

Sport and exercise medicine in the United Kingdom comes of age

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Sport and exercise medicine in the United Kingdom is awarded specialty status, but now the real work begins, to deliver on promises made

The 21 February 2005 proved to be a red letter day for sport and exercise (SEM) medicine in the United Kingdom, as the Department of Health announced that it was approving the application for specialty status submitted in early 2004. This ended a process that began in 1998 with the formation of the Intercollegiate Academic Board of Sport and Exercise Medicine (IABSEM), under the auspices of the Academy of Medical Royal Colleges. Progress was slow until early 2003, when the intervention of the Minister of Sport led to an educational forum and the subsequent formation of a working party tasked with developing the application. The working party consisted of medical professionals, representatives of UK Sport, the Department of Culture, Media and Sport, and the Department of Health. The application had to clearly establish that the creation of a new medical specialty was the best and most effective way of answering a service need, or exceptionally a national need. The working party were able to argue for the recognition of SEM on both counts, and furthermore make a case based on the other 11 principles for new specialties as set out in the 2001 Department of Health document, "Developing specialties in medicine".

The timing was favourable, as the government was increasingly turning its attention to strategies to defuse the public health time bomb posed by spiralling national levels of physical inactivity and obesity.¹⁻⁴ This, coupled with London's bid to host the 2012 Olympic Games, of which the NHS is a key supporter, provided the perfect backdrop highlighting the relevance of SEM to all levels of society. The new specialty was thus founded on a holistic approach to addressing illness and injury in those who exercise, injury prevention, and the safe use of physical activity in the treatment and prevention of illness with encouragement of wellbeing through exercise and physical activity.

The Department of Health accepted the reasoning that SEM practitioners

would be ideally placed not only to provide timely and expert treatment of musculoskeletal injuries which were estimated to cost the NHS some £590 million per annum, but also to coordinate a range of initiatives that would promote physical activity as an effective intervention and prevention tool for a wide spectrum of health problems.

This announcement is undoubtedly a huge breakthrough for SEM, making the UK one of several EU countries to accept SEM as a stand alone medical specialty, some with four year programmes of higher specialty training (HST). However, as the dust settles and we get over the initial euphoria, we must now turn our attention to ensuring that SEM effectively delivers better health for our patients and behaves in a manner consistent with other medical specialties.

WHERE NEXT?

The immediate need is to refine our detailed training curriculum, approve training regions, and identify appropriately qualified trainers. The Specialist Training Authority (STA) or Postgraduate Medical Education and Training Board (PMETB), who will assume the role of the STA later this year, is likely to devolve this task to the IABSEM, who may assume the role of a faculty. A number of committees with responsibility for training (Specialty Advisory Committee), examination, appraisal, and revalidation of SEM doctors will be set up, drawing on the knowledge and experience of doctors from both SEM and other disciplines. The tasks undertaken will include defining the criteria for the retrospective award of a Certificate of Specialist Training (CST) and assessing those doctors currently working in the area to determine if they fulfil the criteria for inclusion on the specialist register.

With an agreed national curriculum, trainers, and training regions (postgraduate deaneries and budgets) identified, the process of enrolling trainees into HST can begin. In the first instance,

there are likely to be a number of doctors who have a considerable amount of relevant experience without fully meeting the criteria for the award of a CST; there will be a mechanism to have this recognised and these doctors will be able to enter HST at year two or three as appropriate. There will continue to be opportunities for doctors in other medical specialties to develop SEM as a subspecialty interest by undertaking a one year subspecialty training programme.

It is envisaged that a relatively small number of trainees will gain entry to the regionalised HST programmes, and that competition for places will be intense. Trainees will become eligible to apply for HST after foundation training, and at present it is likely that MRCP, MRCPCH or MRCS and a Diploma, or MSc in SEM will be essential entry criteria. The training programme will be of four years duration, flexible, and consistent with the aims of existing training and "modernising medical careers". At the end of this programme, the future SEM specialist should have the knowledge and competencies to manage a wide range of exercise related conditions, advise on the therapeutic use of exercise, and provide medical support to athletes at all levels of participation. The future SEM specialist will be expected to integrate with community and primary care services to help promote physical activity strategies that will target those most in need and develop seamless care pathways for those with exercise related injury.

