**INVITED PERSPECTIVE**

The Mystery of Energy Compensation

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Accepted 7/12/2021; Electronically Published 9/16/2021

ABSTRACT

The received wisdom on how activity affects energy expenditure is that the more activity is undertaken, the more calories will have been burned by the end of the day. Yet traditional hunter-gatherers, who lead physically hard lives, burn no more calories each day than Western populations living in labor-saving environments. Indeed, there is now a wealth of data, both for humans and other animals, demonstrating that long-term lifestyle changes involving increases in exercise or other physical activities do not result in commensurate increases in daily energy expenditure (DEE). This is because humans and other animals exhibit a degree of energy compensation at the organismal level, ameliorating some of the increases in DEE that would occur from the increased activity by decreasing the energy expended on other biological processes. And energy compensation can be sizable, reaching many hundreds of calories in humans. But the processes that are downregulated in the long-term to achieve energy compensation are far from clear, particularly in humans—we do not know how energy compensation is achieved. My review here of the literature on relevant exercise intervention studies, for both humans and other species, indicates conflict regarding the role, if any, of basal metabolic rate (BMR) or low-level activity such as fidgeting play, particularly once changes in body composition are factored out. In situations where BMR and low-level activity are not major components of energy compensation, what then drives it? I discuss how changes in mitochondrial efficiency and changes in circadian fluctuations in BMR may contribute to our understanding of energy management. Currently unexplored, these mechanisms and others may provide important insights into the mystery of how energy compensation is achieved.

Keywords: activity, activity energy expenditure, basal metabolic rate, energy management, exercise, nonexercise activity thermogenesis (NEAT).

We might imagine that if we undertake a daily exercise regime burning 300 kcal each session, this results in our daily energy expenditure (DEE) increasing by 300 kcal. Indeed, this is the received wisdom on how activity affects energy expenditure—the more activity is undertaken, the more calories will have been burned by the end of the day (FAO/WHO/UNU 2001; World Health Organization 2014). Yet, there has been recognition for many centuries, in the scientific literature at least, of “physiological limitation” or “material compensation” going back as far as Aristotle (Egerton 1973) and considered similarly by Darwin (“balance of growth”; Darwin 1894) and by Rubner (1910), who argued that not all organs can be in a state of high activity simultaneously. And there is now a wealth of data, both for humans and for other endothermic animals, demonstrating that lifestyle changes involving chronic increases in exercise or other physical activities do not result in commensurate increases in DEE. Rather, humans and animals exhibit at least a degree of what herein will be called “energy compensation” at the organismal level, ameliorating some of the increases in DEE that would occur from the increased activity by decreasing the energy expended on other biological processes. From the perspective of the principle of allocation on which life history theory was founded (Sibly and Calow 1986), energy compensation is to be expected because, given limited resources, an animal will benefit from trade-offs in the allocation of those resources to various endogenous processes (Glazier 2009), which could include not only activity but also feeding, growth, and reproduction.

Yet, at present, work from the field and the lab for both humans and other animals draws contradictory conclusions about the biological processes underpinning energy compensation. It is often unrealized that the literature is very unclear on what aspects of physiology and/or behavior are downregulated, and there may be key factors of importance that have not yet even been considered. In this review, I discuss the lines of evidence that concur and conflict and what future studies are probably necessary to elucidate the behavioral physiology of energy compensation. I focus on energy compensation in response to activity, but note here that energy compensation may arise in response to increases in energy for other processes, such as growth (Sears 2005; Reid et al. 2011) and reproduction (Koch and Wieser 1983; Becker et al. 2013).

Evidence for the Existence of Energy Compensation

DEE adjusted for weight and age is similar between human populations of developing and industrialized nations around the world despite the diversity of lifestyles and wide range of

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Physiological and Biochemical Zoology, volume 94, number 6, November/December 2021. © 2021 The University of Chicago. All rights reserved. Published by The University of Chicago Press. https://doi.org/10.1086/716467
physical activity levels represented by those groups (Dugas et al. 2011; Pontzer et al. 2012; Pontzer 2018). Analogously, while animals in captivity are considerably less active than their wild counterparts—as observed in, for example, macaques (Macaca nigra; Melfi and Feistner 2002), gibbons (Hylobates lar; Warren 2010), chimpanzees (Pan troglodytes), gorillas (Gorilla gorilla; Ross and Shender 2016), and tigers (Panthera tigris; Breton and Barrot 2014)—the energy expenditures of wild and captive animal populations are similar (Stephenson et al. 1994; Munn et al. 2013; Pontzer et al. 2014; Nie et al. 2015). The findings of intervention studies concur with these observations. Human participants exhibit a smaller increase in DEE than expected when prescribed daily exercise levels are increased (Goran and Poehlman 1992; Wing 1999; Keytel et al. 2005; Garland et al. 2011; Dhurandhar et al. 2015; Herrmann et al. 2015; Hand et al. 2020; Willis et al. 2020). Moreover, the longer and greater the exercise intervention, the greater the estimated energy compensation exhibited (for a summary, see Pontzer 2018 and fig. 2 therein).

