

Physical activity and resting metabolic rate

John R. Speakman^{1,2*} and Colin Selman^{2†}

¹Aberdeen Centre for Energy Regulation and Obesity, Division of Energy Balance and Obesity, Rowett Research Institute, Aberdeen AB21 9SB, UK

²Aberdeen Centre for Energy Regulation and Obesity, School of Biological Sciences, University of Aberdeen, Aberdeen AB24 2TZ, UK

The direct effects of physical activity interventions on energy expenditure are relatively small when placed in the context of total daily energy demands. Hence, the suggestion has been made that exercise produces energetic benefits in other components of the daily energy budget, thus generating a net effect on energy balance much greater than the direct energy cost of the exercise alone. Resting metabolic rate (RMR) is the largest component of the daily energy budget in most human societies and, therefore, any increases in RMR in response to exercise interventions are potentially of great importance. Animal studies have generally shown that single exercise events and longer-term training produce increases in RMR. This effect is observed in longer-term interventions despite parallel decreases in body mass and fat mass. Flight is an exception, as both single flights and long-term flight training induce reductions in RMR. Studies in animals that measure the effect of voluntary exercise regimens on RMR are less commonly performed and do not show the same response as that to forced exercise. In particular, they indicate that exercise does not induce elevations in RMR. Many studies of human subjects indicate a short-term elevation in RMR in response to single exercise events (generally termed the excess post-exercise O₂ consumption; EPOC). This EPOC appears to have two phases, one lasting < 2 h and a smaller much more prolonged effect lasting up to 48 h. Many studies have shown that long-term training increases RMR, but many other studies have failed to find such effects. Data concerning long-term effects of training are potentially confounded by some studies not leaving sufficient time after the last exercise bout for the termination of the long-term EPOC. Long-term effects of training include increases in RMR due to increases in lean muscle mass. Extreme interventions, however, may induce reductions in RMR, in spite of the increased lean tissue mass, similar to the changes observed in animals in response to flight.

**Energy expenditure: Physical activity: Non-exercise activity thermogenesis:
Excess post-exercise O₂ consumption: Training: Exercise**

The enormous increase over approximately the last 25 years in the proportion of the population in Western societies that is obese and overweight (Mokdad *et al.* 2000) has a complicated aetiology. Records of food consumption suggest that energy intakes have remained stable (Alexy *et al.* 2002) or even declined (Cavadini *et al.* 2000), and the proportion of energy derived from fat has decreased (Willett & Leibel, 2002). This finding would seem to suggest that reductions in energy expenditure must have outstripped the stability or decline in energy intake, resulting in a positive energy budget and, thereby, the development of obesity. Daily energy expenditure (DEE) can be broadly divided into several

different categories, reflecting the use to which the energy is allocated (Blaxter, 1989). These categories include resting metabolism, the heat increment of feeding and the energy expenditure associated with physical activity. This latter category being subdivided into energy utilised specifically for exercise, and the energy devoted to activity that is not exercise *i.e.* non-exercise activity thermogenesis (NEAT; Levine *et al.* 1999, 2000; Levine, 2002). This NEAT category includes minor movements and general ambulatory activity (Levine *et al.* 2001).

Unfortunately, there is little objective evidence relating to the changes in the magnitude of each of these components of

Abbreviations: DEE, daily energy expenditure; DLW, doubly-labelled water; EPOC, excess post-exercise O₂ consumption; NEAT, non-exercise activity thermogenesis; RMR, resting metabolic rate.

*Corresponding author: Professor John Speakman, fax +44 1224 272396, email J.speakman@abdn.ac.uk

†Present address: Insulin and Growth Factor Signalling Group, Department of Metabolic Medicine, Imperial College Faculty of Medicine, London W12 ONN, UK.

the energy budget to firmly establish which components have declined most and thus contributed most to the obesity epidemic. However, changes in the energy expended on the heat increment of feeding are unlikely to have outstripped the decline in food energy intake, because the heat increment of feeding is itself strongly dependent on food intake. While the heat increment of feeding is also influenced by the protein content of the diet, there is no evidence that protein intakes have disproportionately decreased as total intake has declined (Willett & Leibel, 2002). Resting metabolic rates (RMR), or BMR, have been measured in human subjects for over 200 years, since the pioneering work of Lavoisier and Seguin at the end of the 18th century. Harris & Benedict (1919) summarised data in order to generate a predictive equation for basal metabolism, and periodically since that time further reviews and equations have been published, e.g. the Robertson & Reid (1952), and Fleisch (1951) equations in the 1950s and the Schofield *et al.* (1985) equation based on over 8000 measurements. Several studies have indicated that these equations overestimate the energy demands of populations of tropical and sub-tropical regions (Henry & Rees, 1991; Case *et al.* 1997; Cruz *et al.* 1999; van de Ploeg *et al.* 2001; van de Ploeg & Withers, 2002). These differences are, however, relatively small (approximately 0.5 MJ/d) and their importance remains uncertain. There is no evidence that modern Western populations have reduced BMR relative to the predictive equations (Frankenfield *et al.* 1998; De Lorenzo *et al.* 2001). Thus, physical activity remains the most likely source of declines in energy expenditure.

There is ample anecdotal evidence to support the notion that physical activity in Western society has declined in the last 50 years. There has, for example, been a changing pattern of UK car ownership since the 1950s (Speakman, 2003). At that time, most families (86 %) in the UK did not have access to a motor vehicle and two car families comprised <1 % of the population. By 2000, however, >75 % of families in the UK had cars and 27 % had two or more cars (Department of Transport, 2000), which has affected many aspects of our behaviour, particularly that of children (Mackett, 2002). Such changes in mobility have enabled, for example, large changes in shopping habits. In the 1950s most shopping in the UK was done on foot, by walking around a collection of small local stores and carrying all the produce home in a shopping bag several times each week. The increase in car ownership in the 1960s enabled the development of supermarkets, where one-stop shopping was possible because an entire week's purchases could be loaded into the car to transport it home. The subsequent development was the introduction in the 1980s of large out-of-town hypermarket complexes where single-stop shopping could be taken to its ultimate extreme. With the advent recently of internet shopping, there is a reduction in the need to even drive to collect the shopping, since it can now be ordered on-line and delivered direct to the door.

