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Total daily energy expenditure has declined over the last 3 decades due to declining basal expenditure not reduced activity expenditure.

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Abstract

Obesity is caused by prolonged positive energy balance[1,2]. Whether reduced energy expenditure stemming from reduced activity levels contributes, is debated[3,4]. Here we used the IAEA DLW database on energy expenditure of adults in the USA and Europe (n = 4799) to explore patterns in total (TEE: n=4799), basal (BEE: n = 1432) and physical activity energy expenditure (AEE: n = 1432) over time. In both sexes total energy expenditure (TEE) adjusted for body composition and age declined with time, while adjusted AEE increased over time. In males adjusted BEE decreased significantly, but in females this didn't reach significance. A larger dataset of basal metabolic rate (BMR equivalent to BEE) measurements of 9912 adults across 163 studies spanning 100 years replicated the decline in BEE in both sexes. Increasing obesity in the USA/Europe has probably not been fueled by reduced physical activity leading to lowered TEE. We identify here decline in adjusted BEE, as a previously unrecognized novel factor.

Obesity is a global health threat[5]. Although excess body fat is a result of prolonged positive energy balance[1,2], the exact causes of this imbalance remain elusive. Two major potential factors have been suggested. First, food consumption (net energy consumption accounting for losses in feces) may have increased[2]. Alternatively, declines in energy expenditure, due to reduced work-time physical activity[4], combined with increases in sedentary behavior, partly linked to elevated 'screen time' (TV, computer and phone use)[6,7] may be a key driver. These may be linked in a vicious cycle[8], where low activity leads to weight gain, which inhibits activity, leading to further weight gain.

%see supplementary materials

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JRS, KRW and LH processed and analysed the IAEA data, JMAdJ, JLK, and MCR collected, processed and analysed the mouse data, SS, SG, JRS and AK collected and analysed the retrospective BMR data from the literature. JRS, YY, HS, PNA, LFA, LJA, LA, IB, KBA, EEB, SB, AGB, CVCB, PB, MSB, NFB, SGJAC, GLC, JAC, RC, SKD, LRD, UE, SE, TF, BWF, AHG, MG, CH, AEH, MBH, SH, NJ, AMJ, PK, KPK, MK, WEK, RFK, EVL, AML, WRL, NL, CKM, ACM, EPM, JCM, JPM, MLN, TAN, RMO, HP, KHP, YPP, JPR, GP, RLP, RAR, SBR, DAR, ER, LMR, RMR, JR, SBR, MR, DAS, AJS, AMS, ES, SSU, GV, LMvE, EAvM, JCKW, GW, BMW, WWW, JAY, TY, XYZ contributed data to the database. JRS, YY, HS, SS, AJMM, CU, AHL, HP, JR, DAS and WWW created, curated and administered the database.

Conflict of interest

The authors have no conflicts of interest to declare.

Although there is direct evidence, that physical activity levels have declined and sedentary time has increased[4,6,7,8], these changes do not necessarily translate into alterations in total energy expenditure (TEE). As individuals get larger the energy cost of movement also increases[9]. Thus, the same amount of energy may be utilized even though the actual time spent active has declined. Moreover, increases in one type of activity/behavior may be replaced by decreases in another behavior of equal cost. Consequently, apparently large behavior changes may result in only minor alterations in expenditure. Finally, it has been suggested that we may compensate for changes in physical activity by adjusting expenditure on other physiological tasks[10,11]. Although low TEE is repeatable, and having low TEE is not a risk factor for future weight gain over short timescales[12], this does not negate a possible impact over longer periods. In the present paper we address the idea that reduced physical activity leading to reduced activity energy expenditure (AEE) may have fueled the epidemic.

The doubly-labelled water (DLW) method is a validated isotope based methodology for the measurement of free-living energy demands[13]. A previous analysis using this method suggested there had been no change in TEE between 1986 and 2005, calling into question the reduced physical activity hypothesis[14]. Nevertheless, these observations were based on a limited sample (n = 314) from a single European city over a restricted timespan of about 20 years. Here we expanded this analysis using data for 4799 adults living across Europe and the USA drawn from the IAEA DLW database[15] for which we also had BEE measures in 1429 individuals. All estimates of TEE were recalculated using a common equation[16] that has been shown to perform best in validation studies[16].

We split the data by sex, because this may affect the etiology of energy balance[17,18]. This resulted in 1672 measurements of males and 3127 measurements of females. In addition, for 632 of the males and 800 of the females we also had measurements of basal energy expenditure (BEE) from which we derived activity energy expenditure (AEE) and physical activity level (PAL) – for calculations see methods. The data span a period of over 30 years with the first measurements in late 1981 and the latest measurements made in late 2017, with most data obtained between 1990 and 2017. The distribution of BMI in the sample for both males and females is shown in Supplementary Fig S1. Overall females had higher BMI than males. In the pooled sample the distribution was BMI < 18.5 = 2.3%, BMI 18.5 to 25 = 40.3%, BMI 25 to 30 = 35.1% and BMI >30 = 22.2%. Combined overweight and obesity was 57.3%. In both males and females body weight increased over time (Figure S1) reflecting the secular trend in body weight over the same interval.

