Journal of Sports Sciences, 2001, **19**, 915–929

The physical activity, fitness and health of children

COLIN BOREHAM[1]* and CHRIS RIDDOCH[2]

[1]*Department of Sport and Exercise Science, University of Ulster, Jordanstown BT37 0QB and* [2]*Department of Exercise and Health Science, University of Bristol, Bristol BS8 1TN, UK*

Accepted 7 July 2001

It is clear that, despite their natural tendencies, children have become less physically active in recent decades, with children today expending approximately 600 $kcal \cdot day^{-1}$ less than their counterparts 50 years ago. Although the health consequences of a reduced energy expenditure in adults is well documented, there is little direct evidence linking sedentariness with health in children. However, three main benefits arising from adequate childhood physical activity have been postulated. The first is direct improvements in childhood health status; evidence is accumulating that more active children generally display healthier cardiovascular profiles, are leaner and develop higher peak bone masses than their less active counterparts. Secondly, there is a biological carry-over effect into adulthood, whereby improved adult health status results from childhood physical activity. In particular, childhood obesity may be a precursor for a range of adverse health effects in adulthood, while higher bone masses in young people reduce the risk of osteoporosis in old age. Finally, there may be a behavioural carry-over into adulthood, whereby active children are more likely to become more active (healthy) adults. However, supporting evidence for this assertion is weak. Given this background, recent health guidelines suggesting that children should accumulate 60 min of moderate-intensity physical activity every day – supplemented by regular activities that promote strength flexibility and bone strength – appear to be justified. Future developments should include the implementation of large-scale, longitudinal studies spanning childhood and young adulthood, the further refinement of tools for measuring physical activity accurately in young people, and research into the relative strength of association between fitness – as well as activity – and health in children.

Keywords: exercise, obesity, osteoporosis, risk factors, young people.

Introduction

Observe any school playground full of 7-year-olds and you will see a study in perpetual motion. Children are invariably running, jumping, throwing and kicking in a spontaneous mfilée of physical activity, untutored and unstructured. Clearly, they are doing what comes naturally and they are enjoying themselves. Contrast these images with those of children sitting passively in the car for the 'school run', watching television for up to one-third of their waking day, participating in less and less school physical education as other curriculum pressures take precedence, and mounting evidence that even our youngest children are becoming more obese (Reilly *et al.*, 1999). Clearly, there are paradoxes in the lifestyles of children today that raise important questions. First, if our children enjoy physical activity, why are they apparently doing less of it? Part of the answer lies in the environment within which children now find themselves, an environment full of what Leonard Epstein and co-workers (1995) have called 'sedentary alternatives'. In the USA, children watch an average of 3.5–4.0 h of television per day. Cycling and walking to school have become unusual behaviours and playing in the street has been curtailed by safety concerns. Reports that children's energy intakes over the past 50 years have fallen by 600–700 $kcal \cdot day^{-1}$ (Durnin, 1992) appear to be well founded. An average child walking to and from school for a total of 45 min and playing actively after tea for 60 min would incur an additional daily energy expenditure of approximately 525 kcal (assuming an average exercise energy cost of 5 $kcal \cdot min^{-1}$ for a 50 kg child). Thus, many of the current health concerns relating to children's physical activity and their sequels may be seen as normal physiology within a pathological environment.

Although we instinctively feel that physical activity is

* Author to whom all correspondence should be addressed. e-mail: ca.boreham@ulst.ac.uk

beneficial to children, what is the scientific evidence to support this hypothesis? Surprisingly, and in contrast to adults, relatively little empirical research exists to help answer this question. Certainly, concern has been expressed about the rising prevalence of lifestyle-related chronic diseases in developed nations (DHSS, 1992) and the well-documented decline in physical activity among adults (Blair *et al.*, 1989b; DHSS, 1996) and children (Reiff *et al.*, 1986; Armstrong, 1989). However, the established causal links between health and habitual physical activity in adults are yet to be confirmed in children (Riddoch, 1998). Nevertheless, evidence is growing and, in an attempt to provide a conceptual framework for existing and future research, Blair *et al.* (1989a) have proposed a model for the health consequences of childhood physical activity. The hypothesized relationships within this model suggest that three main benefits arise from adequate childhood physical activity:

1. The direct improvement of childhood health status and quality of life.
2. The direct improvement of adult health status by, for example, delaying the onset of chronic disease in adulthood.
3. An increased likelihood of maintaining adequate activity into adulthood, thus indirectly enhancing adult health status.

Although it is true to say that the most convincing evidence links adult physical activity with adult health status, evidence is mounting to support all three relationships involving childhood physical activity. The aim of this review is to present some of the more compelling evidence in the area of fitness, activity and health as it relates to childhood, to highlight current weaknesses and omissions in the literature and, finally, to suggest implications for further research.

Activity and health in adults

To understand the relationships between activity and health in children, it is first necessary to examine the equivalent relationships in adults, where the consensus is more established. In fact, the evidence that activity is beneficial to health comes almost exclusively from studies with adults. The question that arises is: 'What are the implications of the adult data – which clearly demonstrate that physical activity promotes health – to children?'

In adults, both physical activity and physical fitness are inversely related to mortality (Paffenbarger *et al.*, 1986; Blair *et al.*, 1989b). A strong, independent, inverse relationship between activity or fitness and health has now been established (Powell *et al.*, 1987). Prospective population studies of adults have shown that higher fitness or physical activity leads to a reduced risk of coronary heart disease (Powell *et al.*, 1987; Berlin and Colditz, 1990), stroke (Wannamethee and Shaper, 1992), hypertension (Paffenbarger *et al.*, 1983), non-insulin-dependent diabetes mellitus (Helmrich *et al.*, 1991), osteoporotic fractures (Wickham *et al.*, 1989), depression (Stephens, 1988) and some cancers (Lee, 1994). Better fitness may also aid recovery from major surgery (S.K. Epstein *et al.*, 1995). Meta-analyses of data from more than 40 studies indicate that coronary heart disease is 1.9 times more likely to develop in physically inactive than active adults, independent of other risk factors (Powell *et al.*, 1987). This individual risk is comparable with the risk associated with the other main risk factors for coronary heart disease (hypertension, cigarette smoking and cholesterol). Blair *et al.* (1996) have reported similar results for low fitness, which carries a relative risk for all-cause mortality of 1.52 in men and 2.10 in women, whereas others (Blair *et al.*, 1995; Erikssen *et al.*, 1998) have shown that improving fitness in middle age may reduce all-cause mortality by more than 50%.

The public health burden of a sedentary lifestyle can be quantified by calculating the population attributable risk. This estimates the proportion of the public health burden caused by a specified risk factor, in this case a sedentary lifestyle. By calculating the population attributable risk for physical inactivity, we can estimate the proportion of mortality for chronic diseases such as coronary heart disease that would be avoided if everyone in a given population were sufficiently physically active (Van Mechelen, 1997a). To calculate population attributable risk, we need to know both the relative risk and the prevalence of the risk factor in a given population. Based on available information for these parameters, Powell and Blair (1994) estimated the population attributable risk of sedentary living to be 35%, 32% and 35% for coronary heart disease, cancer of the colon and diabetes mellitus mortality respectively. The scope for public health gains through decreased sedentariness is, therefore, considerable.

Dose–response relationships

A graded dose–response relationship appears to exist between activity and mortality, with mortality being greatest at the lower end of the activity distribution and lowest at the more active end (Paffenbarger *et al.*, 1986). Crucially, *increments* of risk reduction are greatest between activity groups at the low end of the activity distribution with a 'law of diminishing returns' as one moves along the distribution from low activity to high activity. There is also some evidence of a 'levelling off' of benefit at a certain point, which is suggestive of an *optimum* amount of activity, above which few further health benefits are gained. However, this optimum may vary – or not exist – depending on the health outcome selected. Similar relationships can be identified between fitness and morbidity, with the greatest difference in cardiovascular risk being observed between individuals in the least fit groups and those who are slightly fitter. Increments of benefit between these groups and subsequent groups are smaller (Blair *et al.*, 1989b).

At the upper end of the activity spectrum (characterized by high-volume training loads), some body systems can react adversely, even when the body has been conditioned gradually to these physical stresses. Musculoskeletal injuries, renal abnormalities, gastrointestinal disturbances, immune system suppression and menstrual irregularities may accompany high training loads. Although these conditions are mostly reversible with reduced training, they are undoubtedly troublesome and may partly offset any health benefits accruing from greater activity. They may also have an adverse effect on exercise adherence, reinforcing the likelihood of an optimal level of activity for health purposes.

What might this optimal amount of physical activity be? It is probable that, in evolutionary terms, we have developed biologically for the lifestyle of a hunter-gatherer, involving day-long intermittent activities of varying intensity. However, we are now forced by cultural, technological and environmental circumstances into a far more sedentary lifestyle. The current epidemic of sedentary living is likely to be a significant contributory factor to the increased prevalence of degenerative, lifestyle-related conditions, most notably coronary heart disease and stroke in the developed world (Boyd Eaton *et al.*, 1988).

Changing our predominantly sedentary society will not be easy. To inform and encourage people to espouse a more active lifestyle, health-related activity guidelines have been published for adults (US Department of Health and Human Services, 1996). Although still subject to debate (Barinaga, 1997), the central recommendation of these guidelines is the accumulation of 30 min of moderate-intensity activity ('moderate' equating to brisk walking for most people) at least 5 days a week. The evidence that health benefits may be *accumulated* from several relatively short bouts of activity (Hardman, 1999; Boreham *et al.*, 2000; Murphy *et al.*, 2000) may be particularly relevant to children, in whom patterns of activity are invariably intermittent rather than continuous (Bailey *et al.*, 1995).