Similarly, experimental studies on animals have usually found that individuals obliged to do more physical work to gain a unit of food nonetheless exhibit a limited increase in DEE (Pontzer 2015; O’Neal et al. 2017; Lark et al. 2018).

These data representing multiple species in the field and laboratory provide clear evidence that for extended periods of time during which overall activity levels are increased and thus the energy expended during that activity (activity energy expenditure [AEE]) is increased, there is a reduction in the energy expended by the body on certain processes that at least partially compensates. But which processes compensate is somewhat of a mystery. Thurber et al. (2019, p. 2) write that the processes underlying metabolic compensation in ultradistance runners probably include a “reduction in nonexercise activity and reduction of physiological activity in other organ systems,” while Pontzer (2018, pp. 386–387) argues that the evidence to date for both humans and other animals suggests that “changes in other non-musculoskeletal physiological activity contribute to energy compensation.” These broad statements in major, recent papers investigating energy compensation reflect the fact that while energy savings have been documented a multitude of times, we do not yet understand where the body makes those savings.

State of the Art regarding How Energy Compensation Is Achieved

Publications on this topic give credence to the idea that to compensate for higher energy expenditures during activity, two broad categories of the energy budget could in principle be reduced. First, there are the costs of low-level activity, formerly termed “nonexercise activity thermogenesis” (NEAT; Garland et al. 2011) or “spontaneous physical activity” (Martin et al. 2007), which includes fidgeting (Mehrabian and Friedman 1986; Levine et al. 1999) and pottering as well as postural costs, such as to sit or stand (Tickle et al. 2012; Popp et al. 2018). Second, there are the costs of physiological processes that contribute to basal metabolic rate (BMR) and thus daily basal energy expenditure (BEE; an estimated value of total BEE, derived from BMR and which assumes that BMR is constant through the day).

Evidence for Reductions in NEAT

Many, perhaps most, studies and reviews of energy compensation have assumed that the process at play involves energy-saving changes in NEAT (e.g., Goran and Poehlman 1992; Morio et al. 1998; Meijer et al. 1999; Hand et al. 2020), and some work seems to provide indirect evidence for this. For example, an across-school study of children reported that the amount of intense physical activity they undertook at school did not relate to their total levels of activity over the entire day (i.e., levels of NEAT were compensating for levels of intense physical activity; Mallam et al. 2003). Similarly, a study of elderly participants found that they exhibited no increase in daily activity levels during periods when they participated in a physical training intervention (Meijer et al. 1999). A recent experiment on overweight women reported that exercise did not induce an increase in their DEE or in their resting energy expenditure and argued that this left NEAT as the facet of energy expenditure that was decreasing to compensate (Riou et al. 2019).

However, NEAT has also been reported unchanged (e.g., Blaak et al. 1992; Rangan et al. 2011; Willis et al. 2020) or increasing (e.g., Meijer et al. 1999; Westerterp et al. 1992; Hollowell et al. 2009) in response to heightened levels of exercise, and most studies have not directly measured it (Melanson 2017). Reviews of the literature to determine whether NEAT in humans decreases to compensate or partially compensate for increases in AEE deem the evidence to be conflicting (Melanson et al. 2013; Washburn et al. 2014; Fedewa et al. 2017; Melanson 2017). The main conclusion stated by all four papers is that there is not the evidence overall in the literature to infer that NEAT systematically decreases in response to either short-term or long-term increases in daily exercise levels. I summarize their pertinent, subsidiary conclusions here: First, the energy costs of NEAT are a strong predictor of DEE and vary widely within and between people. Second, this variation may in part be explained by marked individual differences in NEAT-driven compensatory responses and the fact that few studies thus far have measured NEAT directly. Third, shorter exercise sessions may influence NEAT less than longer sessions, while decreases in NEAT may attenuate through the exercise period.

The animal literature here is dominated by mouse studies. These papers arguably provide clearer evidence of reductions in NEAT in response to greater activity levels (wheel running) than do the human studies (De Carvalho et al. 2016; O’Neal et al. 2017). However, the resulting degree of compensation appears fairly small (∼5% reduction in DEE representing an attenuation in the increase in DEE due to wheel running of about 20%; Lark et al. 2018), which may mean that other energy compensation mechanisms are also at play. And these could include the arguably confounding reduction of nonshivering thermogenesis in response to the muscle thermogenesis from wheel running (Even and Blais 2016) because mice might often be housed at ambient temperature below their thermoneutral zone (Speakman and Keijer 2013; O’Neal et al. 2017). A study of responses by
starlings (Sturnus vulgaris) to food insecurity offers inconsistent evidence for reduced physical activity (Bateson et al. 2021). For human studies at least, more sophisticated investigations are required to clarify the relationship between AEE and NEAT, employing methods that can study NEAT directly (Dugas et al. 2011), perhaps, for example, with accurately calibrated, sensitive activity monitors.

