Physical activity does not influence obesity risk: time to clarify the public health message

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Accepted 16 July 2013

Introduction

Over the past 3 decades the obesity epidemic has spread inexorably across societies in all parts of the globe. Unfortunately our understanding of the key factors driving body weight upward – and thereby the public health interventions required to reverse this trend – has advanced much more slowly. Since the 1980s innumerable studies have catalogued the descriptive characteristics of this epidemic and detailed maps of obesity prevalence and the associated disease burden have been produced.1–3 Until recently, however, discourse about the underlying aetiology of this quintessential modern epidemic has been confined primarily to commentaries on bad dietary habits and low levels of physical activity, suggesting a failure both to restrict energy intake and to maintain high levels of energy expenditure.4 On closer scrutiny, the empirical data have been insufficient to support either element of the aetiological pathway characterized in those terms, nor is that formulation justified on theory alone. Virchow laid the foundation for our understanding of all human epidemics with the assertion that ‘mass disease means society is out of joint’.5 Recent work by nutrition scientists and economists has led to the formulation of an increasingly sophisticated explanatory model of this latest scourge – based on solid data – which is firmly rooted in traditional public health theory: ‘Changes in the global food system, including reductions in the time-cost of food, seem to be the major drivers of the rise of the global obesity epidemic during the past 3–4 decades’.6 Although recognizing the secondary, or ‘conditional’, role of ‘dietary habits’, this model clearly states that individual choice has not been the primary factor leading to the worldwide rise in long-term positive energy balance.

Within this new phase of aetiological thinking, however, there has continued to be less clarity about the role of ‘low levels of physical activity’.7 In this commentary we challenge the theoretical basis for considering reduced energy expenditure in activity as a cause of the obesity epidemic and summarize the empirical data to support that contention. From both perspectives – physiological theory as well as observational data and trials as set out below – energy expenditure in activity appears to be playing no role in either causing or moderating the obesity epidemic, suggesting that current guidelines need to be reformulated.

Long-term secular trends in activity are not consistent with the dynamics of the obesity epidemic

Over the past century and a half, mechanization has markedly reduced the requirement for hard physical labour and physically active transportation. Estimates of a decline in energy expenditure assumed to accompany increased mechanization are indirect, however, since no objective measurements span this time frame. Nonetheless, these trends away from high levels of physical activity cannot explain the increases in relative weight that have occurred during the latter half of the 20th century, as the ‘labour-saving culture’ was fully in place by the 1960s-70s.8 A recent econometric analysis of calorie intake in the USA demonstrated a slow relative decline through the 1950s, which is presumed to reflect parallel decreases in expenditure since the US population as a whole remained relatively weight stable.9 To use a term borrowed from Swinburn et al., an ‘energy balance flipping point’ appears to have been reached in the mid-1960s; following dramatic changes in the food supply, energy intake increased along with a parallel increase in body mass (Figure 1). In the decades following this ‘flipping point’, energy intake crept slowly upward, while expenditure remained stable,
Energy expenditure does not vary between societies with low vs high obesity prevalence

Comparisons of wealthy countries like the USA and Europe with non-industrialized societies, where subsistence agriculture remains the norm, can complement analyses of long-term historical trends in energy expenditure. Whereas it is often stated that low rates of obesity in rural Africa, India and China, for example, are in part attributable to strenuous daily work routines, this view is contradicted by the evidence. Stable isotopes provide the optimal method to measure energy expenditure in free-living individuals. Recent studies using doubly labelled water in rural Nigeria, for example, demonstrate no difference in calorie expenditure corrected for body size as compared with US samples of comparable age. Likewise, in another recent report, Pontzer et al. present similar findings comparing the Hadza, hunter-gatherers living in Tanzania, to ‘Westerners’; Hadza women did not have higher daily energy expenditure levels than the Western women with much higher body masses, although Hadza men were somewhat more active. In fact, a recent meta-analysis of doubly-labelled water studies concluded that neither total daily expenditure nor physical activity level differ by degree of social and economic development.

Energy intake increases to balance expenditure

Energy flux is regulated by a complex neuro-humoral system which has become increasingly well understood. Feedback mechanisms linking the gut and the brain, with additional input from fat stores and other metabolically more active tissues, regulate appetite and satiety. As with all other organisms in their natural environment, in the past – given adequate nutritional resources – the vast majority of humans ceased gaining weight after reaching full height, and remained weight stable until sarcopenia intervened in old age. This lifelong weight balance was achieved unconsciously by appetite-satiety control mechanisms. Although these regulatory pathways can be overridden, and are obviously not functioning normally in populations with a high prevalence of obesity, they retain the capacity to respond to increased expenditure. Thus, increases in energy expenditure through activity, virtually independent of the baseline state of adiposity, are generally matched over the medium range (i.e. weeks, not days) by increases in calorie intake. Perhaps from an evolutionary perspective one might argue that organisms...
which faced chronic, intermittent food shortage might have no use for a feedback loop that signalled ‘the tank is full’, but rather placed value exclusively on replacing lost stores as soon as possible.