THE CHALLENGES

Our strengths lie in our multidisciplinary approach and the "broad church" of our specialty. To date SEM doctors in the UK have largely worked in isolation, and much of our accepted practice has been based on expert opinion rather than rigorous science. We are now faced with the challenge of demonstrating that our specialty can stand comparison with other conventional specialties by clearly showing that we are prepared to methodically examine what we do, and reject what does not stand up to the closest of scrutiny. Accordingly we must embrace the need for properly conducted research and develop an evidence based practice through the conscientious, explicit, and judicious use of current best knowledge. To this end, research will be integral to HST programmes, and trainees will be encouraged to submit their work for peer review and publication. All trainees should aspire to undertake a higher degree, as is the accepted norm in other specialties.

Ultimately we will be judged on our results: has the introduction of SEM led to a healthier population as seen in lower cardiovascular mortality and morbidity, lower rates of obesity and diabetes, less work absenteeism, etc? We wish to see and encourage a Department of *Health* (not unhealth) and a service of truly National *Health*. We will need to be given time to demonstrate our worth—Rome wasn't built in a day and we cannot be expected to transform a nation of couch potatoes into a nation of athletes overnight. Furthermore, it should be appreciated that the problems of unhealthy living, obesity, and lack of physical activity are not solely the problems of SEM but societal issues. It

has taken 25 years to produce a significant change in our smoking habits (and there were considerably fewer smokers than inactive people). It will take at least this period of time and appropriate funding if we are to see similar results with respect to activity levels and improved health.

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REFERENCES

- 1 **Conway M**. Game plan: a strategy for delivering government's sport and physical activity objectives. Government Publications 2002. Government's Strategy Unit and Department for Culture, Media and Sport.
- 2 **Central Council for Physical Recreation**. Saving lives, saving money: physical activity the best buy for public health. Central Council for Physical Recreation, 2002.
- 3 **Department of Health**. Choosing health, choosing activity: a consultation on how to increase physical activity. DoH/DCMS consultation paper, 2004.
- 4 **Chief Medical Officer**. At least five a week: evidence on the impact of physical activity and its relationship to health. Report of the Chief Medical Officer. Department of Health, 2004.

Skeletal muscle damage

Can oestrogen influence skeletal muscle damage, inflammation, and repair?

P M Tiidus

More research is needed before the use of oestrogen can be recommended for muscle damage

Oestrogen may affect muscle damage and inflammation, but the physiological significance of this, particularly potential effects on muscle repair and recovery in humans, and the mechanisms of its actions are as of yet unknown.

Over the last decade, evidence has accumulated that oestrogen may act to diminish skeletal muscle damage and inflammatory responses after unaccustomed exercise or other damaging insult. These prophylactic effects are similar to the effect of oestrogen in diminishing damage and inflammation in other tissues such as cardiac muscle, liver, and nervous tissue.^{1–3} What is not clear is the physiological significance of this effect of oestrogen and whether it is physiologically meaningful or results in significantly differential responses in skeletal muscle after over-exertion in humans.⁴ The mechanisms by which oestrogen exerts its effect on skeletal muscle responses to damaging exercise, and whether its influence extends to differential rates of muscle repair and recovery also require further investigation.

The primary practical implication of a role for oestrogen in post-exercise muscle damage, recovery, and repair

mechanisms is likely to be for postmenopausal women who have reduced oestrogen concentrations, and thus may be more susceptible to muscle damage and slower recovery than premenopausal women. Sex differences in susceptibility to muscle damage are also of interest. This overview will briefly examine these questions and summarise the current evidence for oestrogenic influence on skeletal muscle damage, inflammation, and repair. Considerable research is still needed before any conclusions can be reached about the physiological significance of these effects of oestrogen in humans.

INFLUENCE OF OESTROGEN ON SKELETAL MUSCLE DAMAGE

Oestrogen is a strong antioxidant and a membrane stabiliser.^{5–6} We have proposed that these properties, which are unique to oestrogen as a steroid hormone, may account for some of its mitigating effects on post-exercise muscle damage indices.^{7–8} Indeed, early studies by Bär and Amelink⁹ showed a significant *in vivo* and *in vitro* attenuation of muscle creatine kinase leakage and lower blood creatine kinase activity in oestrogen treated rodents after damaging contractions. Although creatine

kinase release from skeletal muscle is not a good index of muscle structural damage, it is considered a reasonable indicator of muscle membrane and sarcolemma stability and disruption. Hence, a primary effect of oestrogen may be to protect muscle membranes from exercise induced muscle damage.⁷ The reduction of muscle membrane disruption may also be important in inflammatory responses and muscle repair.