Evidence for Reductions in BMR

The theory behind the idea that BMR (and thus BEE) decreases and therefore compensates for increases in AEE is that certain physiological processes required to maintain homeostasis, such as perhaps immune competency, protein turnover, and somatic repair, are somehow deprioritized and thus become down-regulated when energy compensation is a more pressing homeostatic driver (Wiersma and Verhulst 2005; Pontzer 2018; n.b., whether such changes in metabolism are driven by top-down regulatory mechanisms or are the result of bottom-up intercellular competition for resources needs further debate elsewhere; see, e.g., Archer et al. 2018b). Such downregulation could explain increases in oxidative stress and DNA damage in animals required to apply more effort (for a review, see Soulsbury and Halsey 2018). In extremis, this downregulation could slow growth or cause the onset of disease and impairments to ovulation and reproduction (Perrigo and Bronson 1983; Lebennstedt et al. 1999; Melin et al. 2015). However, the amount that changes in BMR contribute to energy compensate for increases in AEE is far from clear (Herrmann et al. 2015).

It is widely accepted by most, but not all (Mitchell et al. 2017), that BMR in humans decreases in response to a negative energy balance at the organismal level (see also Martins et al. 2020), even when statistically accounting for changes in body mass and condition (Leibel et al. 1995; Martin et al. 2007; Schwartz and Doucet 2010; Johannsen et al. 2012; Hopkins et al. 2014). This is also the case in other primates (Yamada et al. 2013) and rodents (Hambly and Speakman 2005). Interventions that increase activity levels often lead to changes in body composition, such as weight loss and increased levels of fat-free mass, which in turn affect BMR (Silva et al. 2012), making it difficult to justify explaining energy compensation in response to increased activity as being due to a decrease in BMR. To be clear—in response to exercise interventions that induce weight loss, even if a decrease in BMR is adjusted for changes in body composition, such as overall weight, fat-free mass, and/or fat mass (which may be very difficult to do effectively; Heymsfield et al. 2018), we cannot assert that this downregulation in metabolic rate is not at least in part the body responding to negative energy balance (Tremblay et al. 2013). Only studies that report a decrease in BMR alongside increases in activity levels and no substantive decrease in body mass can reasonably be used to infer that energy compensation in response to heightened activity per se can include downregulation of BMR.

It is important to recognize that, in the short-term, BMR often increases in response to increases in activity. Excess post-exercise oxygen consumption (EPOC) is the elevation of metabolic rate above resting levels for a period after the completion of exercise, which tapers away over time. Extensive literature on aerobic and resistance exercise, reviewed by Laforgia et al. (2006), indicates that EPOC typically lasts up to a few hours. Longer bouts of activity, however, can result in EPOC continuing for many hours (Melby et al. 1993; Tuominen et al. 1996; Short and Sedlock 1997), sometimes a day (Bielsinska et al. 1985; Maehlum et al. 1986) or even two (Sjödin et al. 1996; Williamson and Kirwan 1997). Therefore, only studies that compare BMR against activity levels for the long-term have the potential to record a decrease in BMR (and thus BEE; Pontzer 2018; fig. 1). Figure 1 also serves to highlight that AEE is often calculated rather than measured; in these cases, it is determined by subtracting BEE (and sometimes also digestion costs) from DEE, and thus inaccuracies in the former will result in over- or underestimations in AEE.

I find very limited evidence in the human literature of BMR decreasing in response to a chronic period of increased AEE

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Figure 1. Effects of excess postexercise oxygen consumption (EPOC) on the disaggregation of daily energy expenditure (DEE) into basal energy expenditure (BEE) and activity energy expenditure (AEE). If EPOC subsequent to activity is still present at the point that an attempt is made to measure basal metabolic rate, BEE will be overestimated, and in turn, AEE will be underestimated (light gray area in the stack). A color version of this figure is available online.
per se. In most studies where participants exhibited no change or only a marginal decrease in body mass in response to an increase in daily activity levels, they also exhibited no change in BMR (often calculated independently of body condition; Goran and Poehlman 1992; Van Etten et al. 1997; Meijer et al. 1999; Colley 2008; Colley et al. 2010; Willis et al. 2014; Herrmann et al. 2015; Riou et al. 2019; Flack et al. 2020; Hand et al. 2020). In a few studies where participants experienced no change in body mass, they exhibited a small increase in BMR (Morio et al. 1998; Withers et al. 1998; Hunter et al. 2000). Two papers report a decrease in BMR despite little to no change in body weight (Westerterp et al. 1992; Silva et al. 2017), but the decrease is modest and would account for only a small proportion of the observed energy compensation in those studies (Pontzer 2015). A meta-analysis by MacKenzie-Shalders et al. (2020) that focused on studies of healthy nonelderly participants, although typically the participants were overweight, found that papers reporting a stable body mass during the experimental intervention also reported an increase in resting metabolic rate (n.b., none of these studies included a dietary component to the intervention). While they argue that in some studies resting metabolic rate might be overestimated because of EPOC if the measurement was taken too soon after the most recent bout of the exercise intervention (fig. 1), nonetheless there is no evidence from this work of a decrease in BMR in response to an increase in AEE.