We first explored the changes in the unadjusted levels of TEE, BEE and AEE over time (Supplementary Fig S2: Table 1). In males there was no significant relationship between TEE and the date of measurement (date coded as months since Jan 1982) ($r^2 = 0.0015$, $p = 0.14$ (ns): Fig S2a) the least squares regression fit gave a gradient of +1.5 kJ/month (95%CI = ±2.06 kJ/month). This gradient leads to an estimated change in average TEE over 30 years of + 0.55 MJ/day (95%CI = ±0.727 MJ/day). Contrasting the lack of significant change in TEE, there was a significant decline in BEE over time (Fig S2b) ($r^2 = 0.029$, $p = 0.000018$). The gradient of decline (3.3 kJ/month, 95%CI = ±1.4 kJ/month) was equivalent to an average fall in BEE by 1.19 MJ (9.7%) over 30 years (95%CI = ±0.54 MJ/day). As might

be anticipated since TEE*0.9 = BEE + AEE, the absence of a change in TEE and declining BEE was reflected by an increase in AEE over time, but this did not reach significance (Fig S2c) ($r^2 = 0.003$, $p = 0.16$). The gradient of the change in AEE (1.4 kJ/month, 95%CI = ±1.8 kJ/month) was equivalent over 30 years to an increase by 0.50 MJ/day (95%CI = ±0.69 MJ/day). In females, unadjusted levels of TEE, BEE and AEE did not change significantly over time (supplementary Fig S3, Table 1).

All the energy expenditure variables (TEE, BEE and AEE) in both sexes were dependent on body mass (BM) and BMI (illustrated for BMI in supplementary Fig 4). Because of these relationships it is necessary to adjust the raw expenditure data over time (Figs S2 and S3) to account for any changes in body composition over time that might generate a biased estimate of the change in expenditure variables. We adjusted the levels of log transformed TEE, BEE and AEE for body size and composition using residuals from general linear models with $\log_e$ fat-free mass, $\log_e$ fat mass and age as predictors. In this analysis the data were logged because the relationships between energy expenditure components and body composition follow power law relationships. In males, adjusted TEE significantly declined over the measurement period (Fig 2a: $r^2 = 0.0103$, $p < .0001$). The gradient of the fitted regression was −32.5 kJ/month (95%CI = ±1.20 kJ/month) leading to an estimated average change over 30 years of −0.93 MJ/day in adjusted TEE (95%CI = ±0.465 MJ/day), a decline on average of 7.7%. The adjusted BEE showed a highly significant decline over time (Fig 2b: $r^2 = 0.064$, $p < 10^{-9}$) with the gradient of 2.67 kJ/month (95%CI = ±0.82 kJ/month) being equivalent to an average fall in BEE of 0.96 MJ/day (14.7%) over 30 years (95%CI = ±0.15 MJ/day). In contrast, the adjusted AEE increased over time (Fig 2c: $r^2 = 0.0221$, $p < .0003$). The gradient of +2.8 kJ/month (95%CI = ±1.4 kJ/month) was equivalent to a rise of 1.01 MJ/day over 30 years (95%CI = ±0.53 MJ/day).

In females as well, there was a significant decline in the adjusted TEE over time (Fig 3a: $r^2 = 0.006$, $p < .00002$). The gradient of the effect 1.42 kJ/month was equivalent to a reduction in TEE over 30 years of 0.51 MJ (95%CI = ±0.22 MJ/day) or 5.6%. This decline was paralleled by a reduction in adjusted BEE of 2.0% but this did not reach significance (Fig 3b: $r^2 = 0.0015$, $p > .05$). The gradient of the fall in adjusted BEE was 0.3 kJ/month, equivalent to a reduction in adjusted BEE over 30 years of 0.11 MJ/day (95%CI = ±0.21 MJ/day). In contrast, and again similarly to the males, adjusted AEE significantly increased over time (Fig 3c: $r^2 = 0.0063$, $p = 0.026$). The gradient of increase in AEE of 1.16 kJ/month was equivalent to an increase in AEE of 0.42 MJ/day over 30 years (95% CI = ± 0.37 MJ/day).

