NUTRITION, PHYSICAL ACTIVITY AND BONE HEALTH IN WOMEN

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INTRODUCTION

During the last two decades there has been a marked increase in the number of women undertaking physical activity at both competitive and recreational levels. In particular, the number of women involved in endurance aerobic exercise, such as running, has increased rapidly. Regular physical activity is recognized to have many beneficial health related effects, not least of which is a reduction in the rate of hip fracture due to osteoporosis in later life (Cooper et al. 1988). For this reason, exercise is now promoted as having a role in maintaining bone mineral density (BMD) and preventing the development of osteoporosis. However, there is evidence that intensive exercise at a young age may paradoxically reduce BMD.

Such changes in BMD have been found to be linked to profound alterations in the hypothalamic–pituitary–ovarian pathway in athletes that train intensively. Oligomenorrhoea, amenorrhoea, short luteal phases and late menarche have all been demonstrated with increased frequency in such athletes. Whereas secondary amenorrhoea occurs in only 2–5% of the general population it is common in sports in which thinness may be an added advantage to performance, such as running, gymnastics, ice skating and dancing. In the Great Britain National squads of these sports in 1988, 50–100% of women had some form of menstrual dysfunction (Wolman & Harries, 1989). Although oligomenorrhoea and amenorrhoea due to exercise were initially thought to be relatively benign, it is now known that the hypo-oestrogenic state is associated with reduced bone density. Some regions of the skeleton, for instance the vertebral bodies, may be as much as 25% lower in BMD in amenorrhoeic women compared to their eumenorrhoeic peers. In the short term, musculoskeletal injuries are more common, and in the long term concern has arisen that there may be a risk of early osteoporosis and fracture.

Menstrual dysfunction in athletes is probably multifactorial (Myburgh et al. 1992; Fig. 1); severity of training (Feicht et al. 1978; Sanborn et al. 1982; Drinkwater et al. 1984, 1986), low body fat (Frisch & McArthur, 1974; Sanborn et al. 1982), low weight for height (Shangold & Levine, 1982; Marcus et al. 1985; Drinkwater et al. 1986; Glass et al. 1987; Wolman & Harries, 1989; Harber et al. 1991; Myerson et al. 1992) and chronological age (Drinkwater et al. 1986; Glass et al. 1987; Snead et al. 1992) have all been suggested as playing a part. Amenorrhoeic athletes are also more likely to have had a later menarche (Frisch et al. 1981; Drinkwater et al. 1986; Casey et al. 1991), to have started training either before or soon after menarche (Frisch et al. 1981; Drinkwater et al. 1986; Snead et al. 1992) and to have had a previous episode of irregular periods (Shangold & Levine, 1982; Lloyd et al. 1987). But recent evidence suggests that inadequate nutritional intake and abnormal dietary habits may play a pivotal role in the development of menstrual disorders. Athletes
Fig. 1. Non-pathological factors likely to contribute to the development of menstrual irregularity in athletes. Those women with the greatest number of risk factors are most likely to develop oligomenorrhea.

strive hard to attain the perfect shape and this combines with social and cultural pressures to reward a thin, sylph-like figure. In their efforts to achieve the ideal they may restrict food intake or use abnormal methods for weight reduction despite their high energy requirements. In some, frank eating disorders develop. The consequence of this can be a suppression of normal menstrual function. Attention has therefore focused on the proposed relationship between disordered eating habits, calorie restriction, menstrual irregularity and reduced bone density in female athletes. Early identification of those at risk may reduce the incidence of injury and low bone density.

The mechanisms by which any of the above risk factors induce hypothalamic suppression and hence menstrual dysfunction are unclear. One possibility is that activation of the adrenal axis during exercise inhibits the pulsatile release of gonadotrophin releasing hormone. Consistent with this hypothesis is the finding that cortisol levels are mildly elevated in amenorrhoeic women (Ding et al. 1988). Other putative mediators are thyroxine, endorphins and catecholoestrogens, all of which show changes with exercise, but no evidence is currently available to support a direct link. Of course, in any case of primary or secondary amenorrhoea pathological causes must be excluded and the diagnosis of exercise induced amenorrhoea depends on this.

This review assesses the evidence for nutritional disorders in female athletes and the relationship between these and menstrual abnormalities. The impact of menstrual
dysfunction on bone metabolism is reviewed and the potential for intervention is discussed. Readers are referred to reviews by Loucks (1990), De Souza & Metzger (1991) and Loucks et al. (1992) for more detailed data on the hormonal aspects of athletic amenorrhoea.

NUTRITIONAL NEEDS OF THE ATHLETE

ENERGY EXPENDITURE

Energy expenditure can be extremely high during competition and training (McArdle et al. 1986) and in some sports, such as cycle racing and triathlon, athletes may approach the limit for working capacity on a sustained basis. Energy expenditure of cyclists during 10 days of competitive cycling (total race distance 1600 km) has been estimated to be as high as 4110 (sd 504) kcal d⁻¹ (Snyder et al. 1991) and during the Tour de France cyclists may maintain a daily expenditure of 3.5-5.5 times the basal metabolic rate. Although races such as the Tour de France exemplify the enormous energy requirements of some athletes, more moderate levels of competition and training also make high demands. From McArdle et al. (1986) it can be estimated that a 50 kg female long distance runner completing a marathon in 2 h 37 min (6 min mile⁻¹ pace) has an energy expenditure of 1978 kcal for that race alone.

Nutrition of the correct type and quantity is therefore of great importance to the athlete. In order to maintain energy balance it would be expected that energy intakes would be much higher in athletes than in sedentary peers. Yet recent literature suggests that many athletes, particularly females, do not adequately balance their energy demands with nutritional intake.

THE PROBLEMS OF NUTRITIONAL ASSESSMENT

During the last 10–15 years a number of studies have estimated the calorie intake of female athletes for a variety of sports (Table 1). These surveys have generally used three or seven day dietary records, in some cases backed up by 24 h recall. There are doubts of the accuracy of such records, particularly because of the tendency to underestimate intake (Gersovitz et al. 1978). Although some have demonstrated good correlation between measured food intake and dietary recall (Stunkard & Waxman, 1981; Karvetti & Knutts, 1992), Schoeller et al. (1990) reported large differences between 24-h recall methods and the double labelled water method in elite athletes. Similar disparities have also been shown in non-athletic subjects (Bandini et al. 1990).

There are further difficulties in interpreting nutritional surveys in athletes. Much of the data is presented as total calorie intake without adjustment for weight, which makes comparison across sports difficult. Calorie intake is more meaningful when expressed as a function of the biologically active tissue, which is lean body mass (Webb, 1981). Calorie intake may also vary widely according to time of competitive season. Short & Short (1983) found that, in female University team swimmers, intake was 77 % higher during preseason training than at the end of season. Training intensity and competitive level may vary between studies and even between groups used within each study. A single three-day dietary record may not therefore be representative of long term energy intake in athletes.