There is no doubt that technological advances have also made an enormous impact on physical activity patterns in the space of 50 years. Domestic chores such as washing clothes and dishes, cleaning out fires and sweeping floors, all of which must have involved considerable amounts of physical activity, have now been almost completely mechanised or

eliminated with the development of automatic washing machines, central heating systems and vacuum cleaners. Indeed, the enthusiasm for electric gadgets has led to the development of equipment such as electric toothbrushes and carving knives to remove the expenditure of energy from even the most trivial of tasks. As robotics starts to become a feature of life these trends are set to continue. It has been possible since 2000 to buy robotic vacuum cleaners and lawnmowers that rove the home and garden continuously cleaning carpets and cutting lawns, although their cost at present remains prohibitive.

Alongside the decline in expenditure of time and energy on domestic activities there has been an increasing trend towards spending the time saved on sedentary pursuits. This shift to a more sedentary lifestyle is important, because the reduction in domestic activity would pose no problems if it was replaced with, for example, sporting activity. Probably the largest increase in sedentary time in the past 50 years has been the expansion of television viewing. In 1955 only 30 % of UK households owned a television set and there were two channels offering about 5 h of programming daily. By the early 1970s television ownership was almost universal, but programming has steadily increased, with the appearance of satellite and cable television available 24 h/d to ensure that there is always something of interest available to root the viewer to the couch. Consumer surveys reveal that television viewing has actually declined from a peak in the mid 1990s. However, it is still the case in the USA that 20 % of children aged 9 years watch >5 h of television daily. Moreover, the decline in television viewing may be attributable to the increase in ownership and use of computers, which is only marginally less sedentary in nature. Many studies have found correlations between the number of hours of television viewing and obesity (for example, see Tucker & Bagwell, 1991; Vioque *et al.* 2000; Dennison *et al.* 2002; Eisenmann *et al.* 2002; Janz *et al.* 2002; Lowry *et al.* 2002), although such associations are not universally found (for example, see Crawford *et al.* 1999). That these associations might be causal is suggested by the changes in physical activity and inactivity over 1 year in a large sample of 11 887 adolescents, which were correlated with changes in BMI (Berkey *et al.* 2003). Even stronger evidence for a causal link is provided by intervention studies in which an enforced reduction in television viewing reduced BMI (Gortmaker *et al.* 1999). One interesting recent study involved contingent television viewing, i.e. children could watch as much television as they wanted, but doing so was contingent on them also cycling on a tread-wheel (Faith *et al.* 2001). This massively reduced television viewing, marginally increased physical activity and produced a significant reduction ($P < 0.05$) in BMI.

Although convincing, the bulk of evidence in relation to changing activity patterns is still anecdotal, in the sense that these patterns cannot be causally linked to the development of obesity. In fact, there are some contrary trends, e.g. the expansion in enrolments at fitness clubs and the sales of home exercise equipment. Between 1987 and 1997, for example, sales of cardiovascular training equipment in the USA increased by 63 % and those of tread-wheel walking equipment by 43 %, leading to a market worth US \$2.35 × 10⁹ in 1998. Moreover, enrolments at fitness clubs

were only 13.8×10^6 in 1987, but had increased by 65 % to 22.8×10^6 10 years later (statistics from the Sporting Goods Manufacturers Association available at www.sgma.com). These trends suggest that at least some of the time released from domestic chores is being devoted to other active pursuits. Whether the reduction in energy expended on physical activity has been greater than the reduction in food intake therefore remains uncertain. At least some studies indicate that there are socio-economic (Salmon *et al.* 2000) and ethnic (Brownson *et al.* 2000) biases in these trends, perhaps explaining why some groups have proved more susceptible to obesity than others. Moreover, it is possible that the suggested reduction in energy intake itself stems from reductions in appetite linked to the reduction in activity (see Blundell *et al.* 2003), since the causes of this decrease remain uncertain. Indeed, some doubt can be cast on whether there actually has been a stable or decreased energy intake over time (Nielsen *et al.* 2002). Gross economic indicators of activity in the food segment of the economy paint a conflicting picture. Sales of most food items, with the exception of red meat, have actually increased over the past 20–30 years (for data from USA, see Kenkel & Ray, 2001); with confectionery sales and soft drink sales in particular experiencing enormous growth. Sales of chocolate confectionery in the UK, for example, grew by 22 % between 1997 and 2002, and are now worth $\pounds 3.7 \times 10^9$, while sugar confectionery has increased by 28 % since 1993 and is now worth $\pounds 1.7 \times 10^9$ annually (data from the food sector market report available at www.readymealsinfo.com).

Although elucidation of the aetiology of the obesity epidemic continues to be difficult, it is certain that our present levels of physical activity are very low relative to those of other animals. This position has been confirmed by objective measurements of daily energy demands (obtained using the doubly-labelled water (DLW) method; Schoeller *et al.* 1986, Speakman, 1997) relative to RMR, and these data can be compared with corresponding values for wild animals. Black *et al.* (1996) reviewed the available data (n 574 measurements) for daily energy demands using DLW and concluded that DEE:RMR was 1.66 for adults in contemporary Western society. This value is generally termed the physical activity level since, ignoring the minor contribution of the heat increment of feeding, it expresses the extent to which extra energy above RMR is expended on activity. A physical activity level of 1.66 shows that in Western societies the majority of energy is expended on rest (60 %), with only 40 % being expended on activity. Several reviews have been made of the daily energy demands of animals using DLW relative to RMR (Ricklefs *et al.* 1996; Nagy *et al.* 1999; Speakman, 2000). The distribution of the values is shown in Fig. 1. The use of the term physical activity level for wild animals is misleading because these animals expend energy above resting rates not only on activity but also on thermoregulation, which, with some exceptions, human subjects do not. With this caveat in mind the data indicate that wild animals vary in their physical activity levels from 1.4 – $7.5 \times$ RMR, with an average of $2.6 \times$ RMR. For wild animals the expenditure of energy on rest is, therefore, only about 32 % of the daily energy budget compared with 60 % in human subjects (Black *et al.* 1996).