Because there was a small sample of measures in the early 1980s in males these may have exerted undue leverage in the regression models. We therefore repeated the analysis excluding these data. Their removal had no impact on the detected relationships (Supplementary Table S1). Since individual studies may also exert undue leverage we performed additional sensitivity analyses on the BEE effect (post 1987) where the data for each study was systematically removed and the regression recalculated. In males removal of no individual study resulted in the loss of significance (Supplementary Table S2). In females however, the absence of significance was due to inclusion of data from a single study (Supplementary Table S3). We have no reason to exclude these data, but their undue

influence may explain the anomalous lack of decline in female BEE when TEE is declining and AEE is rising (Table 1 and fig 2).

Hence, in both males and females there was a decline in the adjusted TEE by 7.7 and 5.6% respectively and in males in the adjusted BEE over time by 14.7% over 30 years (females declined by 2% which was not significant). In both sexes the confidence limits for the decline in adjusted TEE overlapped with the confidence limits for the decline in adjusted BEE, suggesting the decline in adjusted BEE could be sufficient to explain the reduction in adjusted TEE. In both sexes there was in contrast a significant increase over time in adjusted AEE. The comparable declines in adjusted TEE and BEE resulted in a significant increase in PAL (=TEE/BEE) in males (Males supplementary Fig S5a: $r^2 = 0.0215$, $p < .0003$) but in females the change in PAL over time was not significant (females supplementary Fig S5b: $r^2 = 0.0037$, $p = 0.085$).

To replicate and check our observation of decreasing BEE over time we systematically reviewed data from the literature on mean BMR over the last 100 years, restricted to studies in the USA and Europe, to match the restricted regions included in the time course from the IAEA database (Figs 1,2 and Table 1). For the distinction between BEE and BMR see the methods. The main effect on Log_e BMR was Log_e BM (Fig 3a), with additional effects of sex and age (total $r^2 = 0.88$). Including the date of measurement, sex, age and log_e body mass as predictors in a weighted regression analysis there was a significant negative effect of date of measurement ($R^2 = 0.024$, $p = 0.022$) on the adjusted log_e BMR (Fig 3b). On average, BMR adjusted for BM, age and sex has declined by about 0.34 MJ/d over the last 100 years. This decline is consistent with, but at a lower rate, than the data from the IAEA database reported above (Table 1).

Basal metabolism may be influenced by many factors one of which is diet. Human dietary changes during the epidemic have included many things such as changes in the amounts of fiber and fat, and the types of fat consumed. Because evaluating the impacts of long-term diets on human metabolism is difficult, we explored the potential impact of dietary fatty acids on metabolic rate using the mouse as a model. Working with mice has the advantage that diets can be rigorously controlled and maintained constant over protracted periods. We exposed adult male C57BL/6 mice to 12 diets (for details see supplementary Tables S2 and S3) that varied in their fatty acid composition for 4 weeks (equivalent to 3.5 years in a human). Mouse BMR (kJ/d) was strongly related to body weight (regression $r^2 = 0.512$, $p = 3\times10^{-11}$: Fig 4A). We included the total intake of different fatty acids (SAT: saturated fatty acids, MUFA: mono-unsaturated fatty acids and PUFA: poly-unsaturated fatty acids) with body weight into a general linear model. Only intake of saturated fatty acids was significant (SAT: $F = 11.05$, $p = 0.002$ (Fig 4B); MUFA: $F = 1.38$, $p = 0.245$; PUFA: $F = 0.17$, $p = 0.686$) with higher levels of SAT linked to higher energy expenditure (Fig 4B).

Overall the data we present do not support the idea that lowered physical activity in general, leading to lowered energy expenditure, has contributed to the obesity epidemic during the last 30 years. Unadjusted AEE was higher in individuals with greater BMI (supplementary Fig S4). This is because, as shown previously, despite on average moving less, individuals with greater BMI have higher costs of movement[9]. Rather than adjusted AEE declining,

it has significantly increased overtime in both sexes. Yet TEE (adjusted for age and body composition) has declined significantly in both males and females over the past 3 decades. Because adjusted AEE has increased at the same time that TEE has declined there has been a corresponding reduction in adjusted BEE (which only reached significance in males). The observation that adjusted AEE (and PAL in males) has significantly increased over time is counter intuitive given the patterns established in worktime physical activity and the suggested progressive increase in sedentary behavior[4,6–8]. One possibility is that lowered work time physical activity may have been more than offset by increased engagement in leisure time physical activity. For example, sales of home gym equipment in the USA increased from 2.4 to 3.7 Bn US$ between 1994 and 2017[19]. Time spent in leisure time PA in the USA also increased between 1965 and 1995,[20] suggesting leisure time PA has replaced the decline in worktime PA levels[20]. Leisure time PA has also changed in other westernized populaions[21]. Although time spent on computers has increased, much of the increase in this time has largely come at the expense of time spent watching TV. Since these activities have roughly equivalent energy costs[22] this change would not be apparent as a decline in overall adjusted AEE.