ENERGY INTAKE IN FEMALE ATHLETES

Despite these drawbacks, it appears that some female athletes have lower energy intakes than might be expected from their level of activity. Highly active women have greater energy requirements than sedentary women, yet many studies report very similar energy
### Table 1. Estimated energy intakes of female athletes from various sports assessed from 24 hour, 2 day, 3 day and 7 day records/recall. Mean ± SD or (range)

<table>
<thead>
<tr>
<th>Activity</th>
<th>n</th>
<th>Calorie intake (kcal day⁻¹)</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Aerobic sports</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Running</td>
<td>19</td>
<td>1973 ± 145</td>
<td>Snead <em>et al.</em> 1992</td>
</tr>
<tr>
<td>Running</td>
<td>5</td>
<td>1690 ± 272</td>
<td>Wilmore <em>et al.</em> 1992</td>
</tr>
<tr>
<td>Running</td>
<td>9</td>
<td>1611 ± 76</td>
<td>Wilmore <em>et al.</em> 1990</td>
</tr>
<tr>
<td>Running</td>
<td>7</td>
<td>1973 ± 350</td>
<td>Mulligan &amp; Butterfield 1990</td>
</tr>
<tr>
<td>Running</td>
<td>9</td>
<td>1933 ± 382</td>
<td>Myerson <em>et al.</em> 1991</td>
</tr>
<tr>
<td>Running</td>
<td>103</td>
<td>1603 ± 488</td>
<td>Pate <em>et al.</em> 1990</td>
</tr>
<tr>
<td>Running</td>
<td>9</td>
<td>1611 ± 76</td>
<td>Schulz <em>et al.</em> 1992</td>
</tr>
<tr>
<td>Running</td>
<td>20</td>
<td>2488 ± 302</td>
<td>Bergen-Cico &amp; Short, 1992</td>
</tr>
<tr>
<td>Swimming</td>
<td>10</td>
<td>1894 ± 634</td>
<td>Risser <em>et al.</em> 1990</td>
</tr>
<tr>
<td>Swimming</td>
<td>9</td>
<td>3988 (5874—2267)</td>
<td>Short &amp; Short, 1983</td>
</tr>
<tr>
<td>Swimming</td>
<td>7</td>
<td>2248 (3532—1516)</td>
<td>Short &amp; Short, 1983</td>
</tr>
<tr>
<td>Swimming</td>
<td>18</td>
<td>1892 ± 446</td>
<td>Benson <em>et al.</em> 1990</td>
</tr>
<tr>
<td>Rowing</td>
<td>24</td>
<td>2340 (3580—1260)</td>
<td>Short &amp; Short, 1983</td>
</tr>
<tr>
<td><strong>Anaerobic/Aerobic sports</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Basketball</td>
<td>9</td>
<td>3240 (3879—1872)</td>
<td>Short &amp; Short, 1983</td>
</tr>
<tr>
<td>Basketball</td>
<td>9</td>
<td>1797 ± 870</td>
<td>Risser <em>et al.</em> 1990</td>
</tr>
<tr>
<td>Volleyball</td>
<td>11</td>
<td>2446 (3199—1144)</td>
<td>Short &amp; Short, 1983</td>
</tr>
<tr>
<td>Volleyball</td>
<td>12</td>
<td>1610 ± 574</td>
<td>Risser <em>et al.</em> 1990</td>
</tr>
<tr>
<td>Lacrosse</td>
<td>7</td>
<td>2219 (3059—1438)</td>
<td>Short &amp; Short, 1983</td>
</tr>
<tr>
<td><strong>Appearance’ sports</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Gymnastics</td>
<td>12</td>
<td>1544 ± 398</td>
<td>Benson <em>et al.</em> 1990</td>
</tr>
<tr>
<td>Ice skating</td>
<td>23</td>
<td>1174 ± 454</td>
<td>Rucinski, 1989</td>
</tr>
<tr>
<td>Dancers</td>
<td>10</td>
<td>1431 ± 500.2</td>
<td>Frusztaier <em>et al.</em> 1990</td>
</tr>
<tr>
<td>Dancers</td>
<td>9</td>
<td>1909 (2909—898)</td>
<td>Short &amp; Short, 1983</td>
</tr>
<tr>
<td>Dancers</td>
<td>16</td>
<td>1701 ± 448</td>
<td>Williford <em>et al.</em> 1989</td>
</tr>
<tr>
<td>Dancers</td>
<td>19</td>
<td>1405 ± 379</td>
<td>Hergenroeder <em>et al.</em> 1991</td>
</tr>
</tbody>
</table>

### Table 2. Energy intake of regularly menstruating non-athletes estimated from 1-day to 7-day dietary recall/records. Mean ± SD

(The data presented in this Table are from subjects used as controls in the studies presented in Table 1)

<table>
<thead>
<tr>
<th>Age</th>
<th>n</th>
<th>Calorie intake (kcal day⁻¹)</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>20.5 ± 4.3</td>
<td>10</td>
<td>1672 ± 641</td>
<td>Frusztaier <em>et al.</em> 1990</td>
</tr>
<tr>
<td>30.6 ± 5.6</td>
<td>5</td>
<td>1744 ± 367</td>
<td>Mulligan &amp; Butterfield 1990</td>
</tr>
<tr>
<td>19.8 ± 1.4</td>
<td>13</td>
<td>1750 ± 368</td>
<td>Risser <em>et al.</em> 1990</td>
</tr>
<tr>
<td>29.6 ± 2.2</td>
<td>8–9</td>
<td>1832 ± 161</td>
<td>Snead <em>et al.</em> 1992</td>
</tr>
<tr>
<td>25.0 ± 4.6</td>
<td>5</td>
<td>1763 ± 420</td>
<td>Wilmore <em>et al.</em> 1992</td>
</tr>
<tr>
<td>26.3 ± 1.5</td>
<td>6</td>
<td>1779 ± 365</td>
<td>Myerson <em>et al.</em> 1991</td>
</tr>
<tr>
<td>13.5 ± 1.2</td>
<td>34</td>
<td>1849 ± 391</td>
<td>Benson <em>et al.</em> 1990</td>
</tr>
</tbody>
</table>

Intakes in the two groups. The mean calorie intake of all the runners in the papers presented in Table 1 is 1874 kcal d⁻¹. Non-athletic women, of a similar age group, presented in Table 2 have an estimated mean intake of 1772 kcal d⁻¹; only 102 kcal d⁻¹ less. To evaluate this further, energy balance studies have compared total energy expenditure and estimated...
Table 3. Categorization of sports according to whether low body mass is likely to improve performance or increase marks obtained during performance

<table>
<thead>
<tr>
<th>Low weight not of specific benefit to performance</th>
<th>Low weight likely to improve performance</th>
<th>Performance judged on aesthetic appeal</th>
<th>Competing in weight categories</th>
</tr>
</thead>
<tbody>
<tr>
<td>Heavyweight rowing</td>
<td>Running</td>
<td>Gymnastics</td>
<td>Judo</td>
</tr>
<tr>
<td>Ball games e.g.:</td>
<td>Middle</td>
<td>Rhythmic gymnastics</td>
<td>Martial arts</td>
</tr>
<tr>
<td>Lacrosse</td>
<td>Long</td>
<td>Figure skating</td>
<td>Rowing, lightweight</td>
</tr>
<tr>
<td>Hockey</td>
<td>Ultra</td>
<td>Ice dance</td>
<td>Wrestling</td>
</tr>
<tr>
<td>Basketball</td>
<td>Crosscountry</td>
<td>Dancing</td>
<td>Weight lifting</td>
</tr>
<tr>
<td>Netball</td>
<td>Orienteering</td>
<td>Competitive</td>
<td></td>
</tr>
<tr>
<td>Tennis</td>
<td>Race walking</td>
<td>Ballet</td>
<td></td>
</tr>
<tr>
<td>Golf</td>
<td>Jumping</td>
<td>Bodybuilding</td>
<td></td>
</tr>
<tr>
<td>Sprinting</td>
<td>Pole vault</td>
<td>Synchro swimming</td>
<td></td>
</tr>
<tr>
<td>Field events (throwing)</td>
<td>Rowing, cox</td>
<td>Diving</td>
<td></td>
</tr>
<tr>
<td>Contact sports</td>
<td>Jockeys, flat-racing</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Swimming</td>
<td>Cycling</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Water polo</td>
<td>Triathlon</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Skiing</td>
<td>Climbing, competitive</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Speed skating</td>
<td>Windsurfing, Olympic</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Luge, bobsleigh</td>
<td>Sailing, some classes</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Energy intake in female runners. Energy deficits reported in such literature vary from $-221$ to $-788$ kcal $d^{-1}$ (Myerson et al. 1991; Bergen-Cico & Short, 1992; Schulz et al. 1992; Wilmore et al. 1992).