Only a few studies have addressed the possibility of direct oestrogenic influence on muscle structural damage after exercise, and, of these, none have found direct evidence of oestrogen involvement. The most comprehensive of these studies¹⁰ found significant sex differences in histochemical and biochemical indices of muscle damage after downhill running in rats. The attenuation of post-exercise muscle damage reported in the female rats was potentially attributed to differences in circulating oestrogen concentrations. The limited number of studies that have attempted to histochemically quantify differences in indices of post-exercise muscle damage in men and women from biopsy samples have had conflicting results.⁷ Human sex differences in exercise induced muscle damage may be masked by small sample sizes and variability of data obtained from small biopsy samples.¹¹ However, other indicators of muscle damage in humans after exercise have generally also not found major sex differences.⁴ Probably a more important human health issue related to oestrogen is the potential differences in susceptibility to exercise induced muscle damage between premenopausal and postmenopausal women. As of yet, no studies have directly addressed this issue. However, it has been reported that a postmenopausal loss of oestrogen may result in greater aging related

strength loss and a reduced rate of strength gain in older women.¹² Hence, although some information is available, definitive studies on the influence of oestrogen on skeletal muscle damage, particularly in humans, are lacking and further investigation is warranted.

INFLUENCE OF OESTROGEN ON MUSCLE INFLAMMATION AFTER EXERCISE

Although there are still some conflicting data, particularly in the few human studies that have addressed this issue, oestrogen has generally been shown to inhibit inflammation related leucocyte infiltration into both skeletal⁷ and cardiac² muscle after unaccustomed exercise or other damaging insult such as ischaemia-reperfusion injury. Leucocytes such as neutrophils and macrophages play important roles in muscle recovery by assisting the removal of damaged tissue during inflammation and the activation of muscle satellite cells during muscle regeneration.^{5,6} However, neutrophils in particular may also further exacerbate post-exercise damage by generating oxidising agents. Hence it has been suggested that limiting leucocyte infiltration into skeletal muscle during the inflammatory period may lessen total muscle disruption.⁶ Indeed, evidence from studies on cardiac, neural, and other tissues suggest that limiting neutrophil infiltration with oestrogen will reduce damage and speed healing in these tissues.¹⁻³

Several studies have suggested that oestrogen does indeed attenuate muscle neutrophil infiltration after exercise, particularly in animal models. Attenuation of muscle neutrophil infiltration has been repeatedly shown in oestrogen supplemented male and ovariectomised female rats after unaccustomed exercise or ischaemia-reperfusion induced injury.^{8,13,14} A further study has also reported delays in macrophage infiltration into skeletal muscle in female compared with male rats after damage.¹⁵ However, the physiological implications of these effects of oestrogen on subsequent muscle damage and repair have yet to be experimentally documented.

The mechanisms by which oestrogen attenuates muscle neutrophil infiltration after exercise are not fully understood. We have suggested that the antioxidant and/or membrane stabilising characteristics of oestrogen may play a role in decreasing neutrophil chemotaxis to disrupted muscle, possibly by decreasing calpain activation or by influencing other membrane related neutrophil capture, adhesion, or diapedesis mechanisms.^{7,8} As there is evidence

for the presence of oestrogen receptors on skeletal muscle,¹⁶ it is possible that oestrogen may also affect inflammation related responses by direct receptor mediated mechanisms.^{7,8} Further experimental evidence is needed to confirm the mechanism(s) by which oestrogen affects muscle inflammation and ultimately the physiological significance of this effect, particularly in human models.

INFLUENCE OF OESTROGEN ON SKELETAL MUSCLE REPAIR

One consequence of the attenuation of muscle leucocyte infiltration by oestrogen may be a change in the rate of skeletal muscle repair or recovery from injury. As previously noted, leucocytes may exacerbate muscle damage caused by exercise as a consequence of their role in the clearance of damaged tissue. The attenuation of leucocyte infiltration by oestrogen may therefore diminish extraneous damage, leading to faster healing. Alternatively, infiltration of muscle by leucocytes (particularly macrophages) may be important in activating muscle satellite cells and initiating their critical role in muscle regeneration. Hence any attenuation of leucocyte infiltration may delay important stages in the muscle recovery process.⁶

There is other evidence that oestrogen can influence NO and inducible nitric oxide synthase (iNOS) activity in a number of tissues and by so doing influence damage and recovery processes.^{17,18} As NO and iNOS are important regulators of skeletal muscle satellite cell activation,¹⁹ oestrogen may also affect muscle regenerative processes through iNOS mediated influence on muscle satellite cells or other recovery related parameters.