The reduction in adjusted BEE is less easily understood but is consistent with the recent observation that body temperatures have also declined over time[23], over the same interval as the reduction of BMR in the wider data set we analysed (Fig 3b). The magnitude of secular change in BMR is consistent with studies measuring BMR and body temperature in several contexts, including calorie restriction, ovulation, and fever which show a 10–25% increase in BMR per 1ºC increase in core temperature[24,25]. It was recently suggested that changes in both activity and basal metabolism may have contributed to the decline in body temperature (T_b)[26], but our data suggest this is probably dominated by a BMR effect. The reduction in T_b has been speculated to be a consequence of a reduction in baseline immune function because we have greatly reduced our exposure to many pathogens. However, the links between immune function and metabolism are not straightforward. For example, artificial selection on metabolic rate leads to suppressed innate but not adaptive immune function[27], and studies of birds point to no consistent relation between immune function and metabolism either within or between subjects[28]. Experimental removal of parasites in Cape ground squirrels (*Xerus inauris*) led to elevated rather than reduced resting metabolic rate[29]. Nevertheless, some studies in forager-horticulturalist societies in South America have noted elevated BMR is linked to increased levels of circulating IgG[30] and cytokines[31], supporting the view that a long term decline in BEE may be mediated by reduced immune function. Whether this has any relevance to changes in the USA/Europe in the past 30 years is unclear. It is also possible that the long-term reduction in BMR represents methodological artefacts. In the early years, measurements of BMR were often made using mouthpieces to collect respiratory gases, and recently such devices have been shown to elevate BMR by around 6%[32]. A second possibility is that early measurements paid less attention to controlling ambient temperature to ensure individuals were at thermoneutral temperatures[33].

During the past century there have been enormous changes in the diets of US and European populations (USDA and FAO food supply data)[34]. These changes have included alterations in the intake of carbohydrates, fiber and fats, with % protein intake remaining relatively constant[34]. While intake of carbohydrates peaked in the late 1990s the intake of fat has

increased almost linearly since the early part of the 1900s. Moreover, the fat composition has changed dramatically with large increases in soybean oil and seed oils from the 1930s onwards (dominated by the polyunsaturated 18:2 linoleic acid and other PUFAs) and reductions in animal fats (butter and lard) (dominated by saturated fatty acids palmitic (16:0) and stearic acid (18:0) and the mono-unsaturated oleic acid (18:1))[34]. The change has been dramatic, as animal fats comprised >90% of the fatty acid intake in 1910 but are currently less than 15%. Because linoleic acid is desaturated to form arachidonic acid (ARA) and ARA is linked to endocannabinoids it has been speculated that expanding linoleic acid in the diet may be linked to various metabolic issues. Effects on basal metabolic rate however are disputed, and if anything, PUFAs lead to elevated not reduced metabolism[35,36], although many studies suggest no effect[37,38]. This variation in outcome may reflect difficulties in controlling human diet over protracted periods necessary to generate robust changes in metabolism. In mice, where we can rigorously control the diet for prolonged periods (equivalent to many years of human life), we have shown here no effect of PUFAs on metabolic rate, but a clear impact of saturated fat, with greater intake of saturated fat leading to higher metabolic rate (adjusted for body mass). This finding is consistent with earlier reports of relationships between membrane lipids and elevated metabolic rate in mice, particularly a positive effect of palmitic and stearic acids[39,40]. This suggests that alterations in the intake of saturated relative to unsaturated fat over the last 100 years may have contributed to the decline in BEE reported here, although clearly we should be cautious about extrapolations from males of a single inbred mouse strain and further studies in humans are required. Moreover, other aspects of the diet that impact metabolic rate may also have changed over time, for example intake of fiber which has declined in recent years[41] and has been shown in a randomized controlled trial to affect resting metabolic rate[42].

Strengths and limitations

A strength of this study is the large sample of individuals over a restricted geographical area (US and Europe) measured using a complex methodology. This has allowed us to detect a small but nevertheless biologically meaningful signal. However, it is important to be aware that the studies were not designed with the current analysis in mind. Hence while we have adjusted for differences in age and body composition there may be other factors that differed over time that we did not adjust for and that could explain the trends we found. Further, the participants recruited at different time points may not have been representative of the underlying populations, even though the overall distribution seems representative (Fig S1). The data are cross-sectional which limits the inferences that can be made regarding causality in the associations. Finally, while we have speculated on some potential factors that might have contributed to the reduction in BEE (i.e. immune function and diet), these factors were not quantified in most of the participants who had their TEE measured. The mouse work we performed showing potential links of diet to metabolism was only conducted in males of one strain and a single age and may not be more broadly applicable. These potential mechanisms therefore remain speculations until more direct data can be collected.