These guidelines balance what is known to be beneficial to health and what is likely to be feasible for most people. It should be noted that most health benefits should accrue with regular moderate activity, but there is some evidence to suggest that more vigorous activity may be necessary for certain improvements to take place, most notably normalization of blood pressure. However, such high activity is *not* necessary to improve most dimensions of health (White *et al.*, 1993); the previously widely held view that running three times a week is in some way a health-related 'threshold' has now been largely abandoned.

We can conclude, therefore, that (a) avoiding the low end of the activity or fitness spectrum delivers the greatest relative health benefit and (b) high activity is probably unnecessary for the achievement of most health benefits. In this respect, examples of activities that have been shown to confer a significant reduction in risk for coronary heart disease include gardening (Leon *et al.*, 1987), lawn mowing (Leon *et al.*, 1987), walking (Paffenbarger *et al.*, 1986; Leon *et al.*, 1987) and stair climbing (Paffenbarger *et al.*, 1986). Although such activities may not be so attractive to children, these results do support the view that total activity energy expenditure, irrespective of the type, intensity, duration or frequency, may be a key dimension of activity for the improvement of health in a predominantly sedentary population. The important question is, however, does this adult data hold for children?

Activity and health in children

It is clear that young children enjoy active play. If sedentary alternatives are not available (L.H. Epstein *et al.*, 1995), most young children will play or invent active ways of passing time that involve physical activity. Older children might play more organized sports, either formally, in clubs and teams, or informally, in parks and playgrounds. Generally, these forms of play provide a large volume of activity incorporating a wide variety of movements and many muscle groups, thereby promoting cardiorespiratory development, muscular strength, muscular endurance, speed, power and flexibility. In the mid-teens, these relatively large amounts of activity decline, particularly in girls. If activity becomes very low – and we should remember that, in general, activity among adults is undoubtedly too low – then this may constitute a 'problem in the making'. The question is, therefore, 'does children's activity decline over the teenage years to such an extent that either their current or future health is compromised?' To answer this question, we must scrutinize the evidence relating activity to indicators of health status in children.

Unfortunately, for children, a consensus of opinion is yet to be reached on this question. Although there are indications of beneficial associations in some areas, relatively little evidence unambiguously relates childhood physical activity or fitness to childhood health, a more favourable childhood risk profile, or to later adult health (Blair and Meredith, 1994; Rowland, 1996; Riddoch, 1998). Despite this, there is a growing body of literature on children's activity, which is predicated on the conventional wisdom that activity is beneficial for children. We should, therefore, examine more closely the evidence relating to each of the three hypothesized relationships suggested by Fig. 1.

Activity and current health status

It has been reported that strong relationships between activity and various health parameters – bone health, blood pressure, lipid profile and obesity – are hard to find in children (Riddoch, 1998). A summary of relevant evidence follows.

Blood pressure and cholesterol. Armstrong and Simons-Morton (1994) reported that data suggesting a beneficial effect of activity on lipids and lipoproteins are minimal, although there is some evidence that high-density lipoprotein cholesterol (HDL-C) concentrations might be enhanced. Alpert and Wilmore (1994) concluded that aerobic training had only a weak relationship with blood pressure within the normal range, but that such training consistently reduced both systolic and diastolic blood pressure in hypertensive adolescents. Riddoch (1998) reviewed more recent studies and reported that, for lipids and lipoproteins, six studies (Al-Hazzaa *et al.*, 1994; de Visser *et al.*, 1994; Dwyer and Gibbons, 1994; Harrell *et al.*, 1996; Rowland *et al.*, 1996; Webber *et al.*, 1996) showed no association with measures of activity, whereas another six (Suter and Hawes, 1993; Bistritzer *et al.*, 1995; Craig *et al.*, 1996; Gutin *et al.*, 1996; Twisk *et al.*, 1996; Boreham *et al.*, 1997) showed a limited association. For blood pressure, one study (de Visser *et al.*, 1994) reported no association, while seven (Jenner *et al.*, 1992; Al-Hazzaa *et al.*, 1994; Anderson, 1994; Dwyer and Gibbons, 1994; Harrell *et al.*, 1996; Webber *et al.*, 1996; Boreham *et al.*, 1997) reported a beneficial association.

Overweight and obesity. Bar-Or and Baranowski (1994) concluded that both controlled trials and cross-sectional studies have indicated small but significant beneficial effects of activity for both non-obese and obese adolescents. However, no effect was observed in prospective observational studies. Some weak associations were evident, but no threshold 'dose' of activity could be identified. In a review of more recent evidence, Riddoch (1998) pointed out that only two studies (Durant *et al.*, 1994, 1996) observed no effect, whereas others (Durant *et al.*, 1993; Robinson *et al.*, 1993; Wolf *et al.*, 1993; Gutin *et al.*, 1995; Boreham *et al.*, 1997; Gutin and Owens, 1999) observed some effect. These results must, therefore, be treated as highly suggestive rather than definitive.

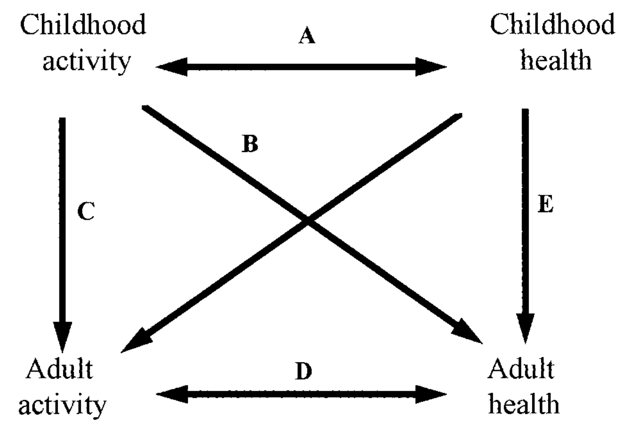

Fig. 1. Hypothetical relationships between physical activity and health in children and adults. Reproduced with permission from Blair *et al.* (1989a).

We can suggest four reasons why an increased prevalence of childhood obesity may be a major source of concern. First, obesity is a major risk factor for insulin resistance and diabetes, hypertension, dyslipidaemia, poor cardiorespiratory fitness and atherosclerosis (Schonfeld-Warden and Warden, 1997; Berenson *et al.*, 1998; Vanhala *et al.*, 1998; Boreham *et al.*, 2001). Secondly, obesity tends to track into adulthood (Clarke and Lauer, 1993). Thirdly, adults who were obese as children have increased morbidity and mortality, irrespective of adult weight (Must *et al.*, 1992; Gunnell *et al.*, 1998). Fourthly, overweight adolescents may suffer long-term social and economic discrimination (Gortmaker *et al.*, 1993). For these reasons, despite the lack of compelling evidence, childhood obesity should be a major target for intervention from both primary prevention and treatment perspectives; physical activity should feature strongly in this intervention. This requirement is made all the more pressing by recent reports of substantial secular increases in paediatric obesity (Campaigne *et al.*, 1994; Troiano *et al.*, 1995; Freedman *et al.*, 1997; Reilly *et al.*, 1999; Chinn and Rona, 2001), despite apparent reductions in daily energy intakes over the past 40–50 years (Durnin, 1992).

Bone health. Osteoporosis is a major public health burden, affecting more than 25 million people in the United States, mainly women. As osteoporosis and the fractures that are associated with it are largely a feature of old age, the scale of the problem will inevitably grow as the population ages. It is estimated, for example, that the 6.26 million hip fractures currently recorded annually on a worldwide basis will increase four-fold by the year 2050 (Cooper *et al.*, 1992). Although osteoporosis is principally a condition of the elderly, the optimal prevention strategy may be the attainment of a strong, dense skeleton during the growing years (Bailey *et al.*, 1996; Vuori, 1996).

Peak bone mass, which is achieved in most people by their late twenties (Theintz *et al.*, 1992; Lu *et al.*, 1994), appears to be largely under the control of genetic influences. Approximately 70–85% of the inter-individual variance in bone mass is genetically determined, with several candidate genes involved in the regulation and metabolism of collagen (COLIA 1 gene), vitamin D (VDR-gene), oestrogen (ER gene) and nitric oxide (ec NOS gene) (Ralston, 1997). However, the residual variance in bone mass is under environmental influences that are amenable to early intervention. The most important environmental factors appear to be body mass, diet – notably calcium intake (French *et al.*, 2000; Heaney *et al.*, 2000) – and the amount and type of physical activity performed throughout childhood and adolescence.

Investigations assessing the relationship between physical activity in childhood and bone mineral acquisition have been reviewed in detail by Bailey *et al.* (1996). Studies of representative populations have, in the main, been conducted retrospectively (Tylavsky *et al.*, 1992; Ruiz *et al.*, 1995; Teegarden *et al.*, 1996), although longitudinal and prospective studies have been reported recently (Slemenda *et al.*, 1994; Gunnes and Lehmann, 1996; Morris *et al.*, 1997). These studies have indicated that weight-bearing physical activity in childhood and adolescence is an important predictor of bone mineral density, while non-weight-bearing activity – such as swimming or cycling – is not (Grimston *et al.*, 1993). The size of the effect of physical activity (difference in bone mineral density between high and low fitness or activity groups) is, typically, between 5 and 15%. In his review, Vuori (1996) estimated that physical activity, which is feasible for many young people, increases peak bone mass by somewhat less than one standard deviation, or 7–8% approximately. However, more research on the optimal type and volume of physical activity required for bone health in young people is required. Based on available information (Lanyon, 1996), it is probable that activities that involve high strains, developed rapidly and distributed unevenly throughout the movement pattern, may be particularly osteogenic. Thus, activities such as aerobics, disco dancing, volleyball, basketball and racket sports may be effective and need not necessarily be of prolonged duration, as the osteogenic response to such movement appears to saturate after only a few loading cycles (Lanyon, 1996). It is also interesting to note that the natural play activities of young children, such as skipping, chasing and climbing, do provide a significant element of high-impact movement and may be optimal – in type – for health.