Illustrative examples of this process have now been identified. Long a high profile candidate for providing excess calories, evidence is mounting that sugar, especially when presented as a beverage, can bypass satiety control mechanisms.19–21 The beverage industry has rebutted this assertion, and its main defence is to claim ‘it’s not just soft drinks’, which is undoubtedly true, given the rise in obesity in widely varying food cultures. Sugar-sweetened beverages may be paradigmatic, however, and provide a crucial entry point to the phenomenon of overeating. This initial skirmish with a satiety-overpowering food carries other implications for those who would formulate policy aimed at obesity control. Attempts by New York City to restrict sales of large sugared beverages has met the predictable resistance from the food industry;22 as with all other public health campaigns against harmful products in the marketplace, we can look forward to a protracted fight over food. Succeeding in an adversarial debate, as the role of hyper-consumption of food and drink required to satisfy the needs of a consumer economy is certain to be, will require great clarity and forcefulness on the part of the public health community.

Conflicting evidence from clinical trials on effect of increased energy expenditure on weight

Consistent with the physiological evidence outlined above, clinical trials provide generally consistent evidence that intake will rise to match expenditure making weight loss from increased exercise difficult, if not impossible for most people. Several carefully controlled randomized trials have shown a match of energy intake to energy expended in exercise – measured as lack of weight change – in groups undergoing supervised exercise in the absence of calorie restriction,18,23,24 although other trials have come to the conclusion that there is variability in the degree of compensation for calories expended in exercise.25–27 At the same time, numerous trials have indicated that exercise plus calorie restriction achieves virtually the same result in weight loss as calorie restriction alone.28

Observational studies show no association between energy expenditure and subsequent weight change

Numerous epidemiological studies have attempted to examine both cross-sectional and longitudinal relationships between relative weight and both total calorie intake and food composition. Most studies used self-reported food intake, resulting in a body of evidence that is fatally flawed by under-reporting of total calories among heavier individuals.29,30 The paradoxical result that has emerged is a negative correlation between body size and total calories – a mathematical equivalent of wishful thinking. When energy intake is measured using stable isotopes, however, the expected strong monotonic positive correlation between body size and actual intake is demonstrated.31 Even with objective measurement techniques, cross-sectional data are potentially confounded by lower volitional activity among those who have already gained weight.32 As a result, observational studies are relevant primarily when stable isotopes are used at baseline and weight change is measured prospectively. Relatively limited data exist using this design, but the entire body of evidence is congruent. Studies conducted among a variety of populations refute the commonly held belief that higher levels of energy expended in free-living physical activity translate into less weight being gained.32–35 Recent reviews on the relationship between physical activity and obesity or weight gain conclude that there does not appear to be one.36,37

Extremely small proportions of our society engage in levels of energy expenditure at a sufficiently high level to impact on long-term energy balance

Like dietary intake, self-report of energy expenditure in activity is subject to large random and systematic bias.38 In most epidemiological research, the construct of ‘physical activity’ has been restricted primarily to moderate and vigorous leisure-time sports, although attempts have been made to capture the more difficult occupational domain.39 Based on these data, ‘minutes spent in moderate or vigorous activity’ is often used to determine whether an individual participant meets the ‘guidelines’.40 There is no doubt that physical activity at any level promotes health, and that activity at a sufficient intensity to raise heart rate and promote cardiovascular conditioning has significant value.41,42 Across the general population in countries like the USA, however, the time spent in what are generally conceived of as ‘sport-like’ moderate-vigorous activities is extremely small. Although some surveys, such as the Behavioral Risk Factor Surveillance System Survey (BRFSS), report that about 50% of individuals in the USA currently ‘meet the guidelines’ of 30 min/day moderate activity on at least 5 days per week, the activities described include ‘gardening’, ‘walking’ and ‘golf’.33 For the first time in a national representative sample, beginning with the 2003–04 survey, the National Health and Nutrition Examination Survey (NHANES) included an
objective measure of activity.\textsuperscript{40} In an analysis of data from NHANES 2003–06, the average adult participant recorded only one bout of vigorous activity lasting a single minute. Moderate activity lasted 35 min/day among men and 21 min/day among women, but again 75% of this activity occurred in bouts of only 1 min and only 0.3% of participants met physical activity ‘guidelines’ requiring a minimum of two 10-min blocks of moderate activity per day.\textsuperscript{44} Although marathon running and other participatory sports have achieved great publicity, these involve minuscule proportions of the population. Given that an hour of vigorous activity is required to expend roughly 500 calories, leisure-time activity cannot be playing any noticeable role in the energy budget of the US population.