Conclusion

Overall our data show that there has been a significant reduction in adjusted TEE over the last three decades, which can be traced to a decline in BEE rather than any reduction in AEE linked to declining physical activity levels. Indeed, our data show that AEE has significantly increased over time. Reductions in BEE extend much further back in time (TEE data do not extend further back than 1981 as that was the first year the DLW technique was applied to humans), and mouse data indicated that one of many possible explanations is decreases in the intake of saturated relative to unsaturated fatty acids. If the decline in BEE over time has not been compensated for by a parallel reduction in net energy intake then the energy surplus resulting would be deposited as fat. This study therefore identifies a novel potential contributor to the obesity epidemic, that has not been previously recognized: a decline in adjusted BEE linked to reduction in overall adjusted TEE. Further understanding the determinants of BEE and the cause of this decline over time, and if it can be reversed, are important future goals.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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Data Availability

With respect to the IAEA database and the meta-analysis of BMR data this work comprises a secondary analysis of data that are mostly already published and available in the primary literature. These data have been compiled into a database, access to which is free. Forms for requesting data can be found at www.dlwdatabase.org and should be directed to the lead corresponding author j.speakman@abdn.ac.uk or Dr Alexia Alford at the (a.alford@iaea.org). The BMR data are available upon request to co-corresponding author Dr Anura Kurpad (a.kurpad@sjri.res.in). The mouse data described in the paper are available upon request to co-corresponding author Dr Matthew Rodeheffer (matthew.rodeheffer@yale.edu).

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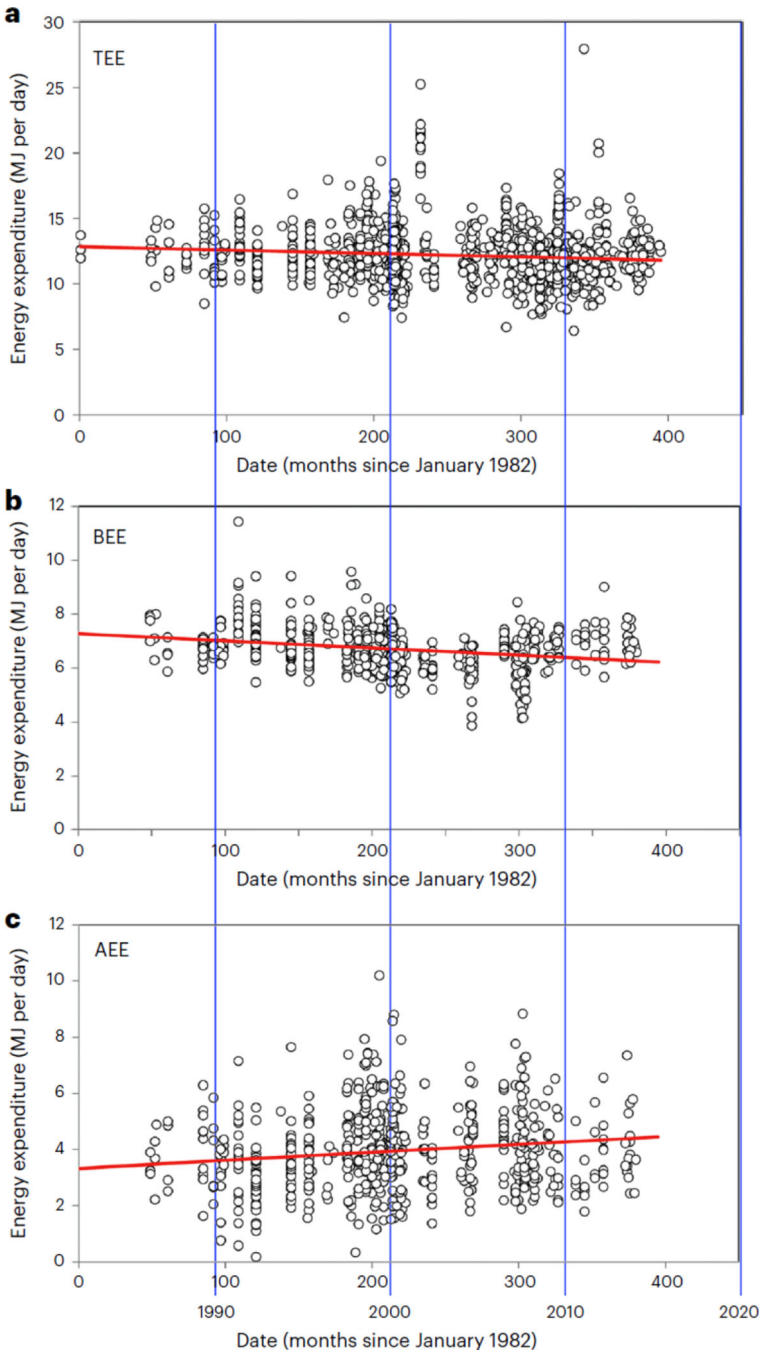

Figure 1:

Trends over time in a) adjusted total energy expenditure, b) adjusted basal energy expenditure, and c) adjusted activity energy expenditure for males. Adjustments were made for body composition (fat and fat-free mass or body mass, and age) – see methods for details. All expenditures are in MJ/d and time is expressed in months since January 1982. Significant years are also indicated. Each data point is a different measurement of expenditure. The red lines are the fitted least squares regression fits. For regression details refer to text and Table 1.

