gender related differences in thermoregulation, and indeed the focus on comparing the responses of men and women has dominated this area of research since. Menstrual cycle effects on thermoregulation have probably formed the next major component of work involving women and thermoregulation.

Women generally have a large surface area to mass ratio, relatively great adiposity, and a menstrual cycle. All these properties may influence thermoregulation with regard to its effectiveness and therefore may affect heat tolerance. Core temperature in the luteal phase of the menstrual cycle is higher than in the follicular phase, at least in non-heat acclimated women.2 With this, the threshold for sweating onset is elevated and there is a greater skin blood flow. Despite this knowledge, the mechanism for these changes has not been elucidated with any certainty, but it seems that the practical exercise thermoregulatory effects due to the menstrual cycle are minimal in heat stress.3

The large surface area to mass ratio of women gives faster heat exchange with the environment while the adiposity alters thermoregulatory effectiveness. The ability to prevent major shifts in core temperature is reduced with heat stress but thermoregulatory effectiveness is increased in the cold. The core temperature of a woman increases more on average than a man’s when exposed to equal climatic heat conditions at rest4 and during exercise.5 The major reasons for this reduced thermoregulatory effectiveness are that in women the onset of sweating occurs at a higher core temperature6 and the rate of sweat production is lower than in men,5 despite having a greater number of heat activated sweat glands per unit surface area as well as an absolute greater number than men.7 It is unclear, however, whether this effect is caused by sex hormones, differences in metabolism of sweat glands, differences in the anatomy of sweat glands, or some other factor(s). The limited information on sweat composition suggests that there seems to be no major gender differences in this area. An elevated iron concentration in female sweat has been reported,8 but absolute losses of iron are not different between the genders because of the smaller volume losses in women.

Exercise tolerance time in the heat is less in women than men,9 certainly for unacclimatised individuals, possibly because of either excessive heat storage or the relatively low cardiac output. With acclimatisation, however, women’s exercise heat tolerance time increases to a greater extent than men’s with heat acclimation,10 removing a significant portion of the gender difference. This probably occurs because of greater circulatory changes rather than greater evaporative heat loss.

The inter-relation between thermoregulation and body fluid balance stems from the use of evaporative cooling, particularly in response to environmental or exercise induced heat stress. Because of a woman’s lower body water content relative to her body mass, any given body water loss expressed as a percentage of body mass is greater than that of a man of the same mass with a lower body fat content. Little work on the fluid balance implications of women exercising in the heat has been undertaken. However, in terms of recovery of whole fluid balance status after a bout of exercise in the heat, the available evidence suggests that, for women who do not retain fluid over the course of their menstrual cycle, there is no difference in the recovery of fluid balance status across the cycle.11 There is also evidence to indicate that the rate and extent of dehydration is unaffected by the stage of the menstrual cycle.

In conclusion therefore, much of the literature on female thermoregulation to date is confounded by variables such as body mass and composition, aerobic power, training, and menstrual status. The effect of gender on temperature regulation during exercise must be evaluated when the metabolic heat stress for the two genders is equated. This, however, brings with it a new set of problems of investigating an unrepresentative female population.

For future research, the influence of an altering hormonal status and greater body fat content should provide sufficient stimulus to study the exercising woman in her own right. Despite this enticement, it is probably inevitable that, for most issues in the field of exercise science, the basic physiological understanding will be sought by investigations on men without their complicating factors, in a physiological sense at least!

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Food for thought

An anonymous referee once advised me to be less hesitant with my conclusions when, with cautious optimism, I wrote, “It is suggested that there is a possibility that glutamine might have a beneficial effect in certain circumstances.”

Many manufacturers of supplements have no such caution. “Constituent X will restore your vitality after exercise!, will stop your muscles aching!, will avoid that heavy legs syndrome!, will improve your immune system etc.” One memorable plug included the statement that “Glutamine will cure glandular fever”. A letter inquiring about the scientific basis for this elicited no response. It appears easier, in principle, for a company to make money from unsubstantiated claims such as these than it is to obtain ethical approval for experimental research on athletes.

Athletes are regularly advised about good nutrition and a sensible, well balanced diet. The reality is different. They are more likely to have a high protein and high fat but low carbohydrate diet and to take high supplementary doses of more than one vitamin and/or mineral product.