Further work also needs to be done to establish the optimal period within childhood in which to perform such activities to promote bone growth. While at one end of the spectrum adult bone appears to be relatively unresponsive to all but the most vigorous of exercise regimes (Friedlander *et al.*, 1995; Lohman *et al.*, 1995; Skerry, 1997), there is some evidence (Haapasalo, 1994; Morris *et al.*, 1997; Bradney *et al.*, 1998) that physical activity during the immediate pre-pubescent and pubescent years may be crucial for maximizing peak bone mass.

Physical activity, therefore, is an essential stimulus for bone structure and has the potential to increase peak bone mass in children and adolescents within the limits set by genetic, hormonal and nutritional influences. Such enhanced bone mass has considerable potential to reduce the risk of osteoporosis and associated fractures in later life, particularly if the increase can be maintained throughout adulthood by exercise.

Other risk factors. It may be of particular importance to note those studies (Saito *et al.*, 1992; Kahle *et al.*, 1996; Vanhala *et al.*, 1998) that have shown a beneficial effect of physical activity on parameters related to insulin metabolism. 'Metabolic syndrome' or 'syndrome X' (Reaven, 1988), which encompasses obesity, hypertension, hypertriglyceridaemia, depressed HDL-C and glucose intolerance or hyperinsulinaemia, is now a recognized clinical condition and it may be that this clustering of metabolic parameters – in both adults and children – will be an important focus for future physical activity research. Finally, several studies (Sallis *et al.*, 1988; Hansen *et al.*, 1991; Al-Hazzaa *et al.*, 1994; Boreham *et al.*, 1999b) have noted consistent relationships between habitual physical activity in children and aerobic fitness, the latter being a graded, independent risk factor for coronary heart disease in adults.

From the above, it is clear that more research is required into the relationships between physical activity and health in children before definitive conclusions can be drawn. However, the absence of evidence may not indicate evidence of absence. In other words, subtle relationships and effects may exist, but we may not currently be able to detect them. In particular, there is a lack of well-conducted, large-scale studies, especially longitudinal cohort studies, which may link childhood risk with clinical outcomes in adulthood.

Activity and future health status

It has been hypothesized that degenerative biological processes are initiated during infancy and childhood and that these processes will manifest themselves in chronic disease in later life. Indeed, there is evidence to suggest that adult health status may be determined, at least in part, by biological events that occur *in utero* (Barker, 1990). This 'lifecourse' model of adult chronic disease suggests that early biological events trigger a morphological or functional change that subsequently becomes a chronic condition. The individual is effectively 'programmed' for susceptibility to later disease through an early biological event. Crucially, the biological event may itself be triggered by an environmental influence (e.g. inadequate maternal or childhood nutrition, smoking) and it is in this respect that adequate physical activity may be important. These assertions remain largely hypothetical, although recent evidence (Van Lenthe *et al.*, 2001) indicates that certain health behaviours (e.g. physical inactivity, smoking and low fruit consumption) are more prevalent in disadvantaged adolescents, lending further credence to a 'lifecourse' approach to the understanding of socio-economic patterns of disease in adult populations. Nevertheless, sufficient evidence exists to indicate that childhood physical activity may influence adult disease outcomes in relation to obesity and osteoporosis.

It has been pointed out above that insufficient physical activity is a prime suspect in the aetiology of childhood obesity. The adverse health effects of childhood obesity may have further consequences for adult health and well-being. For example, Must *et al.* (1992) published the results of a large prospective study with 55 years of follow-up. Overweight in adolescence predicted a wide range of adverse health effects in adulthood that were independent of adult weight. Similar findings were published by Gunnell *et al.* (1998). It is interesting to note that risk factors for adult coronary artery disease, such as obesity, accelerate atherogenesis in the teenage years and their effects are amplified in young adulthood, some 20–30 years before coronary artery disease becomes clinically manifest (Berenson *et al.*, 1998; McGill and McMahon, 1998). Thus, being overweight as an adolescent may be a more significant predictor of future chronic disease than being overweight as an adult.

In relation to osteoporosis, the potential for physical activity of the right type to increase peak bone mass in children and adolescents was referred to in the previous section. The postulated 7–8% increase (Vuori, 1996) would be sufficient, if maintained into old age, to reduce substantially the risk of osteporotic fracture (Rubin *et al.*, 1993).

Activity as a habit

It is reasonable to assert that, if high activity in childhood increases the likelihood of being a more active adult – which we know enhances health – then childhood activity can be considered to have an indirect influence on adult health status. However, evidence to support this assertion is rather sparse. The persistence of a behaviour, or attribute, over time is called 'tracking' and refers to the maintenance of a rank order position over time compared to one's peers. Our main concern, therefore, might be whether inactivity in childhood leads to inactivity in adulthood and a subsequent elevated risk of adult disease. Conversely, does high activity in childhood predict high activity in adulthood?

Tracking through all stages of the lifespan has been comprehensively reviewed by Malina (1996). He concluded that activity tracks weakly to moderately during adolescence, from adolescence into adulthood and across various ages during adulthood. Although evidence for tracking of activity from childhood to adulthood is not strong, it could be that substantial tracking should not be expected in the case of physical activity. Many factors can influence this behaviour from day to day, between seasons of the year and between various stages of the lifecourse. Examples of major life events that can disturb activity patterns include changing schools, school-to-work transition, leaving home, moving house, moving to a new neighbourhood, biological and psychological development (especially puberty and adolescence), illness, marriage and child-rearing. Any one or a combination of these can significantly affect activity; therefore, it is to be expected that activity will be rather discontinuous in nature within any one individual over time. Of particular note is the role of family and the concept of 'leaving home'. Taylor *et al.* (1994) described three components of family influences on physical activity. The first, 'modelling', represents family members' current or past physical activity patterns as well as exercising with a family member. In a modelling process, 'the significant others' constitute available and powerful models. Such modelling may be apparent in the play activities of very young children (Sääkslahti *et al.*, 1999). The second component, 'social influence', involves encouragement provided by the family together with persuasion, pressure, expectations and sanctions. The third, 'social support', includes providing information about physical activity and providing material, transport and emotional support. Upon leaving home, such parental influences are lost and other influences predominate.

Linked with this, active individuals are likely to change the choice of activity they favour as they grow older, moving from play, through sport, to social and recreational activities. Finally, the 'background' or lifestyle activity we do – for example, walking to work and housework – confounds the overall picture. It is, therefore, not surprising that tracking coefficients are weak or moderate at best.

Prevalence of activity and inactivity

Cale and Almond (1992) reviewed 15 studies conducted on British children and reported that children seldom participate in sufficient activity to have a cardiovascular training effect or a health benefit. On the other hand, Sallis (1993) examined nine studies and concluded that the average child is sufficiently active to meet the adult recommendations for conditioning activities, with the exception of the average female in mid- to late adolescence. It has been argued that young children are highly and spontaneously active (Rowland, 1990; Åstrand, 1994). Blair (1992) has noted that children are generally fitter and more active than adults and that most of them are active enough to receive important health benefits from their activity.

Saris *et al.* (1990) reported daily physical activity values (total energy expenditure/resting metabolic rate) of 1.95 in 9-year-old boys and 1.71 in 8-year-old girls; Davies *et al.* (1991) reported values of 1.84 in 9-year-old boys and 1.65 in 9-year-old girls. Using energy intake as an indirect measure of activity, Boreham *et al.* (1993) reported energy intakes for 12- and 15-year-old British children equating to average daily physical activity values of 1.8–1.9. These results compare favourably with defined values of 1.7 (moderately active) and 1.9 (very active) (Department of Health, 1991).

As previously mentioned, however, there is some indirect evidence that activity may be falling, because children now have a lower daily calorie intake than previous generations (Durnin, 1992) and yet appear to be getting fatter in both the USA and the UK (Campaigne *et al.*, 1994; Chinn and Rona, 1994, 2001; Troiano *et al.*, 1995; Freedman *et al.*, 1997; Reilly *et al.*, 1999). The only logical explanation for this phenomenon is that activity has declined in children to a greater extent than the reported decline in energy intake. Compared with the compelling evidence suggesting an increase in children's fatness, that relating to energy intake is more suspect. However, we do have supporting evidence from adult studies (Prentice and Jebb, 1995).

From the above, it is clear that we are currently undecided about (a) how much activity children take, (b) whether children's activity is falling and (c) whether children's activity is sufficient to promote health. To exemplify this, Armstrong *et al.* (1991) reported an extremely low prevalence of activity that equates to the intensity, frequency and duration recommended for developing cardiopulmonary fitness. In their study, no girls and only 2% of boys achieved three 20 min sessions of sustained activity at a heart rate above 139 beats · min^{-1}. In contrast, Blair *et al.* (1989a) reported that 94% of boys and 88% of girls achieve an energy expenditure greater than 3 kcal · kg^{-1} · day^{-1}, which is known to be related to health in adults.

How is it that such diverse conclusions can be reached by experienced researchers on the basis of well-conducted studies? There are various explanations, including measurement error, different measurement methods, population differences, age-group differences and the measurement of different dimensions of physical activity. In addition, there is currently no universal consensus as to what criterion of physical activity should be adopted. For example, the criteria for developing cardiovascular fitness might be very different from those for health promotion.

To further demonstrate this important point, Welk (1994) reported that only 17% of children achieved a single activity session with a heart rate above 140 beats · min^{-1}. However, when the *same results* were re-analysed according to health-related criteria (i.e. achieving an activity energy expenditure of 4 kcal · kg^{-1} · day^{-1}), 99% of children complied. This demonstrates vividly how the selection of differing experimental criteria for a health-related threshold can lead to substantial variations in the estimates of how much activity children take part in and whether they are sufficiently active.