This suggests that female athletes may be restricting nutritional intake, most noticeably in endurance and aesthetic sports. However, the desired body weight patterns vary by sport (see Table 3), and it is possible that the demands of individual sports influence the energy intake and nutritional habits of the athlete.

**Sports in which low weight is of no direct benefit**

In sports such as hockey, volleyball, lacrosse, basketball and heavyweight rowing, athletes may lose fat to improve fitness but performance is unlikely to be improved by low weight. Overall energy intake appears to be higher in these activities and there are few reports of restricted eating or abnormal eating habits.

Young swimmers may be an exception to this. A study of 487 female and 468 male swimmers (age 9–18) found that girls were particularly likely to misperceive themselves as overweight, and although only 16.5% of swimmers were actually classified as overweight, 41.7% had attempted to lose weight. Of the girls who were actively dieting, 17.9% were underweight and 60.5% were average weight (Dummer et al. 1987). The decision to lose weight was based on misperceptions of body image induced by peer group comments rather than on parental or coach advice.

**Sports in which low weight may improve performance**

Sports which involve movement against gravity favour those with a low body mass. For example, running performance is improved by weight loss (Wilmore & Costill, 1987; Brandon & Boileau, 1992) and vertical jump power is negatively correlated with body weight in skaters (Delistraty et al. 1992). Both athletes and coaches may aim for a weight that is inappropriate for an individual athlete. Top distance runners are usually very slim and this combines with cultural influences to increase the desire for leanness. Anecdotal reports
abound of the adolescent athlete who finds that a small amount of weight loss results in improved performance, reinforcing the desire to lose yet more weight and precipitating the onset of severe food restriction or abnormal weight loss habits.

Middle and long distance runners have high energy expenditure yet estimated nutritional intakes have been very low (Schulz et al. 1992; Wilmore et al. 1992). Pate et al. (1990) found that energy intakes in 103 female runners (age range 25–34 years) and 74 sedentary controls were remarkably similar even when adjusted for body size (27.5 (SD 0.9) v. 26.0 (SD 1.3) kcal kg⁻¹ daily). Baer (1993) also reported almost identical intakes in eumenorrhoeic runners and sedentary controls (1944 (SD 75) v. 1950 (SD 56) kcal d⁻¹).

Not all authors agree that there is nutritional restriction in runners. Mixed results were obtained by Mulligan & Butterfield (1990) who found that intake was highest in a group of moderately or very active runners compared to non-runners (37.5, 37.0 and 32.2 kcal kg⁻¹ daily respectively) although when body weight was accounted for it was evident that the very active runners may have been relatively energy restricted (49.0, 43.5 and 43.7 kcal (kg fat free mass)⁻¹ daily in moderately active, very active and sedentary respectively). Relatively high energy intakes (2397 (SD 104) kcal d⁻¹) have been recorded in Olympic marathon hopefuls (Deuster et al. 1986) and Blair et al. (1981) estimated a calorie intake of 2386 kcal d⁻¹ in runners and 1871 kcal d⁻¹ in controls.

Sports which are judged on aesthetic appeal

In this group, which includes gymnastics, dancing and ice skating, weight is lost in a bid for higher marks. Research supports this concept; women placed in a National gymnast team had markedly lower body fat levels than non-placers (Falls & Humphrey, 1978) and elite gymnasts have much lower calorie intakes per unit body weight than subelite gymnasts (van Erp-Baart et al. 1989). Dancers also have a low intake; mean calorie intake was similar in 16 female dancers and 11 controls (1702 (SD 448) v. 1603 (SD 405) kcal d⁻¹ respectively; Williford et al. 1989). And in a study of adolescents (mean age 12.9 years), Benson et al. (1990) found that intakes were similar in swimmers, gymnasts and non-athletic controls (39.2 (SD 13.4), 39.5 (SD 10.6), 39.4 (SD 13.3) kcal kg⁻¹ daily respectively).

Female bodybuilders also fall into this category because they are judged on muscle definition which is improved when subcutaneous fat is low. However, they also compete in weight categories and in terms of eating habits they are better considered with the next group.

Sports in which the athlete competes in a weight category

In for instance judo, karate, tae kwon do and weight lifting, athletes are classed according to their body weight. Rowing also comprises a light weight category in which women must be 59 kg or under. Some athletes have a natural weight above that of the weight category in which they wish to compete. They therefore need to reduce weight before competitions (Short & Short, 1983; McCargar et al. 1993; Walberg-Rankin et al. 1993). Although many are able to do this over several months, others use starvation diet methods or resort to illicit practices such as purgation or the use of diuretics (Walberg & Johnston, 1991).
Table 4. Energy intake of female athletes estimated from 1-day to 7-day dietary recall and records

(Comparison of energy intake between eumenorrhoeic (EU) and amenorrhoeic (AM) athletes)

<table>
<thead>
<tr>
<th>Reference</th>
<th>EU (kcal day⁻¹)</th>
<th>n</th>
<th>AM (kcal day⁻¹)</th>
<th>n</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Runners</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2490</td>
<td>1582*</td>
<td>8</td>
<td>2489</td>
<td>152*</td>
<td>11</td>
</tr>
<tr>
<td>2210 ± 141</td>
<td>2151 ± 152*</td>
<td>12</td>
<td>1715 ± 281</td>
<td>1272 ± 136</td>
<td>14</td>
</tr>
<tr>
<td>2419 ± 132</td>
<td>1732 ± 236</td>
<td>6</td>
<td>1971 ± 145</td>
<td>2046 ± 115</td>
<td>12</td>
</tr>
<tr>
<td>1937 ± 383</td>
<td>1623 ± 145</td>
<td>14</td>
<td>1690</td>
<td>1730 ± 152*</td>
<td>12</td>
</tr>
<tr>
<td>1965 ± 98</td>
<td>1222 ± 136</td>
<td>11</td>
<td>1817</td>
<td>1730 ± 152*</td>
<td>12</td>
</tr>
<tr>
<td>1715 ± 281</td>
<td>1222 ± 136</td>
<td>11</td>
<td>1984</td>
<td>1730 ± 152*</td>
<td>12</td>
</tr>
<tr>
<td>1690 ± 227</td>
<td>1781 ± 283</td>
<td>8</td>
<td>2151 ± 236</td>
<td>2151 ± 236</td>
<td>6</td>
</tr>
<tr>
<td>1817 ± 523</td>
<td>1832 ± 463</td>
<td>38</td>
<td>1627 ± 75*</td>
<td>1116 ± 365*</td>
<td>19</td>
</tr>
<tr>
<td>1944 ± 45</td>
<td>1627 ± 75*</td>
<td>10</td>
<td>2151 ± 236</td>
<td>2151 ± 236</td>
<td>6</td>
</tr>
<tr>
<td>Dancers</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1405 ± 379</td>
<td>1116 ± 365*</td>
<td>19</td>
<td>1627 ± 75*</td>
<td>1116 ± 365*</td>
<td>19</td>
</tr>
</tbody>
</table>

Mean ± sd. Comparison between women with amenorrhoea and regular menstrual cycles: *P < 0.05.