Despite these theoretical postulations, there is as yet almost no experimental evidence on the potential for oestrogen to influence muscle recovery or repair mechanisms. Very recent preliminary work from our laboratory, which compared ovariectomised, oestrogen supplemented female rats with unsupplemented rats found greater numbers of active muscle satellite cells in hind-limb muscles of rats 72 hours after an intense eccentric exercise bout.²⁰ If these preliminary findings are confirmed by more extensive experimentation and more comprehensive measures of muscle recovery, the role and importance of oestrogen in muscle repair could be further clarified. Any such information would add to the current discussions on the benefits versus drawbacks of postmenopausal hormone replacement therapy.

A ROLE FOR PROGESTERONE?

As much of the animal based research noted above has used oestrogen supplemented male or ovariectomised animals, the physiological significance of progesterone in muscle damage and repair may have been under-reported. There is little evidence that progesterone has any direct effect on muscle damage, inflammation, or repair mechanisms. However, a number of studies have reported antagonistic effects of progesterone on oestrogen actions in other tissues. A recent study found that, with vascular injury in ovariectomised rats, oestrogen supplementation reduced leucocyte infiltration by 50% and progesterone had no independent effect on leucocyte infiltration after damage.²¹ However, when progesterone was supplemented in combination with oestrogen, it completely negated the attenuating influence of oestrogen on post-damage vascular leucocyte infiltration. Oestrogen and progesterone interactions have also recently been reported in relation to their potential to influence muscle fatigue.²²

Hence the potential interactive effects of oestrogen and progesterone need to be further investigated, particularly in relation to the *in vivo* physiological significance of oestrogenic influence on muscle recovery in women.

SUMMARY AND CONCLUSIONS

Experimental evidence, primarily from animal models, suggests a role for oestrogen in mitigating muscle damage and disruption and in inflammation related leucocyte infiltration into skeletal muscle. There are also a number of theoretical reasons to suspect that oestrogen has some effect on the rate of skeletal muscle recovery and repair. However, much more research is required before any physiologically significant role of oestrogen in muscle repair and recovery can be attributed, particularly in human females. Hence, any practical application of oestrogen supplementation, particularly in postmenopausal women, as a prophylactic to muscle damage or catalyst to muscle healing must await further experimentation.

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REFERENCES

- 1 Wise PD, Dubal D, Wilson M, *et al*. Minireview: neuroprotective effects of estrogen: new insights into mechanisms of action. *Endocrinology* 2001;**142**:969-73.
- 2 Squadrito F, Altavilla D, Squadrito D, *et al*. 17 β -Oestradiol reduces cardiac leukocyte

- accumulation in myocardial ischemia-reperfusion injury in rats. *Eur J Pharmacol* 1997;**335**:185–92.
- 3 Harada H, Pavlick KP, Hines IN, *et al.* Effects of gender on reduced-size liver ischemia and reperfusion injury. *J Appl Physiol* 2001;**91**:2816–22.
 - 4 Clarkson PM, Hubal MJ. Exercise induced muscle damage in humans. *Am J Phys Med Rehabil* 2002;**81**(suppl):S52–69.
 - 5 Kendall B, Eston R. Exercise-induced muscle damage and the potential protective role of estrogen. *Sports Med* 2002;**32**:103–23.
 - 6 Tiidus PM. Estrogen and gender effects on post-exercise muscle damage, inflammation and oxidative stress. *Can J Appl Physiol* 2000;**25**:274–87.
 - 7 Tiidus PM. Influence of estrogen on skeletal muscle damage, inflammation, and repair. *Exerc Sport Sci Rev* 2003;**31**:40–4.
 - 8 Tiidus PM, Holden D, Bombardier E, *et al.* Estrogen effect on post-exercise skeletal muscle neutrophil infiltration and calpain activity. *Can J Physiol Pharmacol* 2001;**79**:400–6.
 - 9 Amelink GJ, Bär PR. Exercise-induced muscle protein leakage in the rat: effects of hormonal manipulation. *J Neurol Sci* 1986;**76**:61–8.
 - 10 Komulainen J, Koskinen S, Kalliokoski R, *et al.* Gender differences in skeletal muscle fibre damage after eccentrically biased downhill running in rats. *Acta Physiol Scand* 1999;**165**:57–63.
 - 11 Stupka N, Lowther S, Chorneyko K, *et al.* Gender differences in muscle inflammation after eccentric exercise. *J Appl Physiol* 2000;**89**:2325–32.
 - 12 Sipilä S, Poutamo J. Muscle performance, sex hormones and training in peri-menopausal and post-menopausal women. *Scand J Med Sci Sports* 2003;**13**:19–25.
 - 13 Tiidus PM, Bombardier E. Oestrogen attenuates post-exercise myeloperoxidase activity in skeletal muscle of male rats. *Acta Physiol Scand* 1999;**166**:85–90.
 - 14 Stupka N, Tiidus PM. Effects of ovariectomy and estrogen on ischemia-reperfusion injury in hindlimbs of female rats. *J Appl Physiol* 2001;**91**:1828–35.
 - 15 St Pierre-Schneider B, Correia L, Cannon JG. Sex differences in leukocyte invasion in injured murine skeletal muscle. *Res Nurse Health* 1999;**22**:243–50.
 - 16 Wiik A, Glenmark B, Ekman M, *et al.* Oestrogen receptor β is expressed in adult human skeletal muscle both at the mRNA and protein level. *Acta Physiol Scand* 2003;**179**:381–7.
 - 17 Node K, Kitakoze M, Kkosaka H, *et al.* Amelioration of ischemia- and reperfusion-induced myocardial injury by 17 β -estradiol. *Circulation* 1997;**96**:1953–63.
 - 18 Prorock AJ, Hafez-Moghadam A, Laubach VE, *et al.* Vascular protection by estrogen in ischemia-reperfusion injury requires endothelial nitric oxide synthase. *Am J Physiol* 2003;**284**:H133–40.
 - 19 Anderson JE, Wozniak AC. Satellite cell activation on fibers: modeling events in vivo: an invited review. *Can J Physiol Pharmacol* 2004;**82**:300–10.
 - 20 Tiidus PM, Deller M, Liu XL. Oestrogen influence on myogenic satellite cells following downhill running in male rats: a preliminary study. *Acta Physiol Scand* 2005;in press.
 - 21 Xing DQ, Miller A, Novak L, *et al.* Estradiol and progestins differentially modulate leukocyte infiltration after vascular injury. *Circulation* 2004;**109**:234–41.
 - 22 St Pierre-Schneider B, Fine JP, Nadolski T *et al.* The effects of estradiol and progesterone on plantarflexor muscle fatigue in ovariectomized mice. *Biol Res Nurs* 2004;**5**:265–75.