On the other hand, a reanalysis of the Westerterp (1992) study by Careau (2017) suggests that when this repeated-measures data set is analyzed for not only across individuals but within-individual correlations, there is clear evidence of energy compensation within individuals on the basis of the slope of the relationship between DEE and BEE being <1. Caution, however, must be taken with such analyses because the statistical effect of regression dilution will to some degree underestimate the true slope value, creating artifactual energy compensation (Halsey and Perna 2019). Colleagues and I recently conducted an analysis on another repeated-measures data set comprising paired energy expenditure data for a field survey of 1,756 elderly individuals, with the first set of measurements taken between 1998 and 2000 and the second set taken about 7 yr later. Within individuals, variations in AEE were negatively associated with variations in BMR, and thus BEE, accounting for age, fat-free mass, and fat mass (Careau et al. 2021), and subsequent analysis confirms that AEE and body mass are not correlated (V. Careau, personal communication). These findings, then, provide strong evidence of substantial decreases in BEE resulting from increases in AEE per se. Specifically, the data suggest that the decreases in BMR can compensate for more than a quarter of the increase in energy expenditure due to increases in activity (Careau et al. 2021).

Does the Animal Literature Mirror the Human Literature on BMR-Driven Compensation?

Microchiroptera bats reduce their resting metabolic rate after a period of flying (Speakman and Racey 1991), but I am not aware of other field studies that have reported or investigated this phenomenon. There are, however, a number of lab-based exercise intervention studies on birds and mice that measured BMR and DEE. In 2015, Herman Pontzer reanalyzed these studies to show that, in most cases for both birds and mammals, when activity levels increased (such as more time spent flying or wheel running), BMR decreased, sometimes subtly and sometimes substantially (Pontzer 2015 and fig. 2 therein). However, in all of the studies reviewed by Pontzer (2015) that visually demonstrate a substantial decrease in BMR, daily food intake and body mass had also decreased substantially in association with the increased activity levels (Perrigo and Bronson 1983; Tiebout 1991; Bautista et al. 1998; Vaanholt et al. 2007). In contrast, for those studies that visually indicate at most only a subtle decrease in BMR (Deerenberg et al. 1998; Weimerskirch et al. 1992; Westerterp et al. 1992; Wiersma and Verhulst 2005), body mass also decreased at most only slightly. There is one exception: in a study of starlings, body mass decreased quite a lot, and yet BMR decreased only modestly (Wiersma et al. 2005). This is also the one study where food intake increased as AEE increased. Overall, then, interpretation of exercise interventions in the animal literature across birds and mammals indicates that reductions in BMR due to increased AEE per se are modest at best when body mass remains constant—this concurs with much of the human literature with the exception of at least the aforementioned field study by Careau et al. (2021).

Field data on animals encourage a contraposition, albeit with caveats. Reduction in basal metabolism appears to have been observed indirectly in a range of animal species in the field through long-term measurements of heart rate. Heart rate correlates with metabolic rate in endothermic species (Green 2011). The harder an animal is working, the faster its heart beats to support upregulation of the cardiorespiratory system, in accordance with Fick’s principle (Fick 1870). The lowest mean heart rate per day probably represents heart rate while the body’s metabolism is basal, and changes in daily lowest mean heart rate probably indicate changes in daily BMR. Halsey et al. (2019) found that individuals of many species, including birds, mammals, and fish, exhibited lower minimum heart rates during periods when their daily heart rate, and thus daily metabolic rate, was higher. This suggests that during periods when AEE is higher, BMR decreases. Indeed, investigating the year-round data sets that were available for red deer (Cervus elaphus), alpine ibex (Capra ibex), and graylag geese (Anser anser) showed that during months when daily heart rate was higher (which tended to coincide with key annual events such as reproduction), the slope between daily heart rate and minimum heart rate was particularly shallow, indicating strong energy compensation through the decrease of BMR. There is not, however, information on body mass changes over time, and so we cannot rule out the possibility that reductions in BMR are driven by body mass loss rather than heightened AEE per se. There is also the possibility that during periods when activity levels are high, the heart adaptively increases in size, affecting the relationship between heart rate and metabolic rate; a slower, larger heart could be associated with the same metabolic rate as a faster, smaller heart. Thus, the aforementioned relationships for deer, ibex, and geese are conceivably driven by changing levels of physical fitness.
A data set of more direct measures of metabolic rate alongside changes in body mass is necessary to confidently ascertain whether heightened AEE per se is associated with a down-regulation of BMR in animals. Until then, it is worth noting that the findings from the aforementioned heart rate–based data for animals in the field (Halsey et al. 2019) are analogous to those of the study mentioned earlier on humans in the field (Careau et al. 2021), perhaps offering support that the animal data can indeed be interpreted at face value.