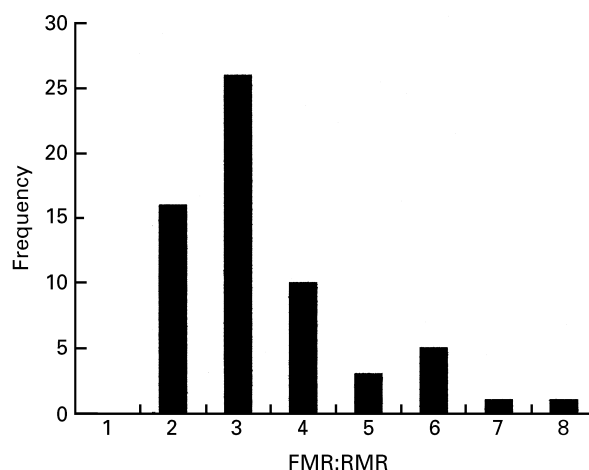


Fig. 1. Daily energy expenditure (field metabolic rate; FMR):resting energy expenditure (RMR) in free-living small mammals. The range is 1.4 – $7.5 \times$ RMR and the mean value is $2.6 \times$ RMR for n 72 species of mammal weighing < 4 kg. (Data from Speakman, 2000.)

The low DEE of modern man is exemplified by comparing the actual energy expenditures with the predictions based on allometry from the animal reviews. Adult energy expenditures reviewed by Black *et al.* (1996) suggested that the average DEE was 11.7 MJ/d. The allometric prediction based on animal DEE suggests that a 70 kg human subject should be expending 18.7 MJ/d (Speakman, 2000). Measurements of DEE for human subjects in primitive rural societies using DLW support the notion that modern Western lifestyle levels of expenditure are low. For example, male Gambian farm workers weighing only 62.1 kg on average expended 16.2 MJ/d (Heini *et al.* 1996).

An obvious question is: if reduction in energy expenditure due to reduced physical activity is the problem, can reversing this trend also be the solution and, if so, how much would need to be done (Hills & Byrne, 1998; Brill *et al.* 2002)? If the theoretical impact that a fairly typical exercise programme would have on daily energy demands is measured, it would appear that the answer to this question is no. Vigorous exercise such as jogging or rowing involves expenditure of energy at approximately 2.9 MJ/h for a 70 kg subject, which is approximately $10 \times$ BMR. Exercising at a vigorous rate of 2.9 MJ/h for 30 min on three occasions each week would amount to a total energy expenditure on this exercise of 4.35 MJ. Yet, total energy demands in 1 week using the estimated expenditure form Black *et al.* (1996) amount to 11.7×7 MJ/week or 81.9 MJ. Three 30 min vigorous exercise sessions per week might be expected then to elevate energy demands by only about 5.3 %, i.e. $(4.35/81.9 \times 100)$. This small increase in energy expenditure could be easily offset by a minor adjustment in appetite that would be undetectable at the current levels of precision for monitoring food intake.

In fact to achieve levels of expenditure compatible with expenditures of human subjects in primitive societies it would be necessary to engage in vigorous activity for approximately 90 min every day, and to push expenditures up to levels commensurate with wild animals it would be necessary to increase this activity to approximately 2.5 h

every day. Since exercise programmes involving three 30 min sessions per week generally involve substantial dropout rates (Westerterp *et al.* 1992), these much more intensive programmes would clearly be impossible to adopt in modern Western society.

Despite this theoretical argument that exercise regimens should be ineffective at reversing the tide of obesity, practical experience suggests that exercise programmes are successful at producing and sustaining marked weight and/or fat loss (Westerterp *et al.* 1992; Abdel-Hamid, 2003; Dubnov *et al.* 2003; Jakicic & Gallagher, 2003). This finding suggests that there are additional effects of exercise on energy budgets that amplify the effects of the energy expended on the exercise alone. There are three basic mechanisms that may be relevant in this context. First, exercise might suppress appetite. Second, exercise may alter fitness levels and as fitness increases individuals might alter their other behaviours, particularly the NEAT component of the daily energy budget. For example, as individuals lose weight and become fitter they may be more inclined to walk rather than use the car, or to take the stairs rather than lifts. These behavioural changes would increase the effect of the exercise. Several studies have reported positive effects of weight loss on activity (for example, see Jakicic *et al.* 2002), but the effects appear to be different in different ethnic groups (Weinsier *et al.* 2002). Finally, there may be a positive effect of physical activity on resting metabolism. Together these effects would increase the energy imbalance above that generated by exercise alone (Fig. 2) with a consequently greater than anticipated effect on body mass. In the present paper the information pertaining to the effects of physical activity on RMR will be reviewed. It is apparent that there is a large literature which involves investigation of the correlation between habitual physical activity and characteristics of subjects, including their RMR (for example, see Van Pelt *et al.* 1997; Gilliat-Wimberly *et al.* 2001). However, since cause and effect in these studies cannot be disentangled it has been decided to focus only on interventions.

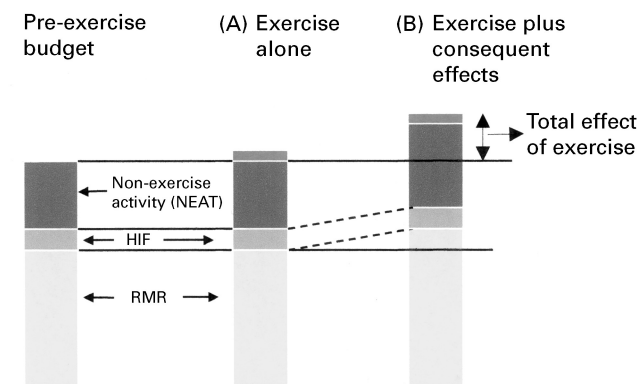


Fig. 2. Theoretical impacts of exercise on daily energy budgets. The effects of the exercise alone (A) are relatively small compared with the pre-exercise daily energy budget. However, exercise may stimulate increases in both the non-exercise activity thermogenesis (NEAT) and resting metabolic rate (RMR) components of the budget, with a resultant, much more marked, total effect (B).