THE RELATIONSHIP BETWEEN AMENORRHoea AND LOW ENERGY INTAKE

ENERGY INTAKE OF AMENORRHoeIC ATHLETES COMPARED TO EUAMENORRHoeIC SUBJECTS

Several recent reports have suggested that the calorie intake of amenorrheic athletes may be even lower than that of eumenorrheic subjects and that restriction of energy intake may be causally related to menstrual dysfunction. Some workers have also shown that amenorrheic athletes weigh less than eumenorrheic athletes (Galle et al. 1983; Marcus et al. 1985; Drinkwater et al. 1986; Harber et al. 1991) and have a lower percentage body fat (Sanborn et al. 1982; Glass et al. 1987) although there is much overlap between the two groups.

Nelson et al. (1986) used a three-day dietary record to compare the intakes of 11 amenorrheic and 17 eumenorrheic highly trained runners. They reported the mean energy intake of amenorrheic runners to be 520 kcal d⁻¹ less than eumenorrheics (see Table 4). The difference in calorie intake was highly significant (P < 0.005) when expressed as a function of lean body mass. Similar findings were reported by Kaiserauer et al. (1989) although the intake of eumenorrheic sedentary controls was very similar to that of the amenorrheic women. Baer (1993) also found lower intakes (P < 0.05) in amenorrheic runners compared to both eumenorrheic runners and controls (1627 (SD 75), 1944 (SD 45), 1950 (SD 56) kcal d⁻¹ respectively). And in adolescent dancers, Hergenroeder et al. (1991) estimated intakes of 1405 and 1116 kcal d⁻¹ (P = 0.03) in eumenorrheic and amenorrheic subjects respectively using a 24-h recall and food frequency form.

Four out of a further seven studies on runners have also shown lower total calorie intakes in the amenorrheic athletes but these differences have not been statistically significant, largely because of the wide variance of the mean (see Table 4). In one of these studies (Drinkwater et al. 1984) energy expenditure was estimated to be higher in the amenorrheic subjects compared to the eumenorrheics and the two groups were therefore not directly comparable. In only two studies has calorie intake been shown to be higher in amenorrheic athletes compared to eumenorrheic athletes (Snead et al. 1992; Wilmore et al. 1992). Most of these reports are based on small sample numbers but Watkin et al.
Fig. 2. Percentage of total energy supplied by main constituents of diet in amenorrhoeic and eumenorrhoeic female runners. Data accumulated from Nelson et al. (1986), Myerson et al. (1991), Sneed et al. (1992), Kaiserauer et al. (1989), Drinkwater et al. (1984), Deuster et al. (1986), Watkin et al. (1991) and Baer (1993). AM = amenorrhoeic, EU = eumenorrhoeic.

(1991) surveyed 38 matched pairs of eumenorrhoeic and oligomenorrhoeic ultra-marathon runners. Similar energy intakes were found in both groups; however, of the 38 oligomenorrhoeic athletes only 53% were amenorrhoeic, the remainder having a cycle length of 35–90 d. These oligomenorrhoeic athletes may represent a subset with differing eating habits which may have skewed the results.

It is unclear whether short term energy restriction has such profound effects on menstrual function as long periods of reduced intake. Walberg-Rankin et al. (1993) surveyed six bodybuilders for two months which included a competition. Weight loss of 0.7–10.8% during the month prior to competition was achieved in most by gradual energy restriction. Mean energy intake in the month before the competition varied between 1536 (SD 28) and 1839 (SD 33.8) kcal d⁻¹. Immediately after the competition, intake rose to 3237 (SD 59) kcal d⁻¹. Of the three women not on the oral contraceptive, two reported missing one menstrual cycle during the past year although none had prolonged menstrual irregularity. Other workers have shown that it is possible to reduce weight slowly, by alternating constituents of the diet rather than energy restriction, without any physiological disturbance (McCargar et al. 1993).

Chronic nutritional restriction therefore appears to be a causative factor in the development of amenorrhoea, in addition to other physiological, psychological or training stresses, but data are scant for sports other than running, gymnastics and dancing. Even within this subgroup of athletes, opinion is divided. Some authors have suggested that amenorrhoeic runners are metabolically more efficient and do not require a high energy intake (Mulligan & Butterfield, 1990). Others have suggested that in runners the calorie deficit is overestimated and is more likely to be due to either consistent under-reporting of food intake or to restricted eating on the days of the dietary record (Wilmore et al. 1992). Under-reporting does occur in some subsets of women more than others: Bandini et al. (1990) demonstrated energy deficits of 562 and 1455 kcal d⁻¹ in non-obese and obese subjects respectively and Prentice et al. (1986) found a difference of 837 kcal d⁻¹ in obese subjects but no deficit in lean subjects. The suggestion in both of these studies is that obese women under-report food intake. It is possible that, with disturbances in body image, amenorrhoeic women behave in a similar way. In addition, self-reporting of physical activity is only moderately accurate (Klesges et al. 1990) with up to 300% overestimate of aerobic activity in some subjects. Studies relying on self-reporting of activity may therefore exaggerate any energy deficit that is present.
CONSTITUENTS OF THE DIET

There may be other nutritional factors contributing to the development of menstrual disturbance. Deuster et al. (1986) suggested that the total calorie intake may not be as important as the balance between the constituents of the diet and in particular that a low intake of fat may play some role in this relationship. In eight papers in which the constituents of the diet are shown, fat contributed 27.1-35.7% of the calories in amenorrhoeic athletes and 31.3-37.9% in eumenorrhoeic athletes (Fig. 2). However, the difference in fat intake was significant in only two papers and although the overall trend is for fat to provide a lower percentage of calories in amenorrhoeic athletes these differences are remarkably small. It seems unlikely that such small differences in fat intake could have profound effects on the pituitary-ovarian axis but this is another area which requires further work.

VEGETARIANISM

Diet may influence sex hormone production in other ways. In small studies in non-athletes, a vegetarian weight reduction diet has been associated with low oestrogen and progesterone levels, short luteal phases (Schweiger et al. 1987) and anovulation (Pirke et al. 1986). These changes were related to severity of weight loss and were not seen in non-vegetarian diets. Other authors have also noted an increased frequency of menstrual irregularity in vegetarian compared to non-vegetarian women (Lloyd et al. 1987; Pedersen et al. 1991) and some have shown a negative correlation between dietary fibre and sex hormone levels (Barbosa et al. 1990).

In athletes, Lloyd et al. (1991) found that oligomenorrhoeic collegiate women consumed greater amounts of dietary fibre than their eumenorrhoeic counterparts and suggested dietary habits as a cause of their menstrual irregularity. Several studies of small numbers of amenorrhoeic athletes have suggested that vegetarianism is more common than in eumenorrhoeic athletes (Brooks et al. 1984; Slavin et al. 1984; Kaiserauer et al. 1989) and this may be a fruitful area for further longitudinal research.