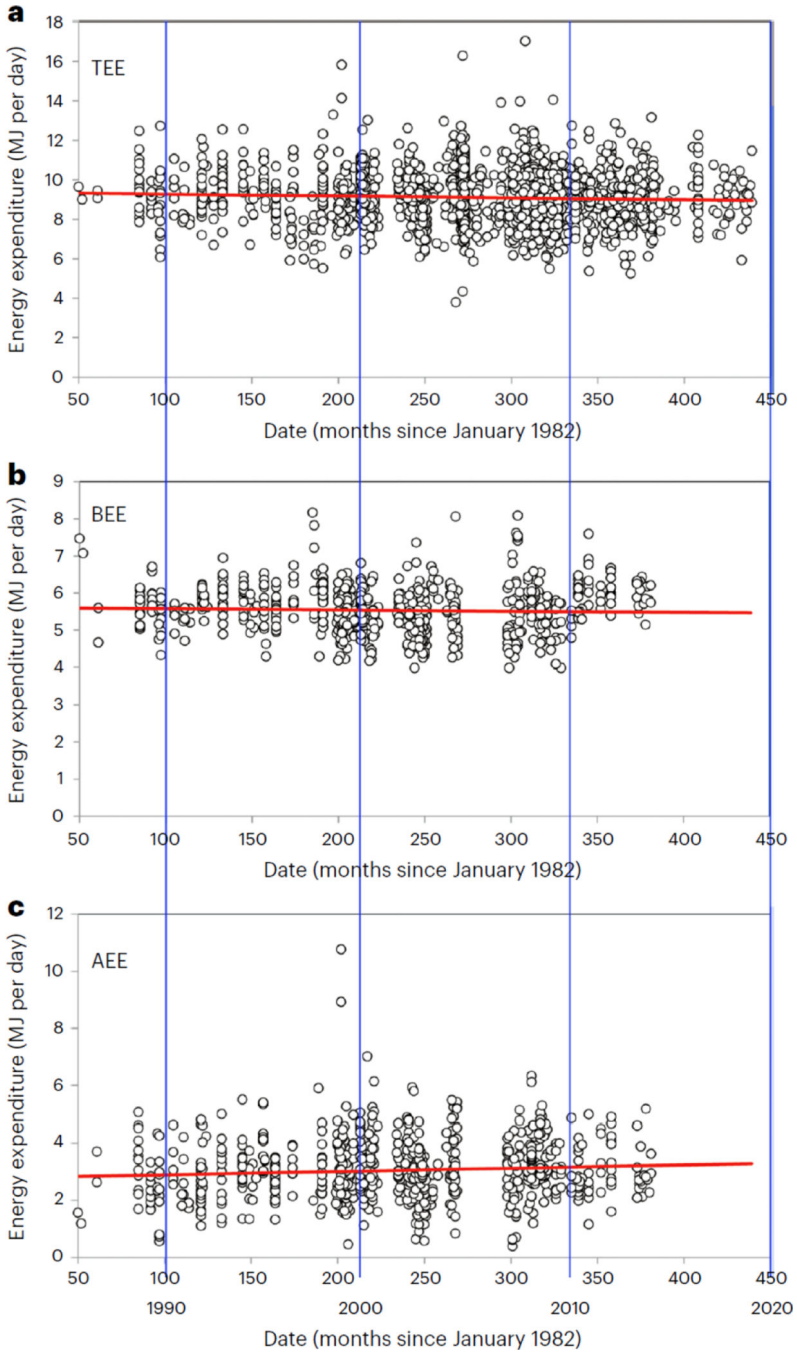

Figure 2:

Trends over time in a) adjusted total energy expenditure, b) adjusted basal energy expenditure, and c) adjusted activity energy expenditure for females. Adjustments were made for body composition (fat and lean mass and age) – see methods for details. Significant years are also indicated. All expenditures are in MJ/d and time is expressed in months since January 1982. Each data point is a different individual measurement of expenditure. The red lines are the fitted least squares regression fits. For regression details refer to text and Table 1.