Theoretical effects of exercise on resting metabolic rate

RMR is measured in a post-absorptive subject at rest in a supine posture, at thermoneutral temperatures, generally using indirect calorimetry to quantify O_2 consumption rates that are then converted to energy using the known or estimated RQ. To achieve these conditions RMR in human subjects is generally measured in the morning following an overnight fast, with the subject roused and not asleep, although some studies use the minimal metabolic rate while sleeping to estimate RMR. There is considerable individual variation in measures of RMR, but a major influencing factor is lean body mass (fat-free mass), which accounts for between 50 and 70 % of the individual variation (for example, see Westerterp *et al.* 1992; Heshka *et al.* 1993; Albu *et al.* 1997; Geliebter *et al.* 1997; Zhang *et al.* 2002). A typical pattern of variation in RMR as a function of lean body mass (Weyer *et al.* 2000) illustrating this point is shown in Fig. 3, which shows clearly that despite the large effect of lean body mass there is still a substantial residual variance in RMR. In fact, at the limits, two individuals of the same lean body mass may differ in their residual RMR by 3 MJ/d; for example, see the data for two individuals with lean body masses of 45 kg marked as A and B. Since the precision of RMR measures is about 3 % (approximately 200 kJ/d) much of this residual variance is biological in nature. This conclusion is consistent with observations that residual variation in RMR is heritable, and appears to be correlated with many physiological and genetic factors, such as thyroid status (Freake & Oppenheimer, 1995), circulating

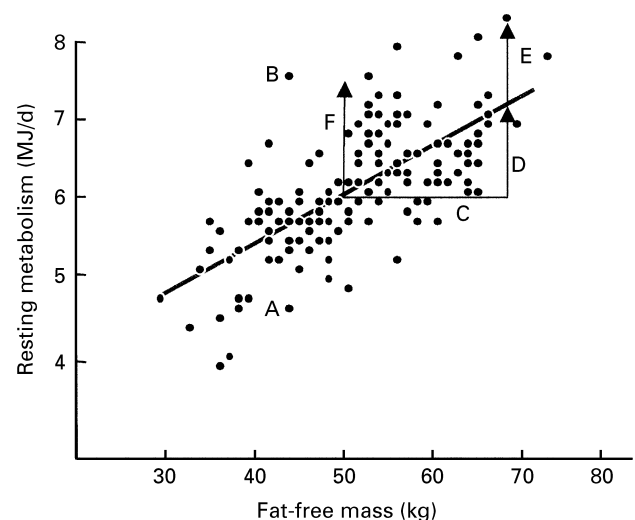


Fig. 3. Resting (sleeping) metabolic rate (RMR) as a function of fat-free mass ($n=152$). Two individuals A and B are marked with very divergent RMR despite having identical fat-free masses. The long-term effects of exercise training on an individual with a pre-exercise body composition including 50 kg fat-free mass and an RMR of 6 MJ/d might be twofold. First, fat-free mass might increase (C; here much exaggerated) leading to an increase in the RMR (D), but exercise may also stimulate those processes influencing residual variation in RMR (E), leading to a greater than expected increase compared with the effects of lean tissue deposition alone. Short-term effects of a single bout of exercise may include only the effects on residual RMR (F). (Redrawn from data from Weyer *et al.* 2000.)

tri-iodothyronine and di-iodothyronine levels (Moreno *et al.* 2002), protein turnover (Badaloo *et al.* 1996), mitochondrial proton leak (Rolfe & Brand, 1996), polymorphisms of uncoupling proteins 2 and 3 (Bouchard *et al.* 1997; Barbe *et al.* 1998; Astrup *et al.* 1999), retinoic acid X- γ (Brown *et al.* 2000), β_3 adrenoreceptors, sulphonylurea receptors (Lohrke *et al.* 1997), circulating leptin levels (Toth *et al.* 1999) and ribosomal protein L3 levels (Allan *et al.* 2000).

These aspects of resting metabolism indicate that physical activity may have two distinct effects on RMR. Since one effect of physical activity training is that it may result in the growth of lean tissue (Fig. 3; see example C), changes in RMR may be associated with the increase in lean tissue mass (Fig. 3; see example D). However, a second effect may result from the effect of physical activity on those physiological processes that influence residual resting metabolism (Fig. 3; see example E). Both these effects may occur in the long term because of changes taking place as a result of protracted periods of physical activity training. However, changes in residual RMR may also occur over much shorter periods of time (Fig. 3; see example F) following single bouts of exercise. This effect would be apparent as a transient change in RMR immediately following a bout of physical activity over a time scale too short to involve any alteration in lean tissue mass. Such changes in resting metabolism following single exercise bouts have been termed the excess post-exercise O_2 consumption (EPOC). In the present review the information available on the nature of such short-term (single event) and long-term (training) effects of physical activity on RMR in both animals and human subjects will be summarised.

Animal studies: short-term responses to single exercise bouts

Many studies have been made of the increased resting metabolism following exercise (EPOC). During activities of long duration and low intensity the proportion of the O_2 consumed by animals during recovery (EPOC) is markedly less than that expended during the actual activity itself. However, this difference is not found during short-term and high-intensity activities, where it has been estimated that EPOC may account for between 80 and 90 % of the total energy expended (Baker & Gleeson, 1998, 1999; Edwards & Gleeson, 2001). Consequently, measurements of the energy cost of exercise that do not include the period of recovery encompassing EPOC may considerably underestimate the total energetic costs of such activities. For example, in mice made to sprint at their maximum velocity on a treadmill for 5, 15, 30 or 60 s, the energetic costs when including EPOC were 1.5–2.5 times greater than when it was not included. Interestingly, the level of EPOC did not alter with the duration or intensity of the activity, if duration remained at <60 s (Baker & Gleeson, 1998, 1999). The rapid (<1 h) component of EPOC in animals appears to be stimulated by, amongst other things, replenishment of muscle and blood O_2 stores and ATP synthesis (Borsheim *et al.* 1998). The stimulatory factor(s) during longer-term EPOC (>1 h) appears to be more complicated, but may include a switch from carbohydrate to fat metabolism. In dogs (*Canis familiaris*) the longer-term EPOC is sensitive to

β -antagonists (Borsheim *et al.* 1998), indicating a role for the β -adrenergic system, which has interesting parallels in studies of human subjects (see p. 628).

Flight is the most energetically expensive mode of energy expenditure performed by any animal (Bishop *et al.* 2002; Jenni-Eiermann *et al.* 2002). Given the greater EPOC following high-intensity terrestrial exercise, compared with low-intensity exercise, it might be anticipated that the magnitude of EPOC following flight would be elevated relative to that observed following even intense terrestrial locomotion. Relatively few studies have followed the pattern of variation in RMR after periods of flight, but the results they have generated are consistent and unexpected. Speakman & Racey (1991) measured the energy costs of flight in small insectivorous bats (*Pipistrellus pipistrellus* and *Plecotus auritus*). This study involved measuring flight costs using a combination of respirometry and the DLW method (Speakman, 1997). The aim of this experiment was to label the bats with isotopes, measure their energy demands for a period of 90 min during which the isotopes reached equilibrium in body water, take an initial sample of blood to record the initial isotope enrichments and then force them to fly for a period of 30–40 min. Following the flight period their energy demands were measured during a second period of respirometry lasting about 3.5 h, before taking a final sample to assess the rates of isotope elimination. The actual energy costs of the period spent in flight could be assessed from the difference between the DLW and post-flight respirometry, while the extent of EPOC could be obtained by comparing pre- and post-flight energy demands.