Guidelines for activity

As the result of two international consensus conferences (Sallis and Patrick, 1994; Biddle *et al.*, 1998), guidelines for health-related activity in children have recently been formulated. Despite the lack of unequivocal evidence suggesting that activity is related to health status in children (Boreham *et al.*, 1997) and that children are insufficiently active (Riddoch and Boreham, 1995), there are intuitive biological and behavioural arguments in favour of promoting physical activity for all children. Guidelines tend to reinforce the concept of a health-related threshold, but it is very difficult to ascertain the amount and type of physical activity during childhood that are appropriate for optimal health.

Early criteria, or thresholds, were generally based on the amount of activity required for the development of cardiovascular fitness. However, they may not only have been too stringent for most children to achieve, but also have been unrelated to the amount of activity needed to achieve a health benefit. Cale and Harris (1993) noted that, from a behavioural perspective, physical activity needs to be seen by children as an achievable and positive experience; adult fitness training guidelines, emphasizing continuous bouts of vigorous exercise, do not fulfil this. In this respect, recent guidelines (Sallis and Patrick, 1994; Biddle *et al.*, 1998) are based more upon the existing evidence of relationships between activity and health in children and the richer adult database. They also incorporate the behavioural issues of activity adoption and maintenance. The most recent guidelines (Biddle *et al.*, 1998) propose the accumulation of 60 min of moderate-intensity activity each day, including activities that promote strength, flexibility and bone health.

Physical activity and risks to the child

In addition to its benefits, physical activity carries inherent risks to both adults and children. Van Mechelen (1997b) has highlighted the potential for childhood injury when free play in various physical activities is replaced by competitive participation in just one or two sports. Whereas all activities carry an increased risk of traumatic (acute) injury, a strong focus on training for competition in a limited range of activities can result in the additional risk of overuse (chronic) injury. Whereas both types of injury normally heal without permanent disability, the costs must be considered – activity time lost, school time lost, predisposition to re-occurrence, the risk of permanent damage and the financial cost of treatment. Among elite child athletes, Baxter-Jones *et al.* (1993) reported an estimated 1 year incidence rate of 40 injuries per 100 children, equating to less than one injury per 1000 h of training. In these young athletes, about one-third of injuries were overuse injuries, which were, in turn, more severe than the traumatic injuries (20 *vs* 13 days lay-off respectively).

It should be emphasized that all sports and active recreational pursuits carry an increased risk of injury. In both adults and children, the risks and benefits must be carefully balanced. However, we should not forget the moral issue of when, or at what age or stage of development, a child is capable of making such important judgements. The roles and responsibilities of teachers, parents, sports governing bodies and coaches in this matter are considerable.

Fitness and health in children

Before examining relationships between children's fitness and health, it is worth defining and delimiting the two terms. Although several dimensions of physical fitness have been identified, the component that is most strongly associated with health is cardiovascular endurance, defined as 'the ability to sustain moderate intensity, whole-body activity for extended time periods' (Baranowski *et al.*, 1992). It can be measured objectively in the laboratory using a variety of ergometers (cycle, treadmill, etc.) with or without respiratory gas analysis, or more simply in the field by maximal running tests or submaximal cycle or step tests. Health is also multifactorial but, possibly owing to its pre-eminence as a cause of mortality in the developed world, coronary heart disease (CHD) and the risk factors predisposing to CHD are the most extensively studied in relation to physical fitness. Many studies of associations between fitness and health have been large-scale, cross-sectional population surveys of adults, using multivariate analysis to adjust for potential confounding variables. More powerful evidence for causal links between fitness and health come from less common longitudinal population studies, or from long-term training studies in which changes in the two variables can be compared over time. Irrespective of study design, one important distinction between adult and child studies is that the former have the advantage of examining associations between fitness and mortality, whereas children's studies in this field are restricted to examining risk factors for CHD rather than death arising from the disease. Finally, recent work on the genetics of fitness (Montgomery *et al.*, 1998) may improve our understanding of how fitness and health are predetermined and interrelated, particularly if aspects of fitness and health appear to share genetic influences.

Several adult population studies (Cooper *et al.*, 1976; Gibbons *et al.*, 1983; Blair *et al.*, 1989b; Van Saarse *et al.*, 1990; Sandvik *et al.*, 1993; Farrell *et al.*, 1998) have shown strong and consistent relationships between cardiovascular fitness and mortality from CHD and all-cause mortality, independent of potential confounding variables. Even more convincing is the evidence from prospective studies (Blair *et al.*, 1995) that have indicated that risk of mortality may be reduced substantially in middle-aged men who improve their fitness over several years.

The relationships for children appear to be less clear-cut, partly because the outcome measure – 'health' – cannot, for obvious reasons, be judged by mortality statistics. Rather, the investigator must rely upon risk factors for CHD mortality, such as high blood pressure, elevated blood lipids and fatness. However, such risk factors may only account for 50% of eventual coronary mortality and are, therefore, a relatively crude yardstick for coronary health (Thompson and Wilson, 1982). Furthermore, as a result of maturation, these biological risk factors are perturbed during adolescence and may or may not relate to adult values (Raitakari *et al.*, 1994). Finally, 'new' risk factors are being discovered on a regular basis, some of which may have a bearing on relationships between fitness, activity and health status. Despite these complications, some population studies have shown an independent relationship between cardiovascular fitness and risk factors for CHD in children (Tell and Vellar, 1988; Hofman and Walter, 1989). Further evidence of a causal relationship between fitness and coronary risk status in children comes from long-term fitness training studies that have reported concomitant improvements in individual risk factors (Eriksson and Koch, 1973; Hansen *et al.*, 1991).

One consistent finding in children's studies is the strong relationship between cardiovascular fitness and fatness (Gutin *et al.*, 1994; Hager *et al.*, 1995; Boreham *et al.*, 1997). It is thus not surprising that several studies have indicated that fatness is a major confounding variable in the relationship between fitness and other CHD risk factors. In at least five population studies (Fripp *et al.*, 1985; Sallis *et al.*, 1988; Hansen *et al.*, 1990; Bergstrom *et al.*, 1997; Boreham *et al.*, 2001), robust associations between cardiovascular fitness and risk disappeared once fatness was accounted for statistically; one further study (Tell and Vellar, 1988) reported much weaker relationships. It is worth noting that this feature has also been observed in a study of adults (Haddock *et al.*, 1998) investigating fitness and coronary risk factors; therefore, the confounding influence of body fatness on coronary risk does not appear to be confined to children.

Although a strong relationship has been shown to exist between cardiovascular fitness and CHD risk status in children, it appears to be largely mediated by fatness. Thus, any initiative to improve the health of children should ideally involve measures that simultaneously improve fitness and lower fatness, namely increased physical activity and dietary control.

Activity vs fitness for improved children's health?

It has been argued (Haskell *et al.*, 1985; Cureton, 1987; Seefeldt and Vogel, 1987) that physical training adaptations may not be directly related to, or necessary for, good health. The evidence linking both activity and fitness to health has been discussed, but the interesting question of whether physical activity or fitness is most strongly related to health status remains unresolved. For example, does an individual who has genetically high fitness but who is inactive achieve health benefits from the high fitness? Conversely, can the genetically low-fit individual gain health benefits through being active?

In adults, a reduction in the risk of coronary heart disease (CHD) is associated both with higher physical fitness (Peters *et al.*, 1983; Sobolski *et al.*, 1987; Blair, 1989b; Sandvik *et al.*, 1993) and physical activity (Paffenbarger *et al.*, 1986; Leon *et al.*, 1987; Powell *et al.*, 1987; Berlin and Colditz, 1990). Interestingly, the relationship appears to be stronger for fitness than for activity, but it is unclear whether this is a result of greater misclassification in activity measurement than in fitness measurement (Blair, 1994).

High cardiorespiratory fitness may be directly related to improved health status. The morphological and functional condition of the heart and circulatory system, as well as aspects of fuel metabolism, may lead directly to a reduced risk of, for example, CHD. This may in part be genetically based, in that a high-fit individual would automatically be blessed with better health status. Recent studies on the relationship between polymorphisms of the angiotensin-converting enzyme and cardiovascular fitness (Montgomery *et al.*, 1998) provide some support for this hypothesis. Also, some recent evidence (Boreham *et al.*, 1999a) has linked low birthweight, which may be partly genetic, with low cardiovascular fitness in adolescence. Low birthweight has previously been shown to be associated with a host of CHD risk factors in adults (Barker *et al.*, 1993; Martyn *et al.*, 1995; Phillips, 1996) and, more recently, with blood pressure in 3-year-olds (Whincup *et al.*, 1999). Thus, low fitness in children and later chronic disease may partly share common origins. An alternative, but not mutually exclusive, explanation might be that fitness acts as a marker for high activity. This activity might not only improve cardiovascular function, but might also promote other biochemical and haemodynamic changes (lower blood pressure, higher HDL cholesterol, lower triglycerides, improved glucose tolerance, modified clotting factors and post-prandial lipaemia), which are the underlying mechanisms for improved health. This 'spin-off' effect of activity might be termed 'metabolic fitness'. It is entirely possible that this type of fitness is the true health-related dimension of the generic term 'fitness'.

The jury is undoubtedly still out on all of this, but the implications are of crucial importance because, until we obtain a more fundamental understanding of the mechanisms and the effects of different types of activity upon those mechanisms, it is difficult to establish appropriate and effective activity messages.