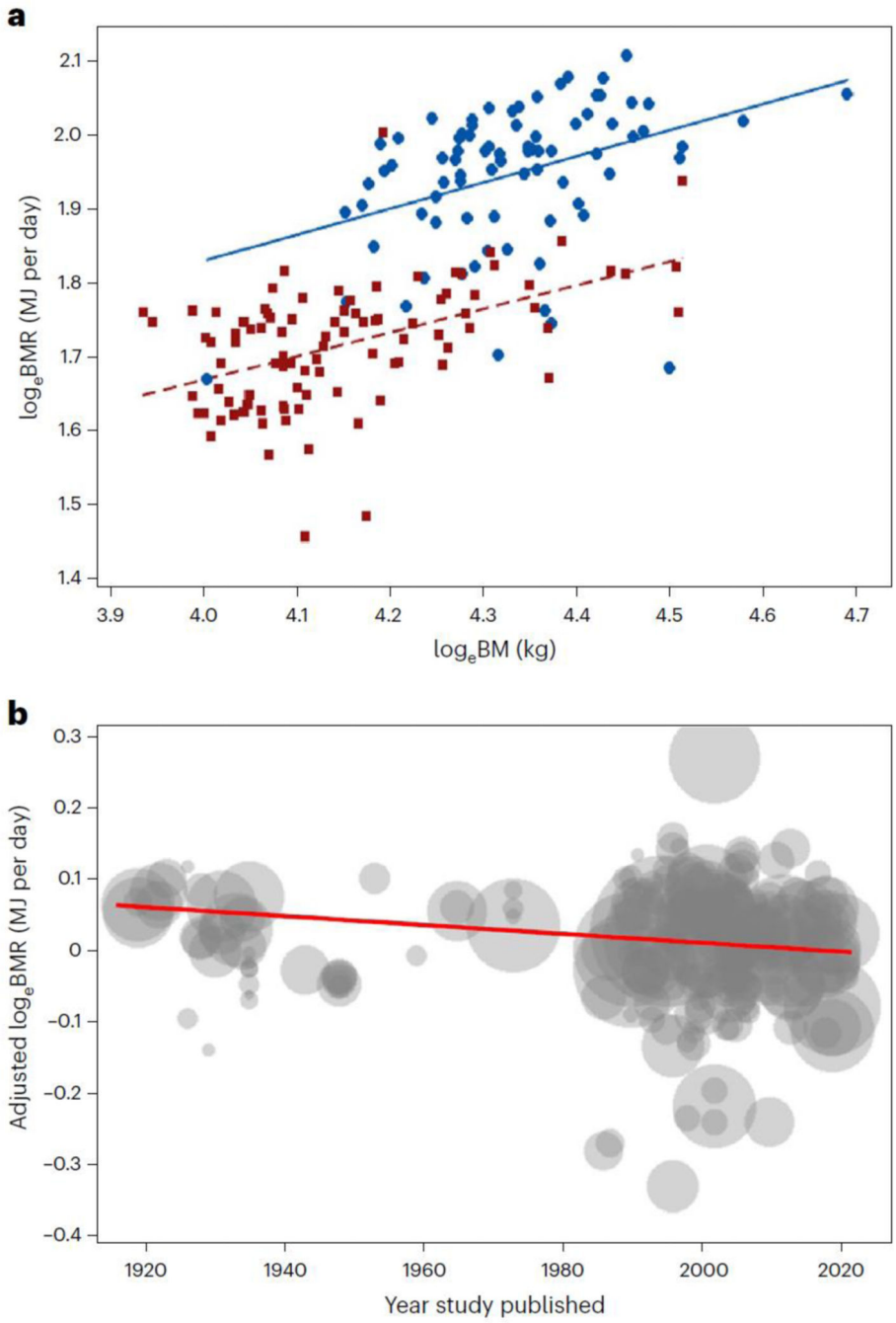

Figure 3:

A: effect of $\log_e$ body mass on the $\log_e$ basal metabolic rate (BMR) in a systematic review of 165 studies dating back to the early 1900s (first study 1919). Data for males in blue and for females in red. Studies with mixed male and female data not illustrated. B: Bubble plot showing the Residual $\log_e$ Basal metabolism derived from a weighted regression of $\log_e$ BMR against sex, age and $\log_e$ (body mass) plotted against date of measurement in the same 165 studies. Bubbles represent the sample size of the studies. The red line is the fitted weighted regression.