**Summary of the Literature on BMR- and NEAT-Driven Compensation**

Synthesizing the findings of the various papers cited above does not return a clear conclusion about the importance of BMR in energy compensation in response to AEE increases. The results of a field survey for humans and field surveys for other animals, where variations in AEE are not usually determined by prescribed exercise regimes, suggest that BMR downregulation can be a substantial—perhaps the substantial—element of energy compensation. In contrast, lab-based exercise interventions indicate a limited role, if any, of BMR. And for humans specifically, lab-based studies also indicate an unclear role for NEAT. When a reduction in NEAT is recorded, it can rarely account for a substantial proportion of the observed energy compensation (Colley et al. 2005; Pontzer 2015; Pontzer et al. 2016). In contrast, the few experiments of NEAT in animals, all on mice, provide clearer evidence of its substantive role in energy compensation.

**Investigating Three Studies in Detail**

What then explains the substantial energy savings reported in many human studies? Certain published papers are the most suitable, on the basis of their data sets, for exploring the possible contributions of both NEAT and BMR to observed energy compensation.

First, Thurber et al. (2019) measured the DEE, BMR, and body condition of six athletes participating in a transcontinental marathon event called the Race Across the USA (RAUSA). While the runners had very high DEEs due to running a marathon each day, they also energy compensated for the running AEE (~3,000 kcal) by an average of 600 kcal/d, according to calculations of predicted versus measured DEE. None of this energy compensation is explained by decreases in absolute BMR, which, if anything, marginally increased (despite a slow but cumulatively substantive decrease in body mass of 4.1 kg over ~18 wk). Potentially, a considerable proportion of this 600 kcal can be explained by a reduction in NEAT or fidgeting-like behaviors (Ravussin et al. 1986; Levine et al. 1999), for example, sitting or lying motionless rather than sitting or standing while fidgeting (Levine et al. 2000), perhaps driven by fatigue. While NEAT was not measured in this study, if we assume that the runners were sleeping 8 h/d and running 5 h/d (leaving 11 h/d to vary the amount of fidgeting-like behaviors), according to measured energy costs of fidgeting in Levine et al. (2000, table 1 therein), by mostly resting without fidgeting, a runner might save up to 700 kcal/d compared with their typical fidgeting-related energy expenditure during periods outside of ultradistance events. This is very similar to the maximum energy expenditure due to increases in fidgeting-like behaviors by participants in response to overeating reported by Levine et al. (2000), although this value might be considered high because 700 kcal is the typical energy expended to run about 10 km. An alternate possibility is that the metabolic rate of various tissues decreased during the long periods of running because so much available energy was being diverted to the skeletal muscle—an example of the argument that different tissues compete, often asymmetrically, for energy (Archer et al. 2018b). However, taking a BMR of 1,500 kcal/d in a chronically fed state and assuming that this represents the metabolic costs of organs and tissues (Müller et al. 2013), even if most of the organs except the musculature, cardiorespiratory system, and brain consumed no energy during the periods of running, this would result in whole-body energy compensation of little more than 100 kcal during the periods of running. Thus, periodically energy-deprived organs could explain part, but by no means all, of the whole-body energy compensation recorded in the RAUSA athletes.

Second, Westerterp et al. (1992) published analyses for a study somewhat analogous to that by Thurber et al. (2019) of people spending 44 wk training for their first half-marathon. The data ultimately suggest that reductions in NEAT are more substantial than reductions in BMR. While reanalysis of the Westerterp et al. (1992) data, in contrast to the findings of Thurber et al. (2019), provides strong statistical evidence ($P = 0.003$) that the participants exhibited a decrease in BMR, this decrease was fairly moderate in magnitude (median sleeping metabolic rates; week 0: 6.5 MJ/d; week 40: 6.0 MJ/d; Westerterp et al. 1992 and table 2 therein). During that period, fat-free mass increased by 3 kg while body mass decreased by 2 kg (Westerterp et al. 1992 and table 4 therein), suggesting that changes in body composition are unlikely to explain these BMR changes either through a change in body condition or because of negative energy balance at the organismal level (mean rate of mass loss was only 50 g/wk). This apparent change in BMR represents an energy saving per day of 120 kcal (i.e., enough to compensate for moderate activity lasting ~30 min or heavy activity lasting ~10 min). But while this downregulation in BMR is arguably of functional significance, it explains only a fraction (~10%) of the estimated energy savings of around 1,200 kcal d$^{-1}$ (5 MJ; Pontzer 2015 and fig. 2A, 2B therein; fig. 2), leaving the rest to potentially be explained by decreases in NEAT. However, surely this deficit is too large to be filled by less fidgeting and changes in posture.