Conclusions

Although the evidence for causal relationships between activity or fitness and health status in children is relatively weak, this is largely due to (1) a lack of large-scale longitudinal studies and (2) difficulties in measuring health, fitness and activity in children. This might be particularly true of the adolescent period, when naturally occurring shifts in blood pressure, lipids, activity patterns and adiposity can confound relationships. It is of particular note that no prospective study has linked, with any degree of certainty, health in the adult years with childhood activity patterns. However, it is intuitively logical that preventive measures – that is, the fostering of active lifestyles – should be lifelong objectives, commencing in early life (Sääkslahti *et al.*, 1999).

Implications

There are some important implications that can be drawn from this review. First, there is a need for more accurate measurement. Whereas fitness and physiological health status can be accurately quantified using laboratory procedures, this is not the case with physical activity Physical activity is a complex, multi-dimensional behaviour, which is extremely difficult to quantify using recall methods. The problems of measurement are greatly exacerbated when studying children. It is possible that meaningful results can be obtained from children using self-report methods in very large studies, but measurement error is likely to be unacceptably high. Such results could possibly be used to demonstrate the existence and direction of associations between physical activity and other outcomes but, to translate such findings into health policy, a higher order of results is required. Wareham and Rennie (1998) suggested that, to achieve greater accuracy, researchers need to clearly identify the dimensions of the physical activity to be measured and estimate the potential effect size or target difference more precisely. This will, in turn, enable cross-cultural comparisons to be made, temporal trends in activity to be identified and the sometimes subtle effects of interventions to be assessed. This cannot be achieved through gross self-report estimates of activity.

Secondly, the limited relationships between activity or fitness and health identified in this review are of the same importance as those reported for smoking and diet in children. It is clear that both children's smoking and eating habits are matters of considerable public health concern, even in the face of limited evidence. This may be largely because the strong adult relationships have been interpolated backwards through the lifespan and applied to children. We could argue that the same should be the case for fitness and physical activity. However, we require more information and more research should be devoted to identifying evidence for the early biological programming effects of physical activity and fitness.

Thirdly, there should be an increased focus on children's fitness. To date, physical activity has been the focus of health-related research in children. However, there appears to be a growing awareness that the role of cardiorespiratory fitness should not be underestimated. The evidence that fitness is related to health itself, without being mediated by physical activity, is becoming increasingly persuasive. To tease out the relative importance of activity and fitness, further studies accurately assessing both variables in children will be required.

In conclusion, given the strong and consistent relationships between activity or fitness and health in adults, it is probable that ensuring adequate activity and fitness in children will be of ultimate benefit. However, this judgement is based largely on limited paediatric data, a richer adult database, educated guesswork and basic physiological principles. This emerging paradigm must, in future, be supported by more substantial evidence.

Finally, it should not be forgotton that physical activity is our evolutionary heritage. We were 'designed', as a species, for physical activity, and yet we are now living in an environment in which the opportunities to be physically active are quickly disappearing. Only enlightened public policy regarding school curricula, school transportation, play opportunities and youth sports clubs can improve matters for tomorrow's adults.

References

Al-Hazzaa, H.M., Sulaiman, M.A., Al-Matar, A.J. and Al-Mobaireek, K.F. (1994). Cardiorespiratory fitness, physical activity patterns and coronary risk factors in preadolescent boys. *International Journal of Sports Medicine*, **15**, 267–272.

Alpert, B.S. and Wilmore, J.H. (1994). Physical activity and blood pressure in adolescents. *Pediatric Exercise Science*, **6**, 361–380.

Anderson, L.B. (1994). Blood pressure, physical fitness and physical activity in 17-year-old Danish adolescents. *Journal of Internal Medicine*, **236**, 323–330.

Armstrong, N. (1989). Children are fit but not active! *Education and Health*, **7**, 28–32.

Armstrong, N. and Simons-Morton, B. (1994). Physical activity and blood lipids in adolescents. *Pediatric Exercise Science*, **6**, 381–405.

Armstrong, N., Williams, J., Balding, J., Gentle, P. and Kirby, B. (1991). Cardiopulmonary fitness, physical activity patterns, and selected coronary risk factor variables in 11- to 16-year-olds. *Pediatric Exercise Science*, **3**, 219–228.

Åstrand, P.-O. (1994). Physical activity and fitness: evolutionary perspective and trends for the future. In *Physical Activity, Fitness, and Health: International Proceedings and Consensus Statement* (edited by C. Bouchard, R.J. Shephard and T. Stephens), pp. 98–105. Champaign, IL: Human Kinetics.

Bailey, D.A., Faulkner, R.A. and McKay, H.A. (1996). Growth, physical activity and bone mineral acquisition. *Exercise and Sports Science Reviews*, **24**, 233–266.

Bailey, R.C., Olson, J., Pepper, S.L., Porszasz, J., Barstow, T.J. and Cooper, D.M. (1995). The level and tempo of children's physical activities: an observational study. *Medicine and Science in Sports and Exercise*, **27**, 1033–1041.

Baranowski, T., Bouchard, C., Bar-Or, O., Bricker, T., Heath, G., Kimm, S.Y.S., Malina, R., Obarzanek, E., Pate, R., Strong, W.B., Truman, B. and Washington, R. (1992). Assessment, prevalence, and cardiovascular benefits of physical activity and fitness in youth. *Medicine and Science in Sports and Exercise*, **24**, S237–S247.

Barinaga, M. (1997). How much pain for cardiac gain? *Science*, **276**, 1324–1327.

Barker, D.J.P. (1990). The fetal and infant origins of adult disease. *British Medical Journal*, **301**, 1111.

Barker, D.J.P., Martyn, C.N., Osmond, C., Hales, C.N. and Fall, C.H.D. (1993). Growth *in utero* and serum cholesterol concentrations in adult life. *British Medical Journal*, **307**, 1524–1527.

Bar-Or, O. and Baranowski, T. (1994). Physical activity, adiposity and obesity among adolescents. *Pediatric Exercise Science*, **6**, 348–360.

Baxter-Jones, A., Maffulli, N. and Helms, P. (1993). Low injury rates in elite athletes. *Archives of Disease in Childhood*, **68**, 130–132.

Berenson, G.S., Srinivasan, S.R., Bao, W., Newman, W.P., Tracy, R.E. and Wattingey, W.A. (1998). Association between multiple cardiovascular risk factors and atherosclerosis in children and young adults. *New England Journal of Medicine*, **338**, 1650–1656.

Bergstrom, E., Hernall, O. and Persson, L.A. (1997). Endurance running performance in relation to cardiovascular risk indicators in adolescents. *International Journal of Sports Medicine*, **18**, 300–307.

Berlin, J.A. and Colditz, A. (1990). A meta-analysis of physical activity in the prevention of coronary heart disease. *American Journal of Epidemiology*, **132**, 612–627.

Biddle, S., Cavill, N. and Sallis, J. (1998). Policy framework for young people and health-enhancing physical activity. In *Young and Active? Young People and Health-enhancing Physical Activity: Evidence and Implications* (edited by S. Biddle, J. Sallis and N. Cavill), pp. 3–16. London: Health Education Authority.

Bistritzer, T., Rosenzweig, L., Barr, J., Mayer, S., Lahat, E., Faibel, H., Schlesinger, Z. and Aladjem, M. (1995). Lipid profile with paternal history of coronary heart disease before age 40. *Archives of Disease in Childhood*, **73**, 62–65.

Blair, S.N. (1992). Are American children and youth fit? The need for better data. *Research Quarterly for Exercise and Sport*, **63**, 120–123.

Blair, S.N. (1994). Physical activity, fitness and coronary heart disease. In *Physical Activity, Fitness and Health: International Proceedings and Consensus Statement* (edited by C. Bouchard, R.J. Shephard and T. Stephens), pp. 579–590. Champaign, IL: Human Kinetics.

Blair, S.N. and Meredith, M.D. (1994). The exercise–health relationship: does it apply to children and youth? In *Health and Fitness through Physical Education* (edited by R.R. Pate and R.C. Hohn), pp. 11–19. Champaign, IL: Human Kinetics.

Blair, S.N., Clark, D.G., Cureton, K.J. and Powell, K.E. (1989a). Exercise and fitness in childhood: implications for a lifetime of health. In *Perspectives in Exercise Science and Sports Medicine, Vol. 2: Youth, Exercise and Sport* (edited by C.V. Gisolfi and D.R. Lamb), pp. 401–430. New York: McGraw-Hill.

Blair, S.N., Kohl, H.W., Paffenbarger, R.S.J., Clark, D.G., Cooper, K.H. and Gibbons, L.W. (1989b). Physical fitness and all-cause mortality: a prospective study of healthy men and women. *Journal of the American Medical Association*, **262**, 2395–2401.

Blair, S.N., Kohl III, H.W., Barlow, C.E., Paffenbarger, R.S., Gibbons, L.W. and Maccra, C.A. (1995). Changes in physical fitness and all-cause mortality: a prospective study of healthy and unhealthy men. *Journal of the American Medical Association*, **273**, 1093–1098.

Blair, S.N., Kampert, J.B., Kohl III, H.W., Barlow, C.E., Macera, C.A., Paffenbarger, R.S. and Gibbons, L.W. (1996). Influences of cardiorespiratory fitness and other precursors on cardiovascular disease and all-cause mortality in men and women. *Journal of the American Medical Association*, **276**, 205–210.

Boreham, C., Savage, M., Primrose, D., Cran, G. and Strain, J. (1993). Coronory risk factors in schoolchildren. *Archives of Disease in Childhood*, **68**, 182–186.

Boreham, C.A., Twisk, J., Savage, M.J., Cran, G.W. and Strain, J.J. (1997). Physical activity, sports participation, and risk factors in adolescents. *Medicine and Science in Sports and Exercise*, **29**, 788–793.

Boreham, C., Murray, L., Dedman, D., Gray, A., Savage, M., Strain, J.J. and Davey-Smith, G. (1999a). Relationships between birthweight and physical fitness of adolescents: the Northern Ireland Young Hearts Project. *Pediatric Exercise Science*, **11**, 277.