The problem, however, is that the content of many “different” supplements overlaps, and thus the athlete may overdose on some constituents. Responsible manufacturers advise clearly not to take more than the recommended dose—nevertheless, combinations of supplements can lead to overdosing. Tea and coffee can inhibit absorption of iron. Taking a combination of supplements, one of which is iron, with others containing caffeine, may have the opposite effect to that intended owing to the accumulated caffeine content.

Taking 60 mg vitamin C a day will maintain a body pool of 1500 mg and any excess is likely to be excreted. The ingestion of much higher amounts, perhaps double the recommended daily allowance (RDA) commonly consumed by athletes, may create problems with iron loading, copper absorption, or kidney oxalate stone formation in susceptible people.

For vitamin A, not only is there no advantage in ingest ing more than the RDA but also vitamin A is toxic in very high doses. There are anecdotal reports of supplements that contain a dosage substantially in excess of the RDA of some vitamins. It is easy to say that athletes should not be taken in by “charlatans”, but it is difficult for athletes to discern which companies are giving sensible advice in the midst of sophisticated marketing techniques.

A brief survey of scientific publications since 1966 shows that eight supplementation research studies on vitamins and minerals were carried out, including three on iron, and two or three on protein or carbohydrate ingestion between 1966 and 1986. In the succeeding four years interest in vitamins and minerals expanded, as reflected by 16 studies, and reports on carnitine, dihydroxyacetone pyruvate, and inosine also appeared. Between 1991 and 1996 interest in iron increased, and studies on carnitine expanded; vitamins and minerals were still of interest with more than 20 studies reported; carbohydrate was back in fashion (10 studies); and a newcomer, creatine, became very popular, with seven studies carried out. In the past two years, another 11 creatine studies have been published, with carbohydrate (six) and iron still of interest. These figures are approximate, and do not include glutamine and branched chain amino acid supplementation, which will be dealt with separately.

The main aim of creatine supplementation is to increase the levels of phosphocreatine (a major fuel to be used in explosive sports); it is also used to increase muscle mass. Many athletes have reported problems associated with its use, including muscle cramps and gastrointestinal disorders. However, as yet, there is no scientific evidence to substantiate these claims; equally, there is no research into the long term effects of high doses of creatine.

Carbohydrate boosting—“carbo-loading”—is a well known regimen followed by many marathon runners since it was first mooted many years ago, and the major pre-marathon pasta parties attract a huge number of runners. That carbohydrate should provide 60% of energy is well known, but many athletes still do not take sufficient during either training or competition.

It has been claimed by some research workers that provision of glutamine or a precursor reduces the incidence of infections after prolonged, exhaustive exercise. However, the precise effect on immunodepression has yet to be established.

Supplementation of the branched chain amino acids, such as leucine, isoleucine, and valine (BCAA), has been widely studied for its effect on central fatigue, which emanates from the brain, rather than peripheral fatigue, which emanates from muscle. The mental exertion necessary to maintain a given power output is extremely important in central fatigue, and BCAA supplementation has been found to improve mental performance after prolonged, exhaustive exercise.

For the most part, there seems to be no substitute for a healthy, balanced diet, and a “quick fix” is seldom appropriate. However, an athlete’s interpretation of what constitutes a balanced diet may be uncertain. We must continue to emphasise the importance of a high carbohydrate intake for both training and competition, but it is difficult to wean some athletes away from the notion that supplementation is the answer to all their problems. Nevertheless, supplementation may be advantageous in some situations. Ideally, athletes should have easy access to impartial advice on the value of supplementation in their own individual circumstances.

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Exercise and primary dysmenorrhoea

In the last 15 to 20 years, research into the link between physical activity and menstrual disorders has increased significantly. Exercise has been found to affect menstruation in a variety of ways, including inducing amenorrhoea in athletes and perhaps alleviating symptoms involved in premenstrual syndrome and dysmenorrhoea. Primary dysmenorrhoea is characterized by a range of symptoms, foremost of which are lower abdominal pain that may radiate to the lower back or legs, headache, nausea, and vomiting. Some 75–85% of women experiencing dysmenorrhoea report symptoms as mild, although dysmenorrhoea may cause significant absence from school and work. The symptoms are thought to stem from elevated levels of prostaglandins resulting in uterine contractions and ischaemia. The origin of the increase in prostaglandins has not been decisively identified; one likely mechanism involves a decline in progesterone in the premenstrual phase, which results in the synthesis of prostaglandins in endometrial cells by membrane phospholipids. This theory is supported by the success of prostaglandin synthesis inhibitors in pain relief. However, as these inhibitors only provide pain relief in 70–75% of women, other factors are probably also involved. Possible contributors include deficient degradation of prostaglandins as the result of a defect in prostaglandin dehydrogenase, the vasoconstrictive action of antidiuretic hormone, and variations in pelvic blood flow, which may influence synthesis or breakdown of prostaglandins. Primary dysmenorrhoea affects between 47 and 80% of the general population depending on the age group studied, as it tends to decrease with age and after pregnancy.1