Finally, data from a study that attempted to directly measure NEAT in a group exhibiting energy compensation do not indicate that these people are reducing NEAT. However, I will argue that reduced NEAT is still a possible explanation. DEEs of children from the United Kingdom and the United States and those of children of Shuar forager-horticulturalists are very similar despite the Shuar children being 25% more physically active and also having a greater resting energy expenditure (probably due to an upregulated immune system; Wolowczuk et al. 2008; Urlacher...
et al. 2018, 2019 and fig. 1B therein). Urlacher et al. (2019) report that the Shuar children seem to compensate for this by savings in their energy expenditure associated with activity, but they concede that how these savings are made is unclear. Scope for humans to become more energy efficient at a given activity appears very limited. Burgess and Lambert (2010) report that the evidence for increased running efficiency in response to exercise training is mixed and, when present, modest at 3% energy savings although savings may be greater at low speeds. (Tremblay et al. [1997] report a 12%, 7%, and 3% energy saving when walking at 4.5, 5.5, and 6.5 km/h, respectively, after a 93-d training program.) This seems to leave the possibility that, similarly to the proposed mechanism for the ultramarathon runners and the half-marathon runners in training, the Shuar children reduce their fidgeting-like behaviors.

Although the activity counts on the accelerometers worn on the hip by the Shuar children indicate that they are considerably more active than United Kingdom and United States children in total across the waking day, just possibly the Shuar children nevertheless exhibit reduced fidgeting behaviors, such as arm movements under certain circumstances (Fernández-Verdejo et al. 2021)—a difference not sensed by the hip-instrumented device because of its limited capacity to recognize NEAT (Kozy-Keadle et al. 2011). Returning to the values provided by Levine et al. (2000 and table 1 therein) for adults and halving them to account for the equivalent energy expenditures of 8-yr-old children, in the possible scenario that children sit for half the waking day and stand for the other half but Shuar children are motionless while doing so whereas United Kingdom/United States children are fidgeting, the Shuar children would save 400 kcal/d compared with the United Kingdom/United States children, and yet the accelerometer records little of the fidgeting activity in the United Kingdom/United States group. In reality, of course, both groups are moderately or vigorously active for several hours per day during which we can expect the accelerometer count to accurately reflect activity levels; nonetheless, there are a number of hours when they are sedentary (Urlacher et al. 2019 and table 1 therein) such that reduced fidgeting could provide the Shuar children with substantive energy compensation.

Overall, then, these three studies considered in detail together suggest that BMR is at best a minor element of energy compensation. NEAT is a more promising explanation but is yet to be adequately measured.

**What Other Aspects of Energy Expenditure Could Be Involved in Energy Compensation?**

Studies to date tend to assume that energy compensation is explained by BMR, NEAT, or both, but there are other possible explanations yet to be considered.

**Attenuation in Daily Fluctuations of BMR**

Circadian fluctuations in BMR can reach 10% (van Moorsel et al. 2016; Zitting et al. 2018) but are theorized to be attenuated in individuals that are energetically stressed (Urlacher et al. 2019; Pontzer 2021, ch. 8), that is, in individuals that are ingesting fewer calories than required for the body to expend on all energetic processes optimally. BMR measurements are typically taken in the early morning, most commonly around 5:00 a.m., when the circadian rhythm of BMR is at its nadir. Such a measure of BMR therefore underestimates daily BEE in individuals and, if the above theory is correct, particularly in those not...
energetically stressed. Because AEE is typically estimated as DEE minus BEE (and often also minus digestive costs calculated as 10% of DEE), in turn AEE is overestimated (fig. 3A). In energetically stressed individuals, the underestimate in BEE is less, and in turn, the overestimate in AEE is less. Consequently, when assessing the changes in BEE and AEE to energy compensate when activity levels increase, if those increased activity levels cause energy stress that was not previously present, then if BMR does not change but rather its circadian fluctuations attenuate, BEE is mistakenly observed to increase and, in turn, AEE decrease (fig. 3B). Alternatively, if the attenuation of BMR is asymmetrical, involving reduced highest daily values but no changes in lowest values, then true BEE is decreased (fig. 3C). Because of the attenuation, BMR measured at the nadir underestimates BEE by less than is the case in individuals not energetically stressed (fig. 3A), and thus any real decrease in BEE will be underestimated, while any decrease in AEE will be overestimated. Thus, potentially, BEE plays a clearer, greater role in energy compensation than is evidenced by many studies, while NEAT or other aspects of AEE play a smaller role. Measuring BMR closer to midnight or multiple times during the day will somewhat alleviate this putative problem (fig. 3).