Boreham, C., Twisk, J., Van Mechelen, W., Savage, M., Strain, J. and Cran, G. (1999b). Relationships between the development of biological risk factors for coronary heart disease and lifestyle parameters during adolescence: the Northern Ireland Young Hearts Project. *Public Health*, **113**, 7–12.

Boreham, C.A.G., Wallace, W.F.M. and Nevill, A.M. (2000). Training effects of accumulated daily stairclimbing exercise in previously sedentary young women. *Preventive Medicine*, **30**, 277–281.

Boreham, C., Twisk, J., Murray, L., Savage, M., Strain, J.J. and Cran, G. (2001). Fitness, fatness, and coronary heart disease risk in adolescents: the Northern Ireland Young Hearts Project. *Medicine and Science in Sports and Exercise*, **33**, 270–274.

Boyd Eaton, S., Konner, M. and Shostak, M. (1988). Stone Agers in the fast lane: chronic degeneration diseases in evolutionary perspective. *American Journal of Medicine*, **84**, 739–749.

Bradney, M., Pearce, G., Naughton, G., Sullivan, C., Bass, S. and Besk, T. (1998). Moderate exercise during growth in prepubertal boys: changes in bone mass, size, volumetric density and bone strength. A controlled prospective study. *Journal of Bone Mineral Research*, **13**, 1814–1821.

Cale, L. and Almond, L. (1992). Children's activity levels: a review of studies conducted on British children. *Physical Education Review*, **15**, 111–118.

Cale, L. and Harris, J. (1993). Exercise recommendations for children and young people. *Physical Education Review*, **16**, 89–98.

Campaigne, B.N., Morrison, J.A., Schumann, B.C., Falkner, F., Lakatos, E., Sprecher, D. and Schreiber, G.B. (1994). Indexes of obesity and comparisons with previous national survey data in 9- and 10-year-old black and white girls: the National Heart, Lung, and Blood Institute Growth and Health Study. *Journal of Pediatrics*, **124**, 675–680.

Chinn, S. and Rona, R.J. (1994). Trends in weight-for-height and triceps skinfold thickness for English and Scottish children, 1972–82 and 1982–90. *Paediatric and Perinatal Epidemiology*, **8**, 90–106.

Chinn, S. and Rona, R. (2001). Prevalence and trends in overweight and obesity in three cross-sectional studies of British children, 1974–94. *British Medical Journal*, **322**, 24–26.

Clarke, W.R. and Lauer, R.M. (1993). Does childhood obesity track into adulthood? *Critical Reviews in Food Science and Nutrition*, **33**, 423–430.

Cooper, C., Campion, G. and Melton, L.J. (1992). Hip fractures in the elderly: a world-wide projection. *Osteoporosis International*, **2**, 285–289.

Cooper, K.H., Pollock, M.L., Martin, R.P., White, S.R., Linnerrud, A.C. and Jackson, A. (1976). Physical fitness levels *vs* selected coronary risk factors. *Journal of the American Medical Association*, **236**, 166–169.

Craig, S.B., Bandini, L.G., Lichtenstein, A.H., Schaefer, E.J. and Dietz, W.H. (1996). The impact of physical activity on lipids, lipoproteins and blood pressure in preadolescent girls. *Pediatrics*, **98**, 389–395.

Cureton, K.J. (1987). Commentary on 'children and fitness: a public health perspective'. *Research Quarterly for Exercise and Sport*, **58**, 315–320.

Davies, P.S.W., Day, J.M.E. and Lucas, A. (1991). Early energy expenditure and later body fatness. *International Journal of Obesity*, **15**, 727–731.

Department of Health (1991). *Dietary Reference Values for Food Energy and Nutrition for the United Kingdom*. Report on Health and Social Subjects No. 41. London: HMSO.

Department of Health and Social Services (1992). *The Health of the Nation*. London: HMSO.

Department of Health and Social Services (1996). *Strategy Statement on Physical Activity*. London: Department of Health.

de Visser, D.C., Van Hooft, I.M., Van Doornen, L.J., Hofman, A., Orlebeke, J.F. and Grobbee, D.E. (1994). Anthropometric measures, fitness and habitual physical activity in offspring of hypertensive parents: Dutch hypertension and offspring study. *American Journal of Hypertension*, **7**, 242–248.

Durant, R.H., Baranowski, T., Rhodes, T., Gutin, B., Thompson, W.O., Carroll, R., Puhl, J. and Greaves, K.A. (1993). Association among serum lipid and lipoprotein concentrations and physical activity: physical fitness and body composition in young children. *Journal of Pediatrics*, **123**, 185–192.

Durant, R.H., Baranowski, T., Johnson, M. and Thompson, W.O. (1994). The relationship among television watching and body composition of young children. *Pediatrics*, **94**, 449–455.

Durant, R.H., Thompson, W.O., Johnson, M. and Baranowski, T. (1996). The relationship among television watching, physical activity, and body composition of 5- or 6-year old children. *Pediatric Exercise Science*, **8**, 15–26.

Durnin, J.V.G.A. (1992). Physical activity levels – past and present. In *Physical Activity and Health: Symposium of the Society for the Study of Human Biology* (edited by N.G. Norgan), pp. 20–27. Cambridge: Cambridge University Press.

Dwyer, T. and Gibbons, L.E. (1994). The Australian Schools Health and Fitness Survey: physical fitness related to blood pressure but not lipoproteins. *Circulation*, **89**, 1539–1544.

Epstein, L.H., Valoski, A.M., Vara, L.S., McCurley, J., Wisniewski, L., Kalarchian, M.A., Kelin, K.R. and Shrager, L.R. (1995). Effects of decreasing sedentary behaviour and increasing activity on weight change in obese children. *Health Psychology*, **14**, 109–115.

Epstein, S.K., Faling, L.J., Daly, B.D. and Celli, B.R. (1995). Inability to perform bicycle ergometry predicts increased morbidity and mortality after lung resection. *Chest*, **107**, 311–316.

Erikssen, G., Liestol, K., Bjornholt, J. and Thaulow, E. (1998). Changes in physical fitness and changes in mortality. *Lancet*, **352**, 759–762.

Eriksson, B.O. and Koch, G. (1973). Effects of physical training on hemodynamic response during submaximal and maximal exercise in 11–13 year old boys. *Acta Physiologica Scandinavica*, **87**, 27–39.

Farrell, S.W., Kampert, J.B., Kohl III, H.W., Barlow, C.E., Macera, C.A., Paffenbarger, R.S., Gibbons, L.W. and Blair, S.N. (1998). Influences of cardiorespiratory fitness levels and other predictors on cardiovascular disease mortality in men. *Medicine and Science in Sports and Exercise*, **30**, 899–905.

Freedman, D.S., Srinivasan, S.R., Valdez, R.A., Williamson, D.F. and Berenson, G.S. (1997). Secular increases in relative weight and adiposity among children over two decades: the Bogalusa Heart Study. *Pediatrics*, **99**, 420–426.

French, S.A., Fulkerson, J.A. and Story, M. (2000). Increasing weight-bearing physical activity and calcium intake for bone mass growth in children and adolescents: a review of intervention trials. *Preventive Medicine*, **31**, 722–731.

Friedlander, A.L., Genant, H.K., Sadowsky, S., Byl, N.N. and Gluer, C.C. (1995). A two-year program of aerobics and weight training enhances bone mineral density of young women. *Journal of Bone and Mineral Research*, **10**, 574–585.

Fripp, R.R., Hodgson, J.L., Kwiterovich, P.O., Werner, J.C., Schuler, H.G. and Whitman, V. (1985). Aerobic capacity, obesity and atherosclerotic risk factors in male adolescents. *Pediatrics*, **75**, 813–818.

Gibbons, L.W., Blair, S.N., Cooper, K.H. and Smith, M. (1983). Association between coronary heart disease factors and physical fitness in healthy adult women. *Circulation*, **67**, 977–983.

Gortmaker, S.L., Must, A., Perrin, J.M., Sobol, A.M. and Dietz, W.H. (1993). Social and economic consequences of overweight in adolescence and young adulthood. *New England Journal of Medicine*, **329**, 1008–1012.

Grimston, S.K., Willows, N.D. and Hanley, D.A. (1993). Mechanical loading regime and its relationship to bone mineral density in children. *Medicine and Science in Sports and Exercise*, **25**, 1203–1210.

Gunnell, D.J., Frankel, S.J., Nanchahal, K., Peters, T.J. and Davey-Smith, G. (1998). Childhood obesity and adult cardiovascular mortality: a 57-y follow-up study based on the Boyd-Orr cohort. *American Journal of Clinical Nutrition*, **67**, 1111–1118.

Gunnes, M. and Lehmann, E.H. (1996). Physical activity and dietary constituents as predictors of forearm cortical and trabecular bone gain in healthy children and adolescents: a prospective study. *Acta Paediatrica*, **85**, 19–25.

Gutin, B. and Owens, S. (1999). Role of exercise intervention in improving body fat distribution and risk profile in children. *American Journal of Human Biology*, **11**, 237–247.

Gutin, B., Islam, S., Manos, T., Cucuzzo, N., Smith, C. and Stachura, M.E. (1994). Relation of percentage of body fat and maximal aerobic capacity to risk factors for atherosclerosis and diabetes in black and white seven to eleven year-old children. *Journal of Pediatrics*, **125**, 847–852.

Gutin, B., Cucuzzo, N., Islam, S., Smith, C., Moffatt, R. and Pargman, D. (1995). Physical training improves body composition of black obese 7- to 11-year-old girls. *Obesity Research*, **3**, 305–312.