Although exercise is generally thought to alleviate the discomfort associated with dysmenorrhoea, the scientific literature on this phenomenon displays mixed evidence. One study shows a decline in the severity of symptoms after a 12 week aerobic training programme,2 and another reports diminished dysmenorrhoea in exercising junior high school girls.3 Similarly, women who train intensively have been found to report fewer symptoms than women who exercise occasionally.4 However, a number of other studies have failed to find any relation between dysmenorrhoea and levels of physical activity.5–7 Furthermore, Metheny and Smith8 report that, after controlling for disposition and mood, exercise is actually associated with higher levels of menstrual discomfort.

An interesting element of the relation between exercise and dysmenorrhoea is the involvement of stress. A number of studies have shown a correlation between life stress and dysmenorrhoea; one likely mechanism involves a stress induced dysmenorrhoea, it may simultaneously aggravate symptoms. One explanation of this aggravation may be that exercise raises somatic awareness, as regular exercise has been associated with increased sensitivity to bodily states. Alternatively, if exercising women reduce their activity levels in the premenstrual phase, the resulting fall in endorphin levels could intensify dysmenorrhoea.4

Overall, research into the relation between dysmenorrhoea and exercise has been hampered by methodological flaws such as varying definitions of dysmenorrhoea and activity, different modes of data collection, disparate study design, failure to perform blind studies, and retrospective reporting of symptoms. Blind studies are particularly important in this area of research, as it has been shown that women may be influenced by previously held beliefs, taboos, and social expectations involving menstruation when answering menstrual distress questionnaires.14 This complication, along with the confounding effects of stress, points to the variety of factors influencing dysmenorrhoea and its relation to exercise. More carefully controlled longitudinal studies should help elucidate this complex relationship. Research in this area is important given the high prevalence of dysmenorrhoea and the potential benefits of exercise.

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Bone metabolism in exercise associated amenorrhoea: the importance of nutrition

Osteoporosis was shown to be a health risk for young women with exercise associated amenorrhoea in the early 1980s when a series of studies showed that such women had a lower bone mineral density (BMD) than age matched, active, or sedentary eumenorrhoeic women.¹—³ Subsequent research showed that active amenorrhoeic women continued to lose bone while their menstrual disturbance persisted, but that their BMD stabilised or increased if their menses resumed.⁴ Furthermore, retrospective analyses of the menstrual histories of these women showed that their BMD correlated positively with the duration for which they had experienced regular menses.⁵

The “traditional” explanation for premature bone loss in young women with exercise associated amenorrhoea has been an oestrogen deficiency.⁶—⁸ Indeed, the rationale for conducting the aforementioned studies was that it was known that the amenorrhoea of these women coincided with an oestrogen deficiency and that an oestrogen deficiency is the principal cause of bone loss in women with ovarian failure.⁹—¹⁰ Thus it was hypothesised that active amenorrhoeic women may also be at risk of osteoporosis.¹¹—¹³ The results of these early studies not only supported a link between exercise associated amenorrhoea, oestrogen deficiency, and bone loss but, perhaps more importantly, they showed that the exercise undertaken by these active amenorrhoeic women was unable to protect their bone mass.

However, more recent research has challenged the notion that an oestrogen deficiency is the primary cause of bone loss in active amenorrhoeic women. Firstly, these women appear to be less responsive to exogenous oestrogen treatment than women with ovarian failure, for whom oestrogen replacement can offer complete protection against bone loss.¹⁴—¹⁶ Secondly, studies of bone turnover in women with exercise associated amenorrhoea using biochemical markers of bone cell activities and collagen turnover have suggested that their pattern of bone remodelling is atypical of an oestrogen deficient state.¹⁷—¹⁹ This state is usually characterised by increased bone turnover with “excessive” bone resorption, which is normalised by oestrogen replacement.²⁰—²¹ However, studies in our laboratories have shown an apparent reduction in bone turnover and, more especially, reduced bone formation in women distance runners with chronic amenorrhoea (longer than four years) when compared with eumenorrhoeic runners or age matched sedentary eumenorrhoeic women.²² It was particularly noteworthy that none of these amenorrhoeic runners exhibited signs of “excessive” bone resorption.