During the pre-flight respirometry the metabolic rate was constant at approximately 0.55 W. Following flight, the RMR of the bats immediately after they entered the respirometer (within 5 min of exercise terminating) was observed to be at a similar level to that measured pre-flight. However, RMR then declined to a nadir of approximately 0.33 W 1 h after flight, remaining depressed and only recovering to pre-flight levels about 2.5 h later. EPOC in these bats was therefore negative. Interestingly, if the average cost of flight above resting levels was calculated, then the saving that the bats made by suppressing their metabolic rates in the post-flight period almost exactly matched the energy spent on the flights. The individual correspondence was not significant (i.e. if an individual expended less on flight than the others then the corresponding suppression during the post-flight phase was not reduced to a similar extent). However, the absence of this relationship might only reflect the inaccuracies attendant on flight-cost estimates made by subtracting respirometry from DLW (Speakman, 1993).

Bats, however, are remarkable for the adaptable nature of their energy metabolism because of the flexibility in their energetics made possible by their labile body temperatures (Speakman & Thomas, 2003). Bats may consequently have available to them mechanisms for reacting to enforced flight expenditure that obligate euthermic individuals might lack. Since our pioneering paper on negative EPOC following bat flight, other studies have been performed that involve forcing birds to fly. Birds differ from bats in the reduced variability in their body temperatures, being higher and much more stable than those of bats. These studies have reinforced the view that forced short-term flights lead

to compensatory suppression of resting metabolism during the night-time period following the period of exercise (Deerenberg *et al.* 1998).

Animal studies: long-term training effects

As mentioned earlier, longer-term increases in physical activity in animals may cause an elevation in RMR due to an increase in lean tissue mass and/or a change in various physiological processes influencing residual RMR, reflecting an increase in the metabolic rate per unit weight of the various tissues and organs that contribute to lean mass. Relatively few studies have actually measured both metabolic rates during exercise and subsequent changes in body composition (for example, see Gleeson *et al.* 1982; Ballor, 1991*a,b*; Ichikawa *et al.* 2000). Exercise protocols employed when studying animals vary considerably from voluntary exercise (Goodrick, 1980; Girard *et al.* 2001; Garland *et al.* 2002) to forced running or swimming (Gleeson *et al.* 1982; Lerman *et al.* 2002), with forced exercise potentially introducing confounding physiological and psychological variables (Moraska *et al.* 2000), which may hinder subsequent metabolic measurements.

Many studies (for example, see Wilterdink *et al.* 1993; Swallow *et al.* 2001) have reported a significant negative correlation ($P < 0.05$) between body mass and activity, primarily due to an increase in adiposity (Tsai *et al.* 1982; Swallow *et al.* 2001), although a significant positive relationship ($P < 0.01$) was observed in an inter-strain comparison of mice (Lerman *et al.* 2002). In addition, some studies have reported gender differences, with male rats generally not increasing their food intake to compensate for the increase in energy expenditure during wheel running, while females do, thereby maintaining body mass parity with sedentary controls (Holloszy, 1993, 1997).

While changes in body mass with long-term exercise are generally apparent, primarily reflecting reductions in fat mass, changes in lean tissue and in the metabolic output per unit weight of these tissues are less commonly observed. In outbred Hsd:ICR mice selected for voluntary wheel-running activity selected individuals had reduced body masses, but increased (corrected for body mass) kidney mass and a reduced *triceps surae* mass compared with controls (Garland *et al.* 2002; Houle-Leroy *et al.* 2003). These authors suggested that the small *triceps surae* (required for ankle extension) may have discrete functional characteristics and/or the specific allele for small muscles has additional pleiotrophic effects that aid wheel running (e.g. small body mass, relative increased kidney, heart and liver mass). An increase in both heart and gastrocnemius muscle was observed in mice during controlled treadmill running (Kemi *et al.* 2002). In rats after 900 d of voluntary wheel running the gastrocnemius and lateral omohyoideus muscle masses were significantly greater ($P < 0.05$) despite body mass being 'slightly reduced', and while organs such as the kidney, liver, small intestine and heart did not alter, the brain, an important contributor to RMR, was significantly greater ($P < 0.05$) in the runners (Ichikawa *et al.* 2000). In addition, metabolism was significantly higher ($P < 0.05$) in the runners at 900 d of age, with no evidence of the age-associated decline in metabolism observed in the sedentary

controls. A 10 % increase in RMR was reported in rats trained for 56 d on a treadmill compared with *ad libitum*- and pair-fed sedentary individuals, despite body mass not differing between groups (Gleeson *et al.* 1982), with an anticipatory 40 % increase in energy expenditure occurring when individuals were placed on the stationary treadmill. An increase in metabolism has also been observed in several other studies following exercise training, despite significant decreases in both body mass and fat mass (at least $P < 0.05$ in all cases; Hill *et al.* 1984; Ballor, 1991*b*; Wilterdink *et al.* 1993; Pinto & Shetty, 1995). In addition, exercise increases the levels of various aerobic enzymes (e.g. cytochrome-c oxidase and citrate synthase) in tissues closely linked to the exercise, such as skeletal muscle and heart (Moraska *et al.* 2000; Houle-Leroy *et al.* 2003), which suggests that the metabolic rate per unit weight of these tissues is increased following prolonged exercise training.

Long-term exercise training in animals generally results in an increase in RMR (Gleeson *et al.* 1982; Hill *et al.* 1984; Wilterdink *et al.* 1993; Pinto & Shetty, 1995; Ichikawa *et al.* 2000) despite the fact that both total body mass and fat mass generally decline. These changes in RMR appear to be a result of both alterations in lean mass and increases in the metabolic output per unit weight of the lean tissues. Long-term exercise elicits different physiological responses in different tissues and organs (Ichikawa *et al.* 2000; Garland *et al.* 2002), resulting in alterations in the contribution made by various organs and tissues to total lean mass, possibly reflecting the phenotypic plasticity observed in migrating shorebirds (Piersma *et al.* 1999).

In a similar manner to the short-term effects of flight detailed earlier, Nudds & Bryant (2002) examined the effects of a sustained flight-training regimen on the body composition and RMR of zebra finches (*Taeniopygia guttata*). They found that training resulted in a reduction in both daytime and night-time RMR. This reduction in RMR was paralleled by a reduction in overall body mass, which was mostly due to altered fat mass rather than an effect on lean mass. Thus, the effect of training on RMR was not attributable to a reduced lean body mass.