Changing Mitochondrial Efficiency

The classical approaches to measuring energy expenditure involve recording respiratory gas exchange of the entire organism; however, this represents a derivation of respiration at the subcellular level (Koch et al. 2021), where the relationship between oxygen consumption and energy can vary. Mitochondria are essential organelles, generating the majority of energy required for cellular and physiological processes. This energy is provided in the form of ATP generated through the consumption of O$_2$ in a complex process termed “oxidative phosphorylation” (Koch et al. 2021). The ability of mitochondria to generate ATP, in terms of the number of ATP molecules generated for each O$_2$ atom consumed, is known as the phosphate-to-oxygen (P-O) ratio and is a function of mitochondrial efficiency (mitochondrial efficiency is affected by the metabolic substrate being oxidized—efficiency is 15% greater when utilizing fat compared with carbohydrates; Welch et al. 2007). Moreover, not all O$_2$ consumption is coupled to mitochondrial ATP production, and thus some of the available free energy is subjected to proton leakage and lost as heat. This proton leakage presents a significant source of uncoupling and has been estimated to account for 20%–25% of the in vivo BMR (Rolle and Brand 1996). Scenarios where mitochondria exhibit higher efficiency of converting metabolic substrates into energy are met with decreased proton leakage and a lower metabolic rate (Murphy 2009; Stier et al. 2014). In contrast, a lower efficiency in the conversion of metabolic substrates to energy leads to higher proton leakage, heat generation, and increases in metabolic rate (Salin et al. 2015). Importantly, mitochondrial efficiency can vary, both within and between individuals (Salin et al. 2015) and under different conditions. Thus, variability in mitochondrial efficiency can be a significant factor contributing to alterations in BEE (Larsen et al. 2011) and other elements of energy expenditure. Of particular significance here are the observations that both humans and animals show a mitochondrial plasticity to physical exercise (Porter et al. 2015; Stier et al. 2019). In humans, both endurance and resistance exercise promote increased mitochondrial performance (Fernström et al. 2004; Porter et al. 2015), and there is tentative evidence that older humans with a more sedentary lifestyle have a reduced walking capacity and speed relating to a lower mitochondrial efficiency (Coen et al. 2013; Distefano et al. 2018). Changes in mitochondrial efficiency affect the energy

![Figure 3. Calculations of basal energy expenditure (BEE; typically the total energy expended on basal processes over 24 h) and thus activity energy expenditure (AEE) depend on the magnitude of the circadian rhythm of basal metabolic rate (BMR) and the point in the circadian phase when it is measured. A. Fluctuations in BMR are substantial in individuals that are not energetically stressed such that measured BMR can be different depending on the time of day it is recorded. If recorded at a midpoint, perhaps 1:00 a.m. (upper dashed line), the measurement probably accurately estimates true BEE (solid line). If recorded at the nadir, which might typically be around 5:00 a.m. (lower dashed line; when measures are traditionally taken), the BMR measurement underestimates BEE, and in turn, AEE is overestimated (it is calculated to be not only the white but also the light gray section of the stack). B, BMR fluctuations may be attenuated in energetically stressed individuals such that BMR measured at 5:00 a.m. is less of an underestimate of BEE than is the case in A, and therefore the estimate of AEE is less of an overestimate. Note that BEE is the same as in A. C, BMR fluctuations may be attenuated asymmetrically in that the peaks but not the troughs are reduced. Consequently, true BEE is lower (solid line) than in A and B. Similarly to B, if BMR is measured at the nadir (around 5:00 a.m.), then the degree of underestimation of BEE shown in A will not occur. The data presented in A reflect laboratory measurements reported in Zitting et al. (2018, fig. 2 therein); those in B and C are hypothetical. A color version of this figure is available online.](image-url)
expended on any or all energetic processes of the body (Salin et al. 2015) and hence could be the process underlying energy compensation in response to increased activity levels.

Conclusions

The assumption that the more activity is undertaken, the more calories will have been burned by the end of the day is formalized in the additive model of energy expenditure, which has been applied to both humans and other animals (Halsey et al. 2019), and underscores models of public health (FAO/WHO/UNU 2001; Pontzer 2015). However, clear evidence of energy compensation at the organismal level by humans and other animals has now accumulated, and it shows that energy compensation can be considerable, up to 600 kcal/d in humans (Thurber et al. 2019). Despite this, there are many inconsistencies and contradictions across the literature, meaning that we are not in a position to state with any certainty the predominant processes involved in this compensation.