ELECTRONIC PAGES.....

Online original articles

The following electronic only articles are published in conjunction with this issue of *BJSM* (see also pages 266 and 297)

Development and initial validation of the Brunel lifestyle physical activity questionnaire

C I Karageorghis, M M Vencato, N L D Chatzisarantis, *et al*
Objectives: To develop a valid and reliable internet based lifestyle physical activity questionnaire suitable for use among the United Kingdom population.

Methods: After a detailed content analysis and item generation using a panel of experts, an internet based measure of lifestyle physical activity behaviour was developed. Data were collected from 1369 subjects in total. Confirmatory factor analysis was used to examine the two subscales of the Brunel lifestyle physical activity questionnaire among independent samples and by use of multisample analyses.

Results: The confirmatory factor analysis showed the psychometric integrity of two subscales: planned physical activity and unplanned physical activity.

Conclusion: The questionnaire is a valid and reliable instrument designed to provide an online behavioural assessment to be used in conjunction with a 12 week personalised fitness programme delivered through the internet.

(*Br J Sports Med* 2005;**39**:e23) <http://bjism.bmjournals.com/cgi/content/full/39/5/e23>

Acute effects of a single open sea air dive and post-dive posture on cardiac output and pulmonary gas exchange in recreational divers

Z Dujic, D Bakovic, I Marinovic-Terzic, *et al*

Objective: To evaluate the cardiopulmonary effects of open sea scuba air diving to 39 m (30 minutes bottom time) with

standard decompression. To account for possible gravity dependent effects of venous gas bubbles, the variables were measured in different post-dive body postures and compared with the baseline values before the dive in the same posture.

Methods: Eight male divers conducted two similar dives on successive days. Their posture before and after the dive were either sitting or supine, in random order. The divers were evaluated before and 30, 60, and 90 minutes after the dive. Venous bubbles were detected by precordial Doppler after the dive in four divers in the supine posture and two divers in the sitting posture.

Results: Arterialised oxygen tension had decreased at all times after the dive (-11.3 mm Hg, $p = 0.00006$), due to decreased alveolar oxygen tension, irrespective of posture. Apart from an increase in the sitting posture 30 minutes after the dive, pulmonary capacity for carbon monoxide diffusion and cardiac index decreased, mostly 60 minutes after the dive (-9% , $p = 0.0003$ and -20% , $p = 0.0002$ respectively). The decrease in cardiac index was greater in the supine posture ($p = 0.0004$), and the physiological dead space/tidal volume ratio increased more in the sitting position ($p = 0.006$).

Conclusions: Field dives are associated with moderate impairments in cardiac output and gas exchange. Some of these impairments appear to depend on the posture of the diver after the dive.

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