The majority of studies examining exercise in animals (both long and short term) have involved protocols in which the animals are forced into exercise regimens and their subsequent responses are monitored. The responses of the animals in these circumstances might be completely dependent on the forced nature of the exercise (see Moraska *et al.* 2000). If an animal is unprepared or reluctant to perform the exercise then it may engage in a period of EPOC to clear metabolites generated during the exercise or it may suppress its metabolism in the post-flight period to compensate for the energy costs of the activity. Far fewer studies have involved voluntary exercise regimens (for example, see Holloszy, 1993, 1997; Garland *et al.* 2002; Selman *et al.* 2002; Houle-Leroy *et al.* 2003). Are responses to exercise therefore conditional on the forced nature of the exercise protocol?

The effects of voluntary exercise on the body composition, food intake and RMR of short-tailed field voles (*Microtus agrestis*) have been investigated (C Selman, JS McLaren and JR Speakman, unpublished results). Field voles are an excellent model species for this type of study in

that they run voluntarily when wheels are placed in their cages. Running is almost exclusively restricted to periods of darkness, during which they will run almost continuously. During a typical 8 h period of darkness a vole will run about 8–12 km, and will do this night after night (Fig. 4). Same-gender vole sibling pairs were taken, and one was given access to a running wheel and the other one was not. After 2, 9 and 18 months of running RMR and body masses of the animals were measured (Table 1). The responses of the animals to this type of manipulation contrast those of forced-exercise regimens in that the RMR of the animals were virtually unaffected. At 2 months RMR and body mass in the exercising animals was greater than that of the sedentary controls, but by 9 and 18 months this effect had disappeared. In a general linear model including age, gender, treatment and body mass as a covariate, only body mass had a significant effect on RMR ($F_{30.9}$, $P < 0.01$). These voles made no compensatory reductions in their RMR to offset the costs of exercise, but rather elevated their food intakes.

Studies in human subjects: short term responses to single exercise bouts

Many studies of human exercise have shown that there is a positive EPOC event following single exercise bouts. A typical result (Binzen *et al.* 2001) is shown in Fig. 5, which illustrates two factors. First, the EPOC is greatest immediately following the termination of exercise, and progressively declines thereafter. Second, after 2 h there is still a slight elevation of O_2 consumption above the baseline level measured in non-exercising individuals. EPOC may therefore be usefully separated into two phases, the short-term EPOC and the more long-lasting but low level effect.

There are many changes in the physiology post-exercise that are correlated with changes in EPOC. For example, in the study by Binzen *et al.* (2001) the reduction in metabolism post-exercise closely followed blood lactate levels (Fig. 5). At least part of the immediate EPOC may therefore reflect mobilisation of the products of anaerobic metabolism

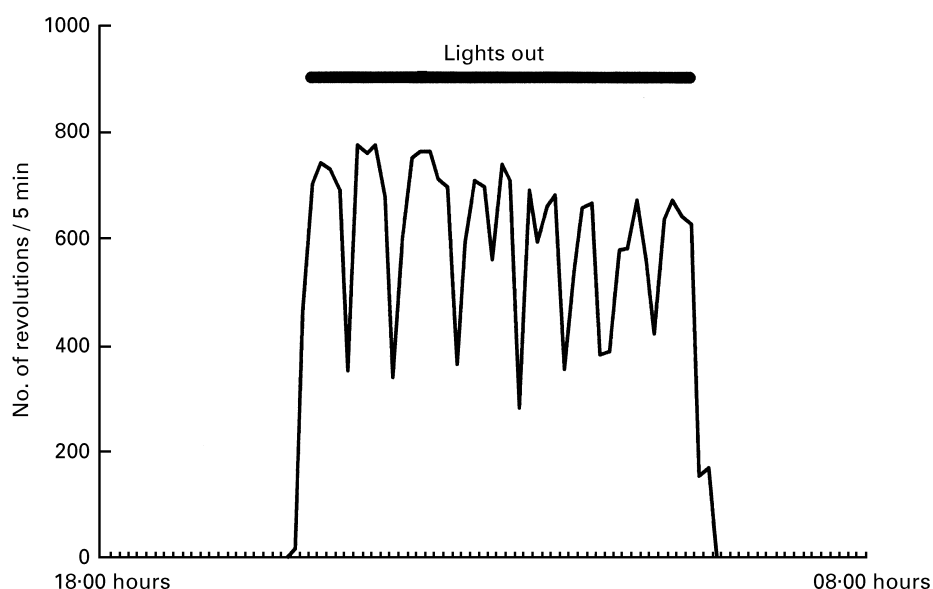


Fig. 4. Activity pattern of a single short-tailed field vole (*Microtus agrestis*) over 14 h in a voluntary running exercise protocol. (—), The room lights were out. Wheel activity (revolutions per 5 min) occurred almost continuously during the period of darkness and involved these 25 g animals running approximately 10–12 km nightly.

Table 1. Effects of voluntary exercise on the resting metabolic rate (RMR; oxygen consumption) and body mass (BM) of short-tailed field voles (*Microtus agrestis*)*

Age (months)		Exercisers	Sedentary	Statistical significance of difference	<i>n</i> (pairs)
2	RMR (ml/min)	1.28	1.06	$P < 0.001$	27
	BM (g)	23.1	20.1	$P < 0.001$	
9	RMR (ml/min)	1.51	1.42	NS	17
	BM (g)	30.8	30.9	NS	
18	RMR (ml/min)	1.43	1.41	NS	15
	BM (g)	29.9	30.9	NS	

*Voles were divided into sibling pairs, one of which was given access to a running wheel (exercisers). Measurements were made when voles were 2, 9 and 18 months of age and differences between exercisers and sedentary voles assessed using a paired *t*-test. Small changes observed after 2 months were no longer evident at 9 and 18 months of age (C Selman, JS McLaren and JR Speakman, unpublished results).