VITAMINS AND TRACE MINERALS

Low intake of a number of nutrients is common in athletes, particularly those with amenorrhoea. Low protein intake (Kaiserauer et al. 1989) and low intakes of some vitamins and trace minerals (Deuster et al. 1986; Kaiserauer et al. 1989; Rucinski, 1989; Benson et al. 1990; Myerson et al. 1991; Bergen-Cico & Short, 1992) have all been demonstrated. In most, the low intakes are due to an overall reduced food intake rather than a specific deficiency of one nutrient. Whether these deficiencies are linked to menstrual disturbance is not yet known.

EATING DISORDERS

Analysis of calorie intake alone may present only half the picture and for this reason it may have been difficult to link directly energy intake with amenorrhoea. Current literature suggests a high incidence of anorexic or bulimic types of behaviour in some athletes, particularly runners and dancers. Preoccupation with weight and food, a desire to maintain a low body weight or body fat and intensive exercise are all hallmarks of anorectic behaviour. Both anorexia and bulimia are associated with menstrual disturbance and it may be that disordered eating rather than energy intake per se is the main contributor to the high incidence of amenorrhoea. Alternatively these eating habits may be normal.
Table 5. *Adapted from the Diagnostic and Statistical Manual of Mental Disorders (American Psychiatric Association, 1987)

<table>
<thead>
<tr>
<th>DSM-III-R* diagnostic criteria for anorexia nervosa</th>
<th>DSM-III-R* diagnostic criteria for bulimia nervosa</th>
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<tbody>
<tr>
<td>1. Refusal to maintain weight over a minimal normal weight for age and height, i.e. weight loss leading to maintenance of body weight 15% below that expected or failure to make expected weight gain during a period of growth leading to body weight 15% below that expected</td>
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<td>2. Intense fear of gaining weight or becoming fat, even though underweight</td>
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<td>3. Disturbance in the way in which one’s body weight, size, or shape is experienced, e.g. the person claims to ‘feel fat’ even when emaciated or believes that one area of the body is ‘too fat’, even when obviously underweight</td>
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<td>4. In females, absence of at least three consecutive menstrual cycles when otherwise expected to occur (primary or secondary amenorrhoea)</td>
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<tr>
<td>1. Recurrent episodes of binge eating (rapid consumption of a large amount of food in a discrete period of time, usually &lt; 2 h)</td>
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<td>2. A feeling of lack of control over eating behaviour during the eating binges</td>
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<td>3. Regularly engaging in self-induced vomiting, the use of laxatives or diuretics, strict dieting or fasting, or vigorous exercise in order to prevent weight gain</td>
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<td>4. A minimum of two binge eating episodes a week for at least three months</td>
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amongst elite athletes in whom the correct timing and constituents of the diet may be very different from the rest of the population. It is likely too that within any group of athletes there are some who use exercise to become thin.

**EATING DISORDERS IN THE GENERAL POPULATION**

Estimates of eating disorders, using strict DSM III criteria of the American Psychiatric Association (1987; see Table 5), vary from 1.0 to 4.8% of female student populations (Schotte & Stunkard, 1987; Drewnowski et al. 1988; Kurtzmann et al. 1989). Using eating habits questionnaires in 2544 high school girls, Johnson & Whitaker (1992) found that 10.2% frequently had episodes of binge eating, 40% frequently self-induced vomiting and 1.6 and 5.1% regularly used laxatives or diet pills respectively. When questioned on dieting habits 43.5% had dieted for less than 28 days and 18.5% had dieted for longer than this. An alarming 7.4% had fasted for longer than three days. Similarly high values for the incidence of dieting (61% black girls, 77% white girls), fasting (33% of dieters) and purging (up to 18%) in high school girls were found by Emmons (1992) in a study of 1269 male and female high school students. Thus although the estimates for strictly defined eating disorders in the general population may be relatively low, abnormal eating patterns and weight control practices are common in this age group of girls. Indeed, in a large study of 603 elite female athletes and 522 controls, Sundgot-Borgen & Larsen (1993) found similar numbers with eating disorders (12 and 11% respectively) or who were at risk of developing an eating disorder (22 and 26%). It is against this background that the eating habits and prevalence of these disorders in athletes must be considered.

**EATING DISORDERS IN ATHLETES**

Many athletes have strange but not pathological eating habits and this makes it difficult to estimate the prevalence of true eating disorders in the athletic population. In addition to this, eating disorders are, by their nature, secretive activities and many will not admit to
such a problem. Roommates and relatives may be entirely unaware that an eating disorder exists. Studies that rely on questionnaires may therefore underestimate this incidence.

Most studies on the athletic population do not use strict DSM criteria and comparison with the normal population is therefore difficult. Research on athletes has usually entailed the Eating Disorders Inventory, the Bulimia Test – Revised or the Eating Attitudes Test (EAT) in which the athlete responds to statements such as “I vomit after I have eaten”, “I am terrified about being overweight”, “I think about burning up calories when I exercise”. A progressive rating of replies from ‘always’ to ‘never’ enables the degree of disordered eating to be assessed. Athletes may under-report symptoms and behaviours associated with eating disorders and it has been suggested that both clinical interviews and questionnaires may provide a better estimate of incidence (Sundgot-Borgen, 1993).

It has been suggested that intensive exercise itself may precipitate eating disorders. Gleaves et al. (1992) tested the hypothesis that weight loss due to running leads to body image disturbance and places runners at increased risk of bulimic disorders. They found no difference in measures of depression, bulimia or body image disturbance between the running group and normal controls whereas bulimia nervosa patients differed from both groups in most measures. However, Davis et al. (1990) identified two groups of women within a cohort of 112 regularly exercising females: ‘dieters’ (57%) and ‘non-dieters’ (43%). The dieters expressed weight and diet concerns equivalent to eating-disordered patients and much more frequently than the non-dieters. They also engaged in more frequent and more intense exercise than the non-dieters. Davis et al. concluded that “regular participation in a fitness programme may be causally related to excessive concern with weight and dieting”. It is possible that, within any group of athletes, those with a tendency towards body image preoccupation are precipitated into a full eating disorder by the combined sociocultural influences and the demands of sport.

PREVALENCE OF EATING DISORDERS IN DIFFERENT ATHLETIC GROUPS

Sports in which low weight is not an advantage

In a study of elite female Norwegian athletes (Sundgot-Borgen & Larsen, 1993), women competing in ball games and power sports (sprinting, discus, shot put etc.) had a very similar prevalence of abnormal weight control habits in comparison with non-athletic controls (8, 6, and 7% respectively). Downhill skiers, golfers and others involved in technical sports also had a lower prevalence of such habits than those in ‘appearance’ and weight dependent sports (10, 16 and 17% respectively). Estimates of eating disorders in young swimmers are high but are also no different from those of the non-athletic population of the same age (Dummer et al. 1987; Benson et al. 1990; Rosenvinge & Vig, 1993).