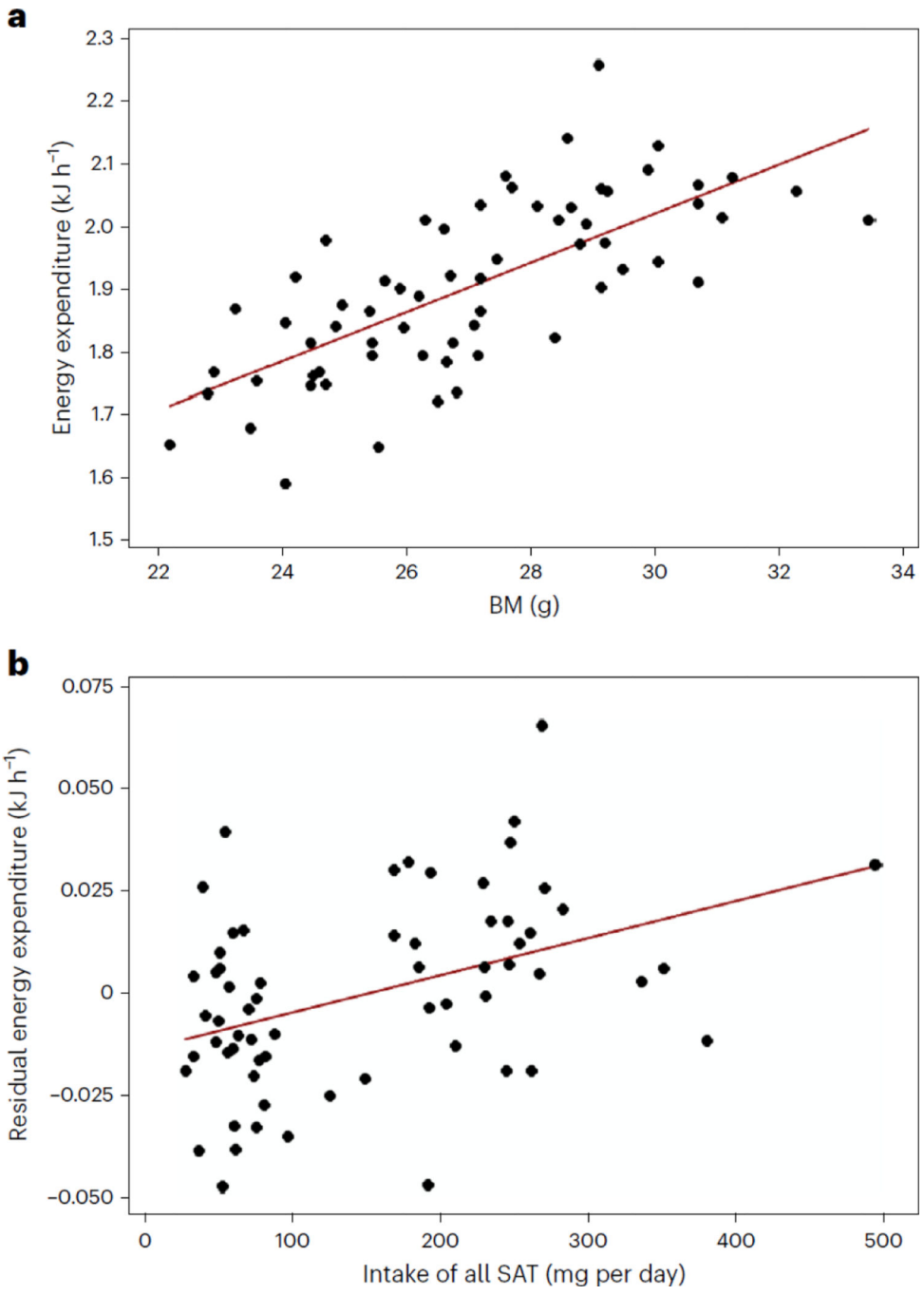

Figure 4:

A: the relationship between body weight and metabolic rate in the mice fed different diets with variable fatty acid compositions. B: the effect of saturated fatty acid intake on residual metabolic rate – corrected for body weight.

Table one:

Patterns of change in components of energy expenditure in males and females since the early 1990s. Data are shown unadjusted and adjusted for body composition and age. The gradient of the fitted relationships with time are translated to the overall change in expenditure (MJ) over 30 years with the 95% confidence intervals (95%CI) for this change. TEE = total energy expenditure, BEE = basal energy expenditure, AEE = activity energy expenditure (=0.9TEE-BEE). Significance of the relationships is also shown. p > .01 was considered not significant (ns).

Males			
Unadjusted data			
Variable	**Mean change over 30 y (MJ/d)**	**95% CI (± MJ/d)**	**Significance**
TEE	+0.55	0.73	ns
BEE	−1.19	0.536	p < .00002
AEE	+0.50	0.695	ns
Adjusted data			
TEE	−0.93	0.46	p < .0001
BEE	−0.96	0.15	$p < 10^{-9}$
AEE	+1.01	0.53	p < .0003
Females			
Unadjusted data			
Variable	**Mean change over 30 y (MJ/d)**	**95% CI**	**Significance**
TEE	−0.16	0.360	ns
BEE	−0.32	0.352	ns
AEE	+0.18	0.452	ns
Adjusted data			
TEE	−0.51	0.26	p < .00002
BEE	−0.12	0.215	ns
AEE	+0.42	0.367	p = 0.026