As these amenorrhoeic runners were significantly lighter and leaner than the eumenorrhoeic runners or sedentary women, we explored relations between serum or urine levels of bone turnover markers and indices of nutritional status in these women. We found that serum levels of bone formation markers correlated positively with estimated energy balance, body mass index (BMI), and insulin-like growth factor I (IGF-I) in the amenorrhoeic group of runners, but not in the eumenorrhoeic groups. The lowest levels of bone formation markers were measured in those amenorrhoeic runners who exhibited characteristic signs of undernutrition, such as a BMI < 17.5 kg/m² and a subnormal serum level of T₃ and IGF-I. There was no correlation between urine levels of bone resorption markers and any of these nutritional indices.

These findings suggest that undernutrition may underly the bone remodelling imbalance and bone loss in active amenorrhoeic women, and, furthermore, that nutritional factors may counteract or override the stimulatory effects of an oestrogen deficiency on bone turnover.

Previous research has also supported a link between undernutrition, reduced bone formation, and bone loss in young women. For example, women with anorexia nervosa, who are typically undernourished and amenorrhoeic, have reduced bone formation and experience rapid bone loss.²³—²⁵ However, with refeeding, they exhibit an increase in the serum level of bone formation markers²⁶ and often a gradual increase in BMD, even without a resumption of menses.²⁷ Also, protein-energy malnutrition appears to play a pertinent role in the development of osteoporosis in the elderly and in individuals with malabsorption disorders.²⁸ Furthermore, it has been hypothesised that in active women amenorrhoea itself is induced by a chronic negative energy balance, or its effects on body mass, body composition, and metabolism.²⁹

Although the metabolic interaction between undernutrition and imbalanced bone remodelling requires further exploration, it is nevertheless known that an acute or chronic energy deficit elicits metabolic aberrations which can lead to inadequate bone formation or excessive bone resorption.³⁰—³² These metabolic aberrations include hypercortisolaemia, low T₃ syndrome, and IGF-I deficiency,³³ all of which have been documented in active amenorrhoeic women.³⁴—³⁶ Of interest, in a recent study, short term fasting (four days) in healthy eumenorrhoeic women was shown to elicit a reduction in the serum level of bone formation markers (as well as IGF-I), which was rectified by raising the serum level of IGF-I in spite of continued fasting.³⁷ Also, administration of recombinant human IGF-I to anorexic women has been shown to effect a dose related increase in the serum level of bone formation markers.³⁸ Thus the correction of metabolic abnormalities that impact on bone formation, such as IGF-I deficiency, is a promising therapy for a nutritionally linked bone remodelling imbalance.

In summary, there is growing evidence to suggest that undernutrition and its metabolic consequences are responsible for precipitating a bone remodelling imbalance which may lead to bone loss in young women with exercise associated amenorrhoea. This evidence does not preclude a role for oestrogen deficiency in the aetiology of this bone remodelling imbalance, as undernutrition is also a suggested cause of ovarian suppression in these women.³⁹ The potential to reverse osteopenia in women with exercise associated amenorrhoea is currently debatable, as a normalisation of BMD has yet to be demonstrated in prospective studies. As reduced bone formation (rather than increased resorption) has been shown in these women,⁴⁰ therapeutic strategies aimed at increasing osteoblast activity and collagen formation are likely to be of greater benefit than agents that retard osteoclast activity. However, as with other forms of osteoporosis, prophylaxis is likely to outweigh the benefits of treatment for these women.⁴¹—⁴³ This in turn depends on the treatment of exercise or dietary behaviour which may precipitate a bone remodelling balance.

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Child abuse and the sports medicine consultation

Are doctors who are involved in sports medicine really aware that child abuse in sport may present to them indirectly in the sports injury clinic? How many of the “injuries” or “fatigue producing poor performances” in children are cries for help that are missed in the consultation room? When should we think harder about possible child abuse? Might signs of physical, emotional, or sexual abuse be the effects of abusive relations at home or in sport rather than just the causes of disappointing sports performance?