Sports in which low weight may improve performance

A large survey was undertaken by Runner's World magazine in the USA; of the 1908 female runners that responded, 38% reported binge eating at least once a month and in 6% this occurred at least three times a week. Purging was common: 26% had purged at least once and 4% purged at least three times a week (Brownell et al. 1988). These results are similar in many ways to those of high school girls in the USA but it is possible that athletes with significant eating problems failed to return the questionnaire. Prussin & Harvey (1991) support a high incidence of bulimic behaviour in female runners: 19% of their cohort (n = 174) fulfilled DSM-IIIIR criteria for bulimia.
Some of the variation in results may be due to the wide range of abilities in large questionnaire based studies. Weight control habits in highly competitive athletes may be more common than in lower achievers (Mulligan & Butterfield, 1990). In one study, 14% of 125 female marathon runners were symptomatic for anorexia and elite runners were more likely to have high EAT scores ($P < 0.05$). Eighty per cent of the runners with both the physical and psychological features of anorexia nervosa were reported to be highly successful competitors (Weight & Noakes, 1987).

**Sports which are judged on aesthetic appeal**

Estimates of eating disorders are high in a number of studies in this group of athletes. In 42 female college gymnasts assessed using the Michigan State University Weight Control Survey, all were actively dieting and 62% were using at least one form of pathological weight control (Rosen & Hough, 1988). Two-thirds of the sample had been told by their coaches that they were too heavy, following which 75% resorted to weight control methods of which self-induced vomiting, diet pills and fasting were most frequently used. In a larger study of 218 female gymnasts (mean age 19.4 years), 48.6% used exercise specifically to burn calories, 49.1% had fasted or used a strict diet in the last year and 58.7% ate uncontrollably at least two or three times a month.

A lower incidence of disordered eating has been found in prepubertal gymnasts (Benson et al. 1990). Body fat stores are lower before puberty and the typical 'ideal' gymnast figure more closely resembles that of the prepubertal girl than a fully mature figure. It becomes harder to achieve this as the athlete ages, which may account for the increased frequency of disordered eating in the older age group.

Rucinski (1989) found 48% of female ice skaters to have EAT scores in the range of clinical anorexia nervosa and also found that energy intake was negatively correlated with EAT scores. In dancers, self-reported anorexia or bulimia ranges from 11% in highly select dancers to 33% in regional and national companies and University dancers (Brooks-Gunn et al. 1987; Evers, 1987; Hamilton et al. 1988).

**Sports in which the athlete competes in a weight category**

Female weight lifters (including a subset who competed in body building) have been found to score highly on the Drive for Thinness subscale of the Eating Disorders Inventory, and of those that competed 67% were terrified of becoming fat, 50% experienced uncontrollable urges to eat and 42% had a history of anorexia (Walberg & Johnston, 1991). Data from male athletes in these sports suggest that they might be at high risk of rapid weight reduction measures in order to 'make weight' (Short & Short, 1983). Methods employed by male wrestlers to induce weight loss include laxative abuse, self-induced vomiting and rapid dehydration methods (saunas, sweat suits, diuretics). Similar problems have been found in American majorettes (Humphries & Gruber, 1986).

**IS THERE A RELATIONSHIP BETWEEN DISORDERED EATING IN ATHLETES AND MENSTRUAL DYSFUNCTION?**

In the study of eating habits in high school girls by Johnson & Whitaker (1992), the relative risk for secondary amenorrhoea was 4.17 for frequent binge purging and 2.59 for weight fluctuation due to weight control measures. Amenorrhoea is sometimes the first sign in anorexia nervosa and the DSM III (American Psychiatric Association, 1987) criteria for this disorder include amenorrhoea (Table 5). In bulimia nervosa endocrine disturbance often results in menstrual irregularity and sometimes amenorrhoea, although it is not a
criterion for diagnosis (Table 5). Eating disorders are therefore likely candidates for a key role in the development of amenorrhoea in athletes.

During a study on diet in long distance runners, Gadpaille et al. (1987) noted a greater preoccupation with diet in amenorrhoeic subjects compared to cyclic women. Analysis of their group of 13 amenorrhoeic and 19 regularly menstruating runners showed that while nearly two thirds of amenorrhoeic runners had evidence of an eating disorder as defined by DSM III criteria, no eumenorrhoeic athlete was affected. A quarter of the amenorrhoeic runners had evidence of a bipolar disorder or major depression and there was a high incidence of first or second degree relatives with a major affective disorder. None of the cyclic women had a depressive disorder and very few had relatives affected. They suggest that athletic amenorrhoea may be either a variant of anorexia nervosa or may be a defence against one of the other disorders, particularly depression (running is known to ameliorate symptoms of depression; Harris, 1986). Higher EAT scores and subscales of the score have also been found in amenorrhoeic runners compared to eumenorrhoeic runners (Myerson et al. 1991).

Prolonged amenorrhoea has been strongly associated with dieting in dancers. Brooks-Gunn et al. (1987) found that 50% of amenorrhoeic dancers reported anorexia nervosa as opposed to only 13% of dancers with normal periods. The amenorrhoeic dancers weighed less and were leaner \( (P < 0.05) \) than their eumenorrhoeic peers and had higher scores on the EAT26 scale \( (P < 0.01) \). Although some workers have found no differences between the incidence of either overt anorexia and bulimia or altered eating behaviour or attitudes in amenorrhoeic athletes (Myburgh et al. 1992; Snead et al. 1992), the majority of workers have shown a trend towards some form of dysfunctional eating or weight control in those with menstrual abnormalities (Walberg & Johnston, 1991; Wilmore et al. 1992).

Although there have been few large scale studies on amenorrhoeic athletes, disordered eating is strongly implicated in the development of menstrual irregularity and this relationship is starting to be taken seriously. In 1990, the National Collegiate Athletic Association of the United States released a videotape series with written information on anorexia nervosa and bulimia. This was aimed at all levels of sport from administrators to athletes and included lists of specific warning signs for both conditions (Wilmore, 1991). The Sports Council of Great Britain and most British bodies governing sports do not yet produce information that is readily available to all levels of sport with the exception of the British Amateur Athletic Federation. A booklet, produced by the International Amateur Athletic Federation and aimed at coaches, is available through their bookshop.

**BONE METABOLISM IN ATHLETES**

**THE EFFECT OF EXERCISE ON BONE**

Julius Wolff (1892) first suggested that bone responds to mechanical stress to increase strength at areas of high strain. Animal models support the concept that dynamic loading of bone results in increased bone strength at predicted sites (Lanyon, 1992). In athletes, BMD of the femur, pelvis, tibia and os calcis have all been shown to be high in athletes undertaking weight-bearing sport (Heinrich et al. 1990; Risser et al. 1990; Wolman et al. 1991; Slemenda & Johnston, 1993; Wilson et al. 1994b), and in tennis players BMD is higher in the dominant arm than on the non-dominant side (Dalén et al. 1985; Pirnay et al. 1987). In rowers, BMD is high in the lumbar spine which correlates well with their greater back strength (Wolman et al. 1990) and bodybuilders have higher lumbar BMD than runners (Heinrich et al. 1990). Indeed, BMD of spine and femur has been shown to be highly correlated with muscle strength (Pocock et al. 1989; Conroy et al. 1993; Eickoff...
Bone density has also been shown to increase during a training programme (Margulies et al. 1986) and in small cross-sectional studies higher bone density has been demonstrated in those who weight train compared to those who are aerobically trained (Davee et al. 1990; Heinrich et al. 1990).