Gutin, B., Cucuzzo, N., Islam, S., Smith, C. and Stachura, M.E. (1996). Physical training, lifestyle education and coronary risk factors in obese girls. *Medicine and Science in Sports and Exercise*, **28**, 19–23.

Haapasalo, H., Kannus, P., Sievanen, H., Heinonen, A., Oja, P. and Vuori, I. (1994). Long-term unilateral loading and bone mineral density and content in female squash players. *Calcified Tissue International*, **54**, 249–255.

Haddock, B.L., Hopp, H.P. and Mason, J.J. (1998). Cardiorespiratory fitness and cardiovascular disease risk factors in postmenopausal women. *Medicine and Science in Sports and Exercise*, **30**, 893–898.

Hager, R.L., Tucker, L.A. and Seljaas, G.T. (1995). Aerobic fitness, blood lipids and body fat in children. *American Journal of Public Health*, **85**, 1702–1706.

Hansen, H.S., Hyldebrandt, N., Froberg, K. and Nielsen, J.R. (1990). Blood pressure and physical fitness in a population of children: the Odense Schoolchild Study. *Journal of Human Hypertension*, **4**, 615–620.

Hansen, H.S., Froberg, K., Hyldebrandt, N. and Nielson, J.K. (1991). A controlled study of eight months of physical training and reduction of blood pressure in children: the Odense Schoolchild Study. *British Medical Journal*, **303**, 682–685.

Hardman, A. (1999). Accumulation of physical activity for health gains: what is the evidence? *British Journal of Sports Medicine*, **33**, 87–92.

Harrell, J.S., McMurray, R.G., Bangdiwala, S.I., Frauman, A.C., Gansky, S.A. and Bradley, C.B. (1996). Effects of a school-based intervention to reduce cardiovascular disease risk factors in elementary-school children: the Cardiovascular Health in Children (CHIC) Study. *Journal of Pediatrics*, **128**, 797–805.

Haskell, W.L., Montoye, H.J. and Orenstein, D. (1985). Physical activity and exercise to achieve health-related physical fitness components. *Public Health Reports*, March–April, pp. 202–213.

Heaney, R.P., Abrams, S., Dawson-Hughes, B., Looker, A., Marcus, R., Matkovic, V. and Weaver, C. (2000). Peak bone mass. *Osteoporosis International*, **11**, 985–1009.

Helmrich, S.P., Ragland, D.R., Leung, R.W. and Paffenbarger, R.S. (1991). Physical activity and reduced occurence of non-insulin dependent diabetes mellitus. *New England Journal of Medicine*, **325**, 147–152.

Hofman, A. and Walter, H.J. (1989). The association between physical fitness and cardiovascular disease risk factors in children in a five-year follow-up study. *International Journal of Epidemiology*, **18**, 830–835.

Jenner, D.A., Vandongen, R. and Beilin, L.J. (1992). Relationships between blood pressure and measures of dietary energy intake, physical fitness, and physical activity in Australian children aged 11–12 years. *Journal of Epidemiology and Community Health*, **46**, 108–113.

Kahle, E.B., Zipf, W.B., Lamb, D.R., Horswill, C.A. and Ward, K.M. (1996). Association between mild, routine exercise and improved insulin dynamics and glucose control in obese adolescents. *International Journal of Sports Medicine*, **17**, 1–6.

Lanyon, L.E. (1996). Using functional loading to influence bone mass and architecture: objectives, mechanisms and relationship with estrogen of the mechanically adaptive process in bone. *Bone Mineral*, **18**, 37S–43S.

Lee, I.-M. (1994). Physical activity, fitness and cancer. In *Physical Activity, Fitness and Health: International Proceedings and Consensus Statement* (edited by C. Bouchard, R.J. Shephard and T. Stephens), pp. 814–831. Champaign, IL: Human Kinetics.

Leon, A.S., Connett, J., Jacobs, D.R.J. and Raurama, R. (1987). Leisure-time physical activity levels and risk of coronary heart disease and death: Multiple Risk Factor Intervention Trial. *Journal of the American Medical Association*, **258**, 2388–2395.

Lohman, T., Going, S., Pamenter, R., Hall, M., Boyden, T., Houtkooper, L., Ritenbaugh, C., Bare, L., Hill, A. and Aickin, M. (1995). Effects of resistance training on regional and total bone mineral density in premenopausal women: a randomized prospective study. *Journal of Bone and Mineral Research*, **10**, 1015–1024.

Lu, P.W., Brody, J.N. and Ogle, G.D. (1994). Bone mineral density of total body, spine and femoral neck in children and young adults: a cross-sectional and longitudinal study. *Journal of Bone and Mineral Research*, **9**, 1451–1458.

Malina, R.M. (1996). Tracking of physical activity and physical fitness across the lifespan. *Research Quarterly for Exercise and Sport*, **67**(suppl.), S1–S10.

Martyn, C.N., Barker, D.J.P., Jespersen, S., Greenwald, S., Osmond, C. and Berry, C. (1995). Growth *in utero*, adult blood pressure and arterial compliance. *British Heart Journal*, **73**, 116–121.

McGill, H.C. and McMahon, C.A. (1998). Determinants of atherosclerosis in the young: Pathobiological Determinants of Atherosclerosis in Youth (PDAY) Research Group. *American Journal of Cardiology*, **82**, 30T–36T.

Montgomery, H.E., Marshall, R., Hemingway, H., Myerson, S., Clarkson, P., Dollery, C., Hatward, M., Holliman, D.E., Jubb, M., World, M., Thomas, E.L., Brynes, A.E., Saeed, N., Barnard, M., Bell, J.D., Prasad, K., Rayson, M., Talmud, P.J. and Humphries, S.E. (1998). Human gene for physical performance. *Nature*, **393**, 221–222.

Morris, F., Naughton, G.A., Gibbs, J.L., Carlson, J. and Wark, J.G. (1997). Prospective ten-month exercise intervention in premenarcheal girls: positive effects on bone and lean mass. *Journal of Bone and Mineral Research*, **12**, 1453–1462.

Murphy, M.H., Nevill, A.M. and Hardman, A.E. (2000). Different patterns of brisk walking are equally effective in decreasing post-prandial lipaemia. *International Journal of Obesity*, **24**, 1303–1309.

Must, A., Jacques, P.F., Dallal, G.E., Bajema, C.J. and Dietz, W.H. (1992). Long-term morbidity and mortality of overweight adolescents. *New England Journal of Medicine*, **327**, 1350–1355.

Paffenbarger, R.S., Wing, A.L., Hyde, R.T. and Jung, D. (1983). Chronic disease in former college students: physical activity and incidence of hypertension of college alumni. *American Journal of Epidemiology*, **117**, 245–257.

Paffenbarger, R.S., Hyde, R.T., Wing, A.L. and Hsieh, C. (1986). Physical activity, all-cause mortality, and longevity of college alumni. *New England Journal of Medicine*, **314**, 605–613.

Peters, R.K., Cady, L.D.J., Bischoff, D.P., Bernstein, L. and Pike, M.C. (1983). Physical fitness and subsequent myocardial infarction in healthy workers. *Journal of the American Medical Association*, **249**, 3052–3056.

Phillips, D.I.W. (1996). Insulin resistance as a programmed response to fetal undernutrition. *Diabetologia*, **39**, 1119–1122.

Powell, K.E. and Blair, S.N. (1994). The public health burden of sedentary living habits: theoretical but realistic estimates. *Medicine and Science in Sports and Exercise*, **26**, 851–856.

Powell, K.E., Thompson, P.D., Caspersen, C.J. and Kendrick, K.S. (1987). Physical activity and the incidence of coronary heart disease. *Annual Review of Public Health*, **8**, 281–287.

Prentice, A.M. and Jebb, S.A. (1995). Obesity in Britain: gluttony or slot? *British Medical Journal*, **311**, 437–439.

Raitakari, O.T., Porkka, K.V.K., Rasenen, L., Ronnemaa, T. and Viikari, J.S.A. (1994). Clustering and six-year cluster-tracking of serum total cholesterol, HDL-cholesterol and diastolic blood pressure in children and young adults. *Journal of Clinical Epidemiology*, **47**, 1085–1093.

Ralston, S.H. (1997). Osteoporosis. *British Medical Journal*, **315**, 469–472.

Reaven, G.M. (1988). Role of insulin resistance in human disease. *Diabetes*, **37**, 1595–1607.

Reiff, G.G., Dixon, W.R., Jacoby, D., Ye, G.X., Spain, G.G. and Hunsicker, P.A. (eds) (1986). *The President's Council on Physical Fitness and Sports National School Population Fitness Survey*. Ann Arbor, MI: University of Michigan Press.

Reilly, J.J., Dorosty, A.R. and Emmett, P.M. (1999). Prevalence of overweight and obesity in British children: cohort study. *British Medical Journal*, **319**, 1039.

Riddoch, C.J. (1998). Relationships between physical activity and physical health in young people. In *Young and Active? Young People and Health-enhancing Physical Activity: Evidence and Implications* (edited by S. Biddle, J. Sallis and N. Cavill), pp. 17–48. London: Health Education Authority.

Riddoch, C.J. and Boreham, C.A. (1995). The health-related physical activity of children. *Sports Medicine*, **19**, 86–102.

Robinson, T.N., Hammer, L.D., Killen, J.D., Kraemer, H.C., Wilson, D.M. and Hayward, C. (1993). Does television viewing increase obesity and reduce physical activity? Cross-sectional and longitudinal analyses among adolescent girls. *Pediatrics*, **91**, 273–280.

Rowland, T.W. (ed.) (1990). *Exercise and Children's Health*. Champaign, IL: Human Kinetics.

Rowland, T. (1996). Is there a scientific rationale supporting the value of exercise for the present and future cardiovascular health of children? The con argument. *Pediatric Exercise Science*, **8**, 303–309.