Most experimental studies of humans indicate that any decreases in BMR in response to increases in AEE are at best only slight; however, a recent large-sample field study suggests that in older individuals, decreases in BMR explain most of the observed energy compensation (Careau et al. 2021). NEAT may decrease, but it may simply be a minor factor resulting from fatigue due to the heightened physical activity, or instead, it could be the main process explaining energy compensation. In young or gestating individuals, the additional energy expenditures of growth and reproduction might be reduced (Urlacher et al. 2019). Economical energy expenditure during activity, through reduced extraneous limb movement (Fernández-Verdejo et al. 2021), more efficient biomechanics, or utilizing fat rather than sugar as the metabolic substrate, might possibly play a role in energy compensation in certain situations (Amati et al. 2008; Burgess and Lambert 2010; Halsey et al. 2017; Lark et al. 2018), although with the caveats that (i) biomechanical efficiency could at best explain only a fraction of substantial energy compensation and (ii) changes in metabolic substrate have only a fairly small effect on measures of metabolic rate and do not in themselves reflect changes in ATP use (Salin et al. 2015; fig. 4A, 4B). For species outside their thermoneutral zone, thermoregulatory costs might be minimized because of the heat produced as a by-product of increased activity (Even and Blais 2016; O’Neal et al. 2017). Even decreases in the costs of digestion and assimilation—the thermal effect of feeding—might be part of the compensation strategy. Although digestion costs are unlikely to be a predominant factor (Morio et al. 1998) because they appear resistant to adaptation (Ocobock 2020), they can vary substantially depending on macronutrient content and thus could be affected by a change in diet (Westerterp 2004). Finally, another possible mechanism serving compensation concerns stress. Given that stress responses are attenuated in people who regularly exercise (Silverman and Deuster 2014), if indeed cortisol and epinephrine release in response to stress increases metabolic rate (Holland-Fischer et al. 2009; Hollstein et al. 2020), then increased activity energy costs may be compensated by decreased stress energy costs.

Another possibility is that rather than energy compensation resulting from the downregulation of one or more supposedly physiologically discrete processes, ranging from basal costs to physical activity, it results from the adjustment of one fundamental driver that underpins the endogenous energy expenditure of all of these processes—mitochondrial P-O efficiency and levels of proton leakage. In principle, basal processes, NEAT, and so forth can all be fully maintained, and yet the substrate energy used for these processes decreased by increasing P-O efficiency (Salin et al. 2015; fig. 4). Thus, ATP use is maintained while metabolic rate decreases. Moreover, rather than assuming explicitly or implicitly that decreases in metabolic processes such as BMR and NEAT are driven by top-down mechanisms enacted by some form of control center—a notion that I would argue is often implied in the literature, including in my own offerings (Hamibly and Speakman 2005; Halsey 2018; Thurber et al. 2019; Pontzer 2021)—we should consider viewing processes of energy compensation in terms of collaboration and competition between cells for finite energy resources (Archer et al. 2018a, 2018b). With this perspective, for example, a putative decrease in BMR is caused by a shift in the competition for energy between

![Figure 4. Comparisons of measurements of (mitochondrial) oxygen consumption between states (such as different individuals or different behavioral states within an individual) do not provide accurate comparisons of energy expenditure (quantified as ATP production) if mitochondrial efficiency differs between those states. For states A and B, if mitochondrial efficiency is higher in state B than in state A, then energy use as ATP production can be the same while oxygen consumption decreases. For states C and D, oxygen consumption is the same, and yet mitochondrial efficiency is lower in state D such that energy expenditure (ATP production) is also lower. ROS = reactive oxygen species; leak = proton leak. Adapted from Koch et al. (2021, fig. 3 therein).](image-url)
cells. For instance, and as discussed earlier with regard to the Thurber et al. (2019) case study, at least during activity, skeletal myocytes may outcompete other tissues in the acquisition and storage of consumed energy, and in turn, other tissues have fewer metabolic substrates to use, resulting in their downregulation (E. Archer, personal communication). Adjusting energy consumption and thus energy availability may be a way to interrogate this proposition.

Alongside understanding how energy compensation is achieved, there are many other important and interesting questions to investigate (Melanson et al. 2013), including whether and how the ceiling to DEE at a given time varies with factors such as body mass and age, how activity type and intensity influence the degree and nature of energy compensation (Riou et al. 2015), and whether energy compensation is more pronounced when fedget less, some people may show lower fluctuations in their BMR, and some people may exhibit increased mitochondrial efficiency, while others may not respond at all.

**Acknowledgments**

Thank you to Jeff Yap for his thoughts on measures of mitochondrial efficiency, Karine Salin for her thoughts on mitochondrial efficiency and also her ideas that form figure 4, Herman Pontzer for discussion about visualizing the concept of a flattened basal metabolic rate through the day, and Edward Archer for discussion about how to conceive changing energy usage by the body. Jon Green gave detailed feedback on the manuscript.

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