The beneficial effect of regular exercise on BMD occurs not only at a young age, but also appears to continue at least until the menopause (Jacobson et al. 1984; Wilson et al. 1994b) and possibly beyond (Talmage et al. 1986). In addition, physical activity may be a powerful tool in reducing hip fractures associated with older age (Cooper et al. 1988) although some of this reduction may be due to improvements in muscle strength, coordination and balance rather than greater BMD. To date most prospective studies using exercise programmes have measured changes in BMD of spine or radius rather than hip. Therefore there is no evidence yet which demonstrates improvements in hip BMD with exercise. These studies are awaited but it is hoped that exercise prescription may be useful in reducing the risk of osteoporosis later in life.

THE EFFECT OF MENSTRUAL IRREGULARITY ON BONE

Early studies on BMD in athletes with amenorrhoea showed reduced BMD compared to sedentary controls or eumenorrhoeic athletes (Cann et al. 1984; Drinkwater et al. 1984; Marcus et al. 1985). Many others have now confirmed these findings especially in the lumbar spine (Nelson et al. 1986; Lindberg et al. 1987; Wolman et al. 1990; Wilson et al. 1994b). In the largest study of its kind, Drinkwater et al. (1990) demonstrated a linear relationship between vertebral BMD and menstrual history in 97 active women. Those who had a long history of oligomenorrhoea had a mean BMD which was 17% lower than those who had always had regular periods. This linear relationship was also demonstrated in our laboratory. In a study of 50 runners we found that mean lumbar BMD in amenorrhoeic runners was 16% below that of eumenorrhoeic subjects and 6% lower than an oligomenorrhoeic group. In a few of the amenorrhoeic group, BMD was as much as three standard deviations below the mean of age matched European data (Wilson et al. 1994b).

There is less agreement about the effect of amenorrhoea on weight bearing bones in which the mechanical stresses of exercise may offset bone loss. Reduction in femoral shaft BMD in amenorrhoeic active women has been noted by one author (Drinkwater et al. 1990) but Wolman et al. (1991) found no difference in femoral shaft BMD between amenorrhoeic and eumenorrhoeic runners, rowers and dancers. Femoral neck and total leg BMD have also been shown to be well maintained despite amenorrhoea (Drinkwater et al. 1990; Myerson et al. 1992; Snead et al. 1992). However, in our recent study of 24 amenorrhoeic national and international standard runners BMD was low in the proximal femur despite the positive osteogenic effects of running (Wilson et al. 1994b). In the neck of the femur BMD was 0·56 SD below the age matched European mean and 16% lower than in eumenorrhoeic runners. If the differential in BMD between these athletes and the normal population persists into later life their risk of hip fracture may be increased as much as five times (Cummings et al. 1993).

MECHANISM OF REDUCED BONE DENSITY IN AMENORRHOEA

Sex hormones are important in bone turnover; bone resorption is reduced in the presence of oestrogen and progesterone is a trophic hormone for bone (Prior, 1990). In athletic amenorrhoea levels of follicle stimulating hormone and luteinizing hormone are low due to suppression of hypothalamic gonadotrophin releasing hormone. In turn oestrogen production in the ovary is reduced and prolonged low levels may result in increased bone
resorption with loss of bone minerals. Bone biopsy may reveal the resorption surface to be greatly increased while the bone formation surface remains normal (Warren et al. 1990).

CALCIUM INTAKE AND BONE DENSITY IN ATHLETES

Bone mass is higher in children and adolescents with a high calcium intake (Chan, 1991; Sentipal et al. 1991) and this may result in increased bone mass in later life (Matković et al. 1979). Work on healthy premenopausal women supports a role for dietary calcium in the development of bone, particularly when associated with exercise (Kanders et al. 1988; Halioua & Anderson, 1989). The synergic effect of calcium and exercise on bone has also been demonstrated in animals (Lanyon et al. 1986).

A positive linear relationship between trabecular BMD in the lumbar spine and calcium intake in athletes has been demonstrated by Wolman et al. (1992), a finding that was independent of menstrual status and which has not been shown in other studies (Nelson et al. 1986; Grimston et al. 1990; Heinrich et al. 1990). These differences may be due to the methods used, particularly in assessing calcium intake. Alternatively the relationship between calcium intake and bone mineral content may not be linear. Kanders et al. (1988) showed a positive relationship between calcium intake and vertebral BMD in normal healthy eumenorrhoeic women but not above a daily intake of 800–1000 mg.

Low calcium intakes have been reported in many athletes (Rucinski, 1989; Benson et al. 1990; Pate et al. 1990; Bergen-Cico & Short, 1992; Delistraty et al. 1992; Frederick & Hawkins, 1992; Stensland & Sobal, 1992) particularly in amenorrhoeic women. Low oestrogen levels are associated with decreased intestinal absorption of calcium and increased urinary loss (Nordin & Heaney, 1990), so dietary calcium requirements may be even higher in amenorrhoeic athletes. Both Marcus et al. (1985) and Nelson et al. (1986) found that 55% of amenorrhoeic athletes failed to meet the recommended daily allowance for calcium compared to 35–40% of cyclic women. Kaiserauer et al. (1989) also noted a lower intake in amenorrhoeic compared to eumenorrhoeic runners (600 mg v. 1200 mg d⁻¹, P < 0.05). Yet it remains unclear how much of the variance in BMD in amenorrhoeic athletes is due to low calcium intake.

CONSEQUENCES OF LOW BONE DENSITY IN ATHLETES

STRESS FRACTURES

Lloyd et al. (1986) reviewed the medical records of 207 collegiate athletes and found X-ray confirmed fractures (type of fracture not defined) in 9% of regularly menstruating women and 24% of women with irregular or absent menses. For dancers, two papers report a relationship between bone injuries or stress fractures and amenorrhoea (Warren et al. 1986; Benson et al. 1989) and a survey of 240 female athletes showed a higher incidence of stress fractures in those with fewer than five menses per year (49%) compared to those with 10 or more menses per year (29%) (Barrow & Saha, 1988). However, two reports have refuted a relationship between menstrual history and stress fractures although both contained small numbers of subjects (Frusztajer et al. 1990; Grimston et al. 1990).

It remains unclear whether the increased rate of stress fractures in amenorrhoeic athletes is related to low BMD. Femoral bone density has been shown to be low in young male military recruits with femoral stress fractures (Pouilles et al. 1989) but Carbon et al. (1990) assessed elite female runners with and without stress fractures and found no difference in the femoral BMD between the two groups. Others have also described a lack of association between tibial BMD and stress fractures in military recruits (Milgrom et al. 1989).
MUSCULOSKELETAL INJURIES

Stress fractures may not be the only injury more prevalent in amenorrhoeic athletes. Participants in a 10 km race who responded to a questionnaire were more likely to have taken time off training owing to any form of musculoskeletal injury if they had irregular menses (Lloyd et al. 1986). In Benson's study (Benson et al. 1989) of 49 female dancers, those with abnormal menses had more 'bone injuries' (mean = 15·0) than normally menstruating dancers (mean = 5·0; \( P < 0·05 \)). Additionally, dancers with a low body mass index (\( < 19·0 \text{ kg m}^{-2} \)) had a greater duration of low grade musculoskeletal injury (mean = 24·1 d) than those with a higher body mass index (mean = 11·6 d; \( P < 0·05 \)).