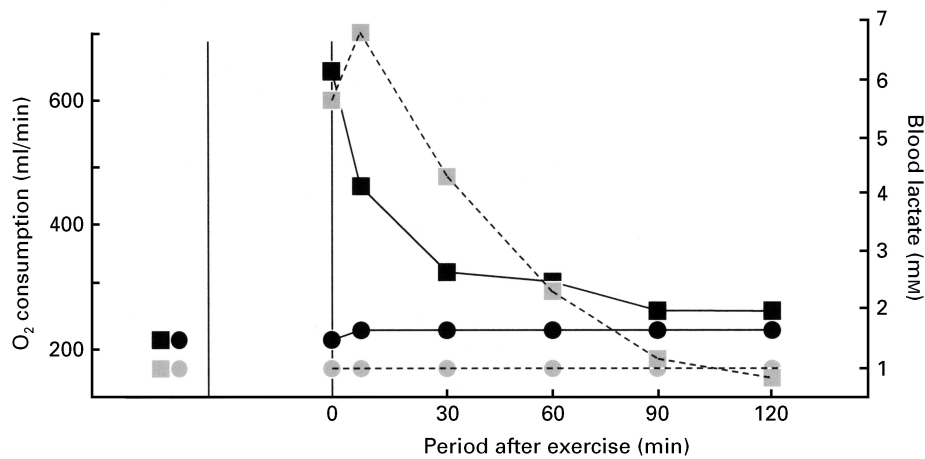


Fig. 5. Pattern of excess post-exercise oxygen consumption (EPOC) in human subjects following a bout of exercise. EPOC (■—■) declines rapidly, but after 2 h there is still a detectable elevation above background levels of non-exercised controls (●—●). The simultaneous pattern of lactate (□—□, exercised individuals; ○—○, non-exercised controls) is similar but not identical. (Redrawn from Binzen *et al.* 2001.)

(Kraemer *et al.* 1987). However, this process does not represent the full explanation, because the correlation with lactate levels is not as close as would be anticipated if it corresponded to the major proportion of the effect (for example, see Fig. 5 in which lactate is rising as metabolism declines during the phase immediately after exercise ends). Moreover, experimental manipulations in which lactate is infused during the post-exercise period do not elicit an appropriate increase in EPOC. Other factors thought to be important include elevated body temperature (Gaesser & Brooks, 1984) and increases in triacylglycerol cycling (Bahr *et al.* 1990). Generally, however, this immediate EPOC event is over by 2 h after exercise.

Some studies (Table 2) suggest that it is still possible to detect a significant ($P < 0.01$) elevation of RMR up to 48 h after an exercise session of 30–45 min (long-term EPOC). By 72 h, however, no elevation is detectable. The magnitude of the long-term EPOC effect appears to be approximately 5–10 % of the RMR. This percentage may appear small, but because the effect is protracted it can be a sizeable percentage of the total energy costs of the exercise bout. For example, if the exercise was performed at a typical rate of 1.4 MJ/h the total expenditure in a 30 min exercise session would be 0.7 MJ. A long-term EPOC lasting 36 h at 7.5 % of the RMR would cost about 0.8 MJ. Hence, more energy would be expended following the exercise than during the exercise event itself. It is clear, therefore, that the knock-on effects of exercise for RMR can be important in terms of the impact of exercise on total energy balance.

The physiological basis of the longer-term EPOC effect is unclear. However, there is some evidence to suggest that stimulation of the β -adrenergic system is involved in at least part of the effect, matching the equivalent studies in dogs mentioned earlier. This finding is important because one of the factors implicated as influencing residual resting metabolism is the status of the β_3 adrenoreceptors. Tremblay *et al.* (1992) studied the effects of β -adrenergic blockade on the long-term EPOC by giving trained and habitually-sedentary

Table 2. Long-term excess post-exercise oxygen consumption (EPOC) levels (percentage elevation of metabolic rate in exercised group when compared with controls) in a sample of studies of human subjects in which measurements extended beyond 2 h after the exercise was completed

Reference	Magnitude of EPOC (% above non-exercised control)	Time interval from completion of exercise (h)
Binzen <i>et al.</i> (2001)	18.6	2
Melby <i>et al.</i> (1992)	7.0	2
Melby <i>et al.</i> (1993)	11.7	2
	4.7–9.4	15
Osterberg & Melby (2000)	4.2	15
Dolezal <i>et al.</i> (2000)		
Trained subjects	11.0	24
	6.4	48
	0	72
Untrained subjects	24.7	24
	16.8	48
	0	72

subjects a β -blocker (propranolol) or placebo treatment following their exercise bout, and then measuring their RMR 15 h later. This long interval avoided the effects of the immediate EPOC. The study showed that when subjects were given the placebo the trained subjects had a greater elevation of RMR than the sedentary group, but that this difference disappeared when the subjects were given propranolol.

Human studies: long-term training effects

Many studies have addressed the effects of long-term exercise regimens on body weight and components of energy balance, including RMR. Generally, however, these studies have been performed most often in the context of whether combining exercise with energy restriction

enhances rates of weight loss (for example, see Geliebter *et al.* 1997). The juxtaposition of the energy restriction and exercise treatments therefore confuses the responses recorded in RMR. Nevertheless, there are many studies that have examined the influence of exercise alone, and with respect to RMR these studies have generated mixed results (Table 3). Studies have been made of both aerobic and resistance training, and in both cases a significant elevation in RMR has been demonstrated, but other studies have failed to find such an effect (Table 3).

This diversity of findings is difficult to explain. However, one potentially important factor is the time at which the RMR is measured relative to the termination of the last exercise bout. This factor could be important, because as indicated earlier the long-term EPOC event may last for up to 36–48 h. If the measurement of post-exercise RMR is conducted within this time window it is likely that an effect on RMR will be detected, which is not a long-term training effect on metabolism, rather detection of the long-term EPOC following the last exercise event. Unfortunately, retrospective examination of these studies does not always allow us to establish exactly when the final RMR measurement was taken relative to cessation of exercise. Although the sample size is small, there is an indication that the absence of an effect of training on RMR occurs in studies that leave >30 h between the last exercise bout and the RMR measurement (*n* 4) compared with

those that find a significant effect on RMR (*n* 4) that generally include measures after <24 h (Table 2).