Rowland, T.W., Martel, L., Vanderburgh, P., Manos, T. and Charkoudian, N. (1996). The influence of short-term aerobic training on blood lipids in healthy 10–12 year old children. *International Journal of Sports Medicine*, **17**, 487–492.

Rubin, K., Schirduan, V., Gendreau, P., Sarfarazi, M., Mendola, R. and Dalsky, G. (1993). Predictors of axial and peripheral bone mineral density in healthy children and adolescents, with special attention to the role of puberty. *Journal of Pediatrics*, **123**, 863–870.

Ruiz, J.C., Mandel, C. and Garabedian, M. (1995). Influence of spontaneous calcium intake and physical exercise on the vertebral and femoral bone mineral density of children and adolescents. *Journal of Bone and Mineral Research*, **10**, 675–682.

Sääkslahti, A.P., Numminen, P., Niinikoski, H., Rask-Nissilä, L., Viikari, J., Tuominen, J. and Välimäki, I. (1999). Is physical activity related to body size, fundamental motor skills, and CHD risk factors in early childhood? *Pediatric Exercise Science*, **11**, 327–340.

Saito, I., Nishino, M., Kawabe, H., Wainai, H., Hasegawa, C., Saruta, T., Nagano, S. and Sekihara, T. (1992). Leisure time physical activity and insulin resistance in young obese students with hypertension. *American Journal of Hypertension*, **5**, 915–918.

Sallis, J.F. (1993). Epidemiology of physical activity and fitness in children and adolescents. *Critical Reviews in Food Science and Nutrition*, **33**, 403–408.

Sallis, J.F. and Patrick, K. (1994). Physical activity guidelines for adolescents: consensus statement. *Pediatric Exercise Science*, **6**, 302–314.

Sallis, J.F., Patterson, T.L., Buono, M.J. and Nadder, P.R. (1988). Relation of cardiovascular fitness and physical activity to cardiovascular disease risk factors in children and adults. *American Journal of Epidemiology*, **127**, 933–941.

Sandvik, L., Erikssen, J., Thaulow, E., Erikssen, G., Mundal, R. and Rodahl, K. (1993). Physical fitness as a predictor of mortality among healthy, middle-aged Norwegian men. *New England Journal of Medicine*, **328**, 533–537.

Saris, W.H.M., Emons, H.J.G., Groenenboom, D.C. and Westerterp, K.R. (1990). Discrepancy between FAO/WHO energy requirements and actual energy expenditure levels in healthy 7–11 year old children. In *Children and Exercise* (edited by G. Beunen, J. Ghesquiere, T. Reybrouck and A.L. Claessens), p. 115. Stuttgart: Enke.

Schonfeld-Warden, N. and Warden, C.H. (1997). Pediatric obesity: an overview of etiology and treatment. *Pediatric Clinics of North America*, **44**, 339–361.

Seefeldt, V. and Vogel, P. (1987). Children and fitness: a public health perspective. *Research Quarterly*, **58**, 331–333.

Skerry, T.M. (1997). Mechanical loading and bone: what sort of exercise is beneficial to the skeleton? *Bone Mineral*, **20**, 179–181.

Slemenda, C.W., Reister, T.K., Hui, S.L., Miller, J.Z., Christian, J.C. and Johnston, C.C. (1994). Influences on skeletal mineralization in children and adolescents: evidence for varying effects of sexual maturation and physical activity. *Journal of Pediatrics*, **125**, 201–207.

Sobolski, J., Kornitzer, M., De Backer, G., Dramaix, M., Abramowicz, M., Gegre, S. and Denolin, H. (1987). Protection against ischemic heart disease in the Belgian fitness study: physical fitness rather than physical activity? *American Journal of Epidemiology*, **125**, 601–610.

Stephens, T. (1988). Physical activity and mental health in the United States and Canada: evidence from four population surveys. *Preventive Medicine*, **17**, 35–47.

Suter, E. and Hawes, M.R. (1993). Relationship of physical activity, body fat, diet and blood lipid profile in youths 10–15 years. *Medicine and Science in Sports and Exercise*, **25**, 748–754.

Taylor, W.C., Baranowski, T. and Sallis, J.F. (1994). Family determinants of childhood physical activity: a social-cognitive model. In *Advances in Exercise Adherence* (edited by R.K. Dishman), pp. 319–342. Champaign, IL: Human Kinetics.

Teegarden, D., Proulx, W.R., Kern, M., Sedlock, D., Weaver, C.M., Johnston, C.C. and Lyle, R.M. (1996). Previous physical activity relates to bone mineral measures in young women. *Medicine and Science in Sports and Exercise*, **28**, 105–113.

Tell, G.S. and Vellar, O.D. (1988). Physical fitness, physical activity and cardiovascular disease risk facors in adolescents: the Oslo Youth Study. *Preventive Medicine*, **17**, 12–24.

Theintz, G., Buchs, B., Rizzoli, R., Slosman, D., Clavien, H., Sizonenko, P.C. and Bonjour, J.P. (1992). Longitudinal monitoring of bone mass accumulation in healthy adolescents: evidence for a marked reduction after 16 years of age at the levels of the lumbar spine and femoral neck in female subjects. *Journal of Clincial Endocrinology and Metabolism*, **75**, 1060–1065.

Thompson, G.R. and Wilson, P.W. (1982). *Coronary Risk Factors and Their Assessment*. London: London Science Press.

Troiano, R.P., Flegal, K.M., Kuczmarski, R.J., Campbell, S.M. and Johnson, C.L. (1995). Overweight prevalence and trends for children and adolescents: the National Health and Nutrition Examination Surveys 1963 to 1991. *Archives of Pediatrics and Adolescent Medicine*, **149**, 1085–1091.

Twisk, J., Kemper, H.C.G., Mellenbergh, G.J., Van Mechelen, W. and Post, G.B. (1996). Relationship between the longitudinal development of lipoprotein levels and lifestyle parameters during adolescence and young adulthood. *Annals of Epidemiology*, **6**, 246–256.

Tylavsky, F.A., Anderson, J.J.B., Talmage, R.V. and Taft, T.N. (1992). Are calcium intakes and physical activity patterns during adolescence related to radial bone mass of white college-age females? *Osteoporosis International*, **2**, 232–240.

US Department of Health and Human Services (1996). *Physical Activity and Health: A Report of the Surgeon General*. Pittsburgh, PA: Department of Health and Human Services, Centers for Disease Control and Prevention, National Center for Chronic Disease Prevention and Health Promotion.

Vanhala, M., Vanhala, P., Kumpusalo, E., Halonen, P. and Takala, J. (1998). Relation between obesity from childhood to adulthood and the metabolic syndrome: population based study. *British Medical Journal*, **317**, 319.

Van Lenthe, F., Boreham, C.A., Twisk, J.W.R., Strain, J.J., Savage, J.M. and Davey Smith, G. (2001). Socio-economic position and coronary heart disease risk factors in youth: findings from the Young Hearts Project in Northern Ireland. *European Journal of Public Health*, **11**, 43–50.

Van Mechelen, W. (1997a). A physically active lifestyle – public health's best buy? *British Journal of Sports Medicine*, **31**, 264–265.

Van Mechelen, W. (1997b). Etiology and prevention of sports injuries in youth. In *Children and Exercise XVIII: Exercise and Fitness – Benefits and Risks* (edited by K. Froberg, O. Lammert, H. Steen Hansen and J.R. Blimkie), pp. 209–227. Odense: Odense University Press.

Van Saarse, J., Noteboom, W.M.P. and Vandenbrouke, J.P. (1990). Longevity of men capable of prolonged vigorous physical exercise: a 32 year follow-up of 2259 participants in the Dutch eleven cities ice skating tour. *British Medical Journal*, **301**, 1409–1411.

Vuori, I. (1996). Peak bone mass and physical activity: a short review. *Nutrition Reviews*, **54**, S11–S14.

Wannamethee, G. and Shaper, A.G. (1992). Physical activity and stroke in British middle-aged men. *British Medical Journal*, **304**, 597–601.

Wareham, N.J. and Rennie, K.L. (1998). The assessment of physical activity in individuals and populations: why try to be more precise about how physical activity is assessed? *International Journal of Obesity*, **22** (suppl. 2), S30–S38.

Webber, L.S., Osganian, S.K., Feldman, H.A., Wu, M., McKenzie, T.L., Nichaman, M., Lytle, L.A., Edmundson, E., Cutler, J., Nader, P.R. and Luepker, R.V. (1996). Cardiovascular risk factors among children after a two and a half year intervention – the CATCH Study. *Preventive Medicine*, **25**, 432–441.

Welk, G.J. (1994). A comparison of methods for the assessment of physical activity in children. Unpublished doctoral dissertation, Arizona State University, Tempe, AZ.

Whincup, P.H., Bredow, M., Payne, F., Sadler, S. and Golding, J. (1999). Size at birth and blood pressure at 3 years of age: the Avon Longitudinal Study of Pregnancy and Childhood (ALSPAC). *American Journal of Epidemiology*, **149**, 730–739.

White, A., Nicholaas, G., Foster, K., Browne, F. and Carey, S. (1993). *Health Survey for England*, OPCS Health Survey No. 1. London: HMSO.

Wickham, C.A.C., Walsh, K., Cooper, C., Parker, D.J.P., Margetts, B.M., Morris, J. and Bruce, S.A. (1989). Dietary calcium, physical activity, and risk of hip fracture: a prospective study. *British Medical Journal*, **299**, 889–892.

Wolf, A.M., Gortmaker, S.L., Cheung, L., Gray, H.M., Herzog, D.B. and Colditz, G.A. (1993). Activity, inactivity, and obesity: racial, ethnic, and age differences among schoolgirls. *American Journal of Public Health*, **83**, 1625–1627.