More severe bone injury also occurs in amenorrhoeic athletes. In dancers, scoliosis was found to be more common in those with delayed menarche and in whom anorectic behaviour was more prevalent (Warren et al. 1986). Warren et al. (1990) also described a 20 year old ballet dancer with long standing anorexia nervosa, primary amenorrhoea and low BMD, who suffered femoral head collapse. Recently we have reported an osteoporotic fracture in the neck of the humerus of a 30 year old marathon runner with a history of anorexia and low bone density (Wilson & Wolman, 1994).

LONG TERM CONSEQUENCES

Amenorrhoeic athletes may be at risk of premature osteoporosis and fractures but as yet there is little long term information on the natural history of bone metabolism in this condition. Bone mass peaks at approximately 30–35 years of age and from the fourth decade onwards there is a progressive decline in bone density of 0·5–1·5 % per year (Riggs & Melton, 1986; Riggs et al. 1986; Slemenda et al. 1987) which increases (3–8 % per year) in the immediate postmenopausal years (Riggs & Melton, 1986). The lifetime risk of hip fracture has been related to bone mass; the higher the bone mass at any age, the lower the lifetime risk of fracture (Melton et al. 1988). Eumenorrhoeic athletes, who have a high peak bone mass, are likely to have reduced risk of osteoporotic fracture whereas amenorrhoeic athletes may never achieve their maximum potential bone mass.

BMD may increase when menstruation returns in these athletes. Drinkwater et al. (1986) followed nine athletes over a 15·5 month period. Seven of the women had regained menses and two had remained amenorrhoeic. Lumbar BMD increased 6·3 % in the former amenorrhoeic women whilst decreasing a further 0·3 % in those who had remained amenorrhoeic. Small increases in BMD were also seen in the radius. These results are very similar to those of Lindberg et al. (1987) who retested seven amenorrhoeic runners at 15 months. Four had recovered menses and showed an improvement of 6·5 % in lumbar BMD. The other three remained amenorrhoeic and showed no improvement in BMD. In our laboratory we have followed 26 oligomenorrhoeic runners over an 18 month period. At one year those who had either regained menstruation or who were on hormone replacement therapy (\( n = 14 \)) had increased BMD by 4·1 % in the lumbar spine while in those who remained amenorrhoeic (\( n = 12 \)), BMD further declined by 1·7 %. Similar trends were seen in the proximal femur (J. H. Wilson, unpublished data).

These short term studies all suggest that small improvements in BMD may occur when menstruation returns. It is unknown whether such increases are maintained over several years. Recently we have compared 13 perimenopausal athletic women (mean age 42·9 years) with a history of menstrual irregularity with 37 similar athletic women (mean age 45·8 years) who had continual eumenorrhoea (Wilson et al. 1994a). Bone density was lower in the first group in both the proximal femur and the lumbar spine but this only reached significance in the spine (\( P < 0·05 \)). More importantly, BMD in the proximal femur and
lumbar spine of the previously amenorrhoeic women was not significantly different from the mean for European age matched women and in the neck of femur it was higher ($P < 0.02$). This suggests that prolonged menstrual irregularity does have long term effects on BMD but these may be offset by continued exercise.

**PREVENTION, ASSESSMENT AND MANAGEMENT OF ATHLETIC AMENORRHOEA**

The available evidence suggests that the onset of menstrual irregularity is associated with one or more of the factors in Fig. 1. Because they are so interlinked it has proved impossible to determine which are of greatest importance. However, alterations in eating habits such as food restriction and purging would seem to lie at the centre of the problem for many and better assessment and management of these behaviours may reduce the incidence. Prevention of eating disorders requires a number of approaches.

**EDUCATION**

Staff at all levels need to have greater awareness of the potential for eating disorders within their own sport. Coaches need advice on correct assessment of body composition and the optimum for performance for each athlete. Individualization of programmes for weight maintenance (and reduction if necessary) will reduce the likelihood that ill-advised comments will precipitate abnormal eating habits. The emphasis should move away from the very slender look in aesthetic sports although this also requires a change in general cultural attitudes. Finally, the athletes themselves must be educated. Unfortunately many athletes will continue to see role models, who are very thin, undertaking high levels of training and competition. This discourages them from weight gain or changes in training. In these cases, reduction of other risk factors and alterations in diet constituents may be sufficient for menstrual function to return (Myburgh et al. 1992).

**IDENTIFICATION OF ‘AT RISK’ ATHLETES**

A high index of suspicion may aid the detection of these problems. The preparticipation examination or routine medical for women and girls is an opportunity to screen for menstrual and nutritional disorders. Eating habit questionnaires specifically designed for athletes will aid identification of disturbed body image and eating attitudes. All sports specialists and primary care physicians should seek such information when female athletes present with problems such as stress fractures, recurrent injuries, weight loss or fatigue. Suggestions for assessment by primary care physicians, sports physicians or others involved in the care of athletes is given in Fig. 3.

**OPTIONS FOR TREATMENT**

Drinkwater et al. (1986) and Lindberg et al. (1987) demonstrated an increase in BMD associated with resumption of menses but in all cases this occurred owing to a reduction in training volume and intensity with a concomitant increase in weight. Not all athletes are willing to alter training habits in order to resume menstruation. In many, menstruation would be a nuisance and in some it might interfere with performance. In such athletes treatment to prevent further bone mineral loss or to improve low bone density may be an option.
Athlete with menstrual dysfunction

Pathological causes?

Yes

No

Appropriate referral

Assess risk factors

Nutrition
(sports nutritionist, parents)

Body composition
(exercise physiologist, coach)

Menstrual history
(GP)

Dietary analysis
Inadequate intake
Disordered eating
Vegetarian

Optimal weight and body fat (%)
Power:weight
Training volume/intensity

Menarche
Parity
Previous dysfunction

Clinical symptoms?

Yes

No

Weight too low?

Yes

No

Reassign targets

Reduce other risk factors:
Competition and training stresses
Social stresses

Referral
Sports Psychologist
Psychiatrist

Monitor:
food intake
weight
performance

Continuing menstrual dysfunction?

Yes

No

Specialist referral:
endocrinologist
gynaecologist
Bone density assessment
Treatment if necessary

Continue to monitor

Fig. 3. Flow diagram for assessment of athletes with menstrual dysfunction by clinicians. Management of athletes requires team approach and suggestions for personnel are given at relevant stages, if not already part of decision making. No stages are mutually exclusive.
Treatment regimes used in women with secondary amenorrhoea include calcium supplementation, hormone replacement therapy, and intranasal calcitonin. Readers are referred to Prior et al. (1992) for further discussion of the treatment of this condition which is beyond the scope of this paper. Management of the athlete with eating disorders requires a team approach with appropriate specialist referral.

CONCLUSION

Amenorrhoea in athletes is a common occurrence and is associated with low levels of sex hormones. The major consequence of this disorder is a reduction in bone density compared to eumenorrhoeic peers. In some, BMD may be so low that fractures occur and there may be a risk of early osteoporosis. However, long term studies on large numbers of amenorrhoeic athletes are required to determine the natural history of BMD in such women and the response to treatment. Multicentre studies and trials may be able to provide these answers.

Prevention of menstrual disturbance and careful management should it occur will help to prevent such dramatic consequences but, unfortunately, the aetiology of athletic amenorrhoea is complex. Abnormalities in eating are implicated but further large scale studies are required to ascertain the true incidence of these disorders and their relationship to menstrual dysfunction in athletes. All those involved in the care and support of young athletes should be aware of the prevalence of eating disorders and should place nutrition high on the agenda for optimum performance.

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