As a result of the confounding effects of long-term EPOC and the fact that many studies do not adequately detail in their protocols the time intervals after which RMR was measured relative to the last period of exercise, reviewing this area is difficult. However, an excellent study, which overcomes these difficulties, was that performed by Byrne & Wilmore (2001). These authors had three groups of subjects who engaged in no exercise, resistance training on 4 d/week or the resistance training plus aerobic walking exercise every week. The training lasted for 9 weeks and the final measures, including body composition and RMR, were measured 72 h after the final bout of exercise (Table 4). This study showed that during both resistance training, and combined resistance and aerobic training there was a significant increase ($P < 0.01$) in lean tissue mass in the subjects, averaging 1.9 kg in both exercise groups, but only 0.5 kg (non-significant) in the controls. In the resistance-trained group RMR also increased significantly ($P > 0.05$) by 3 %. However, if RMR was expressed relative to the fat-free mass the effect was not significant (mean change -0.65 %). This finding indicates that all the increase in RMR in this treatment could be attributed to changes in fat-free mass. The most striking results, however, were in those individuals that did both resistance training and aerobic walking exercise, for whom RMR actually declined by 3.8 %, and hence when RMR was expressed relative to lean tissue mass the difference was exaggerated and declined by 7.4 %. These data are most easily viewed as RMR *v.* body mass for the resistance-trained group measured pre- and post exercise training (Fig. 6). These findings are not unique. Westerterp *et al.* (1992) enlisted subjects into a 44-week training programme for a half-marathon. This training was an intensive exercise programme that elevated energy demands by approximately 30 %. In response to this manipulation the subjects lost fat

Table 3. The long-term effects of aerobic and resistance training on resting metabolic rate*

Reference	Time interval since last exercise
Studies showing a significant increase in RMR following training	
Aerobic training	
Almeras <i>et al.</i> (1991)	
Poehlman <i>et al.</i> (1990)	
Poehlman <i>et al.</i> (1991)	
Poehlman <i>et al.</i> (1988)	24
Poehlman <i>et al.</i> (1989)	
Poehlman & Danforth (1991)	
Tremblay <i>et al.</i> (1985)	36
(1986)	16
Resistance training	
Pratley <i>et al.</i> (1994)	24
Ryan <i>et al.</i> (1995)	
Treuth <i>et al.</i> (1995)	
Studies showing no significant effect on RMR following training	
Aerobic training	
Broeder <i>et al.</i> (1992a)	48
Broeder <i>et al.</i> (1992b)	48
Davies <i>et al.</i> (1985)	
Frey Hewitt <i>et al.</i> (1990)	
Hill <i>et al.</i> (1984)	
Poehlman <i>et al.</i> (1986)	
Schulz <i>et al.</i> (1991)	
Sharp <i>et al.</i> (1992)	36
Resistance training	
Van Etten <i>et al.</i> (1995)	30

*Studies which have observed increases in RMR and studies in which no such increases have been observed. Where available in the original papers the time between the last bout of exercise and the measurement of RMR is also indicated.

Table 4. Changes in fat-free mass (FFM), resting metabolic rate (RMR) and RMR on a per kg fat-free mass basis in subjects engaged in a 9-week exercise intervention involving resistance training alone (RT), resistance training plus aerobic walking (RT/W) compared with a non-exercise intervention control group (C)*

Treatment group	RT	RT/W	C
FFM (kg)			
Pretreatment	49.5	45.6	43.9
Post treatment	51.4	47.5	44.4
Difference	+1.9	+1.9	+0.5
RMR (MJ/d)			
Pretreatment	6.07	5.81	5.89
Post treatment	6.25	5.59	5.97
Difference: MJ/d	+0.18	-0.22	+0.08
%	+3.0	-3.8	+1.35
RMR/FFM (kJ/kg per d)			
Pretreatment	123	127.6	134.7
Post treatment	122.2	118.0	135.1
Difference: kJ/kg per d	-0.8	-9.4	+0.4
%	-0.65	-7.4	+0.3

*RMR measurement was made 72 h after the last bout of exercise, thereby avoiding any confounding effect of a long-term excess post-exercise O_2 consumption event.

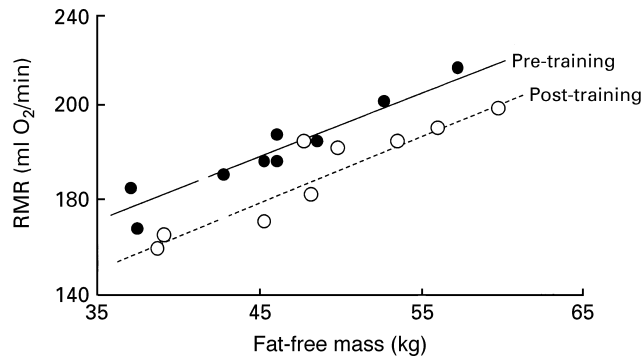


Fig. 6. Resting metabolic rates (RMR) for human subjects engaged in a mixed resistance and aerobic training programme as a function of fat-free mass, both before (●—●) and after the 9-week intervention (○---○). RMR fell despite individuals generally gaining lean mass. (Redrawn after Byrne & Wilmore, 2001.)

mass (3–8 kg in males and 2 kg in females), and gained fat-free tissue (1.6 kg in both males and females). However, despite increasing their fat-free mass, RMR declined in both groups.

The reasons for these declines in RMR in response to long-term exercise training are unclear. One possibility is that down-regulation of uncoupling protein 3 in muscle that accompanies training acts to enhance muscle mechanical efficiency during exercise (Russell *et al.* 2003). However, the knock-on effect of the lowered uncoupling protein 3 levels is an effect on subsequent resting metabolism (see the earlier suggested links between residual RMR and uncoupling proteins 2 and 3). However, this interpretation is not straightforward because the thermogenic and uncoupling nature of uncoupling protein 3 has been questioned (Vidal-Puig *et al.* 2000; Schrauwen *et al.* 2001), as has the effect of exercise on its expression (Hesselink & Schrauwen, 2003; Holloszy & Jones, 2003; Jones *et al.* 2003), and it is unclear why the effect should be more evident in aerobic training than in resistance training (Byrne & Wilmore, 2001). Another possibility, however, is that these reductions in RMR are compensatory responses by individuals to exercise regimens that are perceived to be too intensive. This response may consequently reflect a parallel situation to the forced and voluntary exercise responses in small animals detailed earlier, in which responses of animals in voluntary exercise (increased food intake and no change in RMR) were different from those in forced protocols (altered RMR either upwards or downwards depending on the intensity of the exercise). This distinction does not appear to have been made previously in interpretations of the responses to exercise protocols, but it is interesting that the appetite responses of elite athletes who voluntarily engage in exercise, appear to be different from those of normally-sedentary individuals recruited to participate in short-term exercise interventions (Blundell *et al.* 2003). Elite athletes respond to demands by elevating intake, while volunteers in interventions respond to demands by reducing body weight and compensating other components of their expenditure.

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