Sport at the highest level is demanding more time from its participants, many of whom have been involved in training and competition from a very young age. The closeness of the abuser/athlete relationship could lead to opportunities for either the paedophile or the sexual predator to indulge in unacceptable physical contact or emotional abuse of the athlete. Although all gender permutations have been reported in accounts of sexual abuse in sport, most known cases have occurred between an older male authority figure and a younger female athlete (hence the terminology adopted in this article). It is also important to stress that the term “abuse” refers not only to sexual exploitation but may also apply to physical or emotional exploitation or to neglect.

Abusers are diverse in terms of their sociodemographic characteristics and motives. Most reported abusers in sport have been coaches, but research is still at an early stage and administrators, parents, senior athletes, and medical and paramedical staff, who also have personal contact with athletes, present possible additional risks. These sports for which cases of abuse are already in the public domain are characterised by a high level of social and personal contact between the coach and athlete. This, often intense, relationship requires hours of training time, together, travel to national and international competitions, close proximity at meals, visits to the coach’s home, and often, importantly, shared accommodation. The athlete, over months or years, learns to respect and obey this mentor. She sees him in loco parentis, a confidante, and a shoulder to cry on. The abusing coach uses this dependence, creating a climate of favouritism, secrecy, and closeness with malice aforethought. This process is called “grooming” and is planned to secure complete secrecy, co-operation, and compliance from the targeted athlete.

Although folklore suggests that certain sports are more prone to problems of sexual exploitation than others, it is highly dangerous to focus on just a few. All sports are risk settings; certainly the degree of risk varies from one setting to another but many risks are also shared across sports. On the basis of current research data, it is not possible to say with certainty exactly which sports pose the greatest risks of sexual exploitation to the athlete. However, preliminary analysis suggests that the stage just before peak performance, what Brackenridge and Kirby call the “stage of imminent achievement”, is the time when the athlete has both the most to lose by leaving the sport and everything to gain by complying with the demands of authority figures around her. If the stage of imminent achievement coincides with puberty, then the risks are thought to be greater: in other words, early peaking sports are thought to present the greatest risks of exploitation by sexual predators in authority positions.

It is not uncommon for the athlete to wait several years before disclosing experiences of abuse. The reasons behind this are complex. The athlete usually fears that the coach may victimise or harass her if she withdraws her co-operation or, indeed, that her own athlete peers may resent her, either for receiving favouritism during the abuse or for upsetting the dynamics in the club, team, or squad. It is not unknown for a coach to sexually abuse several athletes at the same time. In one case, a paedophile coach regularly abused more than 30 boys in a community club, and in another an elite coach pursued predatory sex with two separate athletes in his squad of only four young girls. These two athletes did not discover this until several years afterwards. Anyone disclosing abuse risks personal stigma and other severe consequences by putting this knowledge into the public domain. Most frightening of all to the young aspiring medal winner is the prospect of losing favour from the coach and being dropped by him from the squad.

Although many sports have introduced vetting and monitoring procedures for their coaches, at all levels, by no means all have done so. The sports medicine specialist, among others, has the opportunity to identify early signs of inappropriate distress in athletes that may indicate abuse related problems. Doctors and physiotherapists should check the child protection and reporting procedures for their own sports teams or governing bodies to ensure that the athlete and themselves are protected. Unfortunately police data on potential child abusers in sport are flawed. Official registers record only those people with convictions and some for whom there is significant evidence but no conviction. However, until the proposed new Criminal Records Bureau becomes operational, access to these data continues to be strictly controlled and to exclude most voluntary sector groups.
The vast majority of abusers in sport evade detection or continue to operate behind a wall of silence and approval because they are good coaches and/or appear to be "nice men". Sexual abuse is characteristically covert and secretive. Perpetrators prey on the vulnerable in their care, usually targeting athletes who are vulnerable and suffering low self esteem. If suspicion is aroused, the abuser may move on to another sport or to another part of the country. The best treatment of child abuse is, of course, prevention. For the abuser to feel threatened he must know there is an environment within his sport where child abuse is talked about openly. There should be meetings with athletes, officials, and medical staff to establish good practice, and both athletes and their parents must know that there are channels within the sport for reporting harassment.

The next time the symptoms and signs don’t really add up in the consultation, or the young athlete’s demeanor is subdued and inhibited, or she shows signs of disordered eating, or the adult in charge is a little too domineering—think twice.

Practical guidance on child protection and anti-harassment procedures for sport organisations is available from Professor Celia Brackenridge. Email: brackenridge@chelt.ac.uk.

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