Conclusions
First, it must be reiterated that the health-promoting role of physical activity – and the ‘more the better’ – cannot be challenged on any grounds. The purpose of this commentary is therefore not to question the value of public health recommendations to maintain an active lifestyle, nor do we doubt that physical activity by necessity will burn calories. The purpose of this commentary is to present the evidence to support two assertions:

- First, increases in physical activity of the amount common for most individuals, such as 3 days/week of 1 h of aerobic activity, will not lead to weight loss, nor will it help prevent weight gain, for the majority of the population.
- Second, variation in physical activity within the range engaged in by the US population is not modulating obesity risk. Only reduction in calorie intake will result in weight loss, whether done in isolation or together with increases in exercise.

A rare contemporary instance of population-wide food shortage demonstrates both the immediate, universal impact of small reductions in intake, as well as the consequent drop in diabetes and cardiovascular diseases.\textsuperscript{45}

In our view, therefore, given the evolving evidence on this crucial aetiological question, the public health community should re-frame the message we communicate about the obesity epidemic. A sophisticated, coherent body of data now supports the transition to a new food environment as the fundamental cause: the ‘time cost’ of food has been lowered, and the inducements to eat high calorie items have increased, leading the susceptible half of the world’s populations to gain 0.5–1 kg per year from age 18 years onward. At the same time, as outlined above, reduced energy expenditure has been removed from the list of candidate aetiological agents. These messages should be de-coupled and we should focus with great urgency and vigour on the challenge of altering the modern food environment.

Conflict of interest: None declared.

KEY MESSAGES

- Public health guidelines advocate increased physical activity as a key modality in the prevention and treatment of obesity despite little empirical evidence to support its effectiveness.
- Secular trends in activity levels are not consistent with trends in obesity prevalence nor do data from observational or clinical trials support a direct relationship between activity and excess weight gain.
- A complex neuro-humoral system regulates energy balance through satiety and appetite, preferentially matching intake to increased – rather than decreased – activity in free-living individuals.
- Physical activity is crucially important for improving overall health and fitness levels, but there is limited evidence to suggest that it can blunt the surge in obesity.

References
Commentary: Luke and Cooper are wrong: physical activity has a crucial role in weight management and determinants of obesity

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Accepted 1 March 2013

Introduction
Luke and Cooper claim that evidence does not support a relationship between physical activity (PA) and obesity, and that policy recommendations to control the obesity epidemic should be limited to ‘altering the modern food environment’. We disagree and contend that to support their position Luke and Cooper misrepresent and/or ignore an extensive evidence base of observational and experimental studies that clearly support an effect of PA on obesity, as follows.

Secular trends in physical activity
In direct contradiction to claims that secular trends in PA are not consistent with trends in obesity prevalence and that the ‘labour-saving culture was fully in place by the 1960s–70s’, Church et al. examined US occupational PA from more than 140,000 businesses and government agencies, and >440,000 individual worksites. They report a significant decreasing trend in occupational energy expenditure over the past 50 years. Mathematical modelling showed that decrements in occupational PA were 140 and 120 kcal/day in men and in women, respectively. Their model accurately predicted changes in body mass index (BMI) (National Health and Nutrition Examination Surveys) for men and women from 1971 to 2006. We examined household energy expenditure over the past 45 years and found decrements of >1800 kcal/week for all women and >2500 kcal/week in non-employed women. These results combined with decreases in active transport suggest that the decrements in PA energy expenditure in multiple domains surely must be an important cause of population increases in weight. It is implausible that the ‘labour-saving culture’ has not changed since the 1960s–70s.

Observational cohort studies
There are numerous longitudinal studies showing that PA prevents weight gain. One excellent example by Hankinson et al. examined 3554 men and women with seven follow-up measurements over 20 years and concluded that maintaining high activity levels lessens gains in weight and visceral adiposity. Women and men who maintained PA at recommended levels had smaller increases in both BMI and waist circumference when compared with inactive individuals.

Ecological studies
Perhaps the gravest misrepresentation by Luke and Cooper is citation of data from the Cuban economic
