Associations among sedentary and active behaviours, body fat and appetite dysregulation: investigating the myth of physical inactivity and obesity

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Associations amongst sedentary and active behaviours, body fat and appetite dysregulation: investigating the myth of physical inactivity and obesity.

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ABSTRACT

Background

There is considerable disagreement about the association between free-living physical activity and sedentary behaviour and obesity. Moreover studies frequently do not include measures that could mediate between physical activity and adiposity. The present study used a validated instrument for continuous tracking of sedentary and active behaviours as part of habitual daily living, together with measures of energy expenditure, body composition and appetite dysregulation. This cross-sectional study tested the relationship between inactivity and obesity.

Methods

Seventy-one participants (81.7% women) aged 37.4 years (±14) with a body mass index (BMI) of 29.9 kg/m^2 (±5.2) were continuously monitored for 6-7days to track free-living physical activity (light 1.5-3METs; moderate 3-6METs; and vigorous >6METs) and sedentary behaviour (<1.5METs) with the SenseWear Armband. Additional measures included body composition, waist circumference, cardiovascular fitness, total and resting energy expenditure, and various health markers. Appetite control was assessed by validated eating behaviour questionnaires.

Results

Sedentary behaviour (11.06±1.72 hours/day) was positively correlated with fat mass (r=0.50, p<0.001) and waist circumference (r=-0.65, p<0.001). Moderate-to-vigorous physical activity was negatively associated with fat mass (r=-0.72, p<0.001) and remained significantly correlated with adiposity after controlling for sedentary behaviour. Activity energy expenditure was positively associated with the level of PA and negatively associated with fat mass. Disinhibition and Binge Eating behaviours were positively associated with fat mass (r=0.58 and 0.47, respectively, p<0.001).

Conclusion

This study demonstrated clear associations among objective measures of physical activity (and sedentary behaviour), energy expenditure, adiposity and appetite control. The data indicate strong links between physical inactivity and obesity. This relationship is likely to be bi-directional.

What are the new findings

- Habitual sedentary time was associated with higher adiposity.
- Moderate-to-vigorous physical activity (MVPA) was associated with lower adiposity.
- The strongest relationship was with MVPA.
- The relationship between physical (in)activity and adiposity is likely to be bidirectional and depends mainly on MVPA.

Impact on clinical practice

- Patients/clients should be encouraged to replace some sedentary and light activity with at least moderate PA such as brisk walking in order to optimise benefits.

BACKGROUND

In recent years the relative contributions of overconsumption of food and the under-expenditure of energy (physical inactivity) to obesity have been vigorously debated. On one side it has been claimed that an increase in food availability (energy flux) was more than sufficient to account for the increase in average body weight of US citizens over a 20 year period.[1] This argument has recently been extended to a global level.[2] In contrast it has been argued that the decline in work-related physical activity (and therefore energy expenditure) over several decades has been sufficient to account for a positive energy balance and the rise in obesity in the US.[3] In general it seems that the excess food notion of obesity is more favourably received than the low activity idea. This view has been promoted by the print media with headlines such as ‘Why exercise makes you fat’.[4] These headlines have appeared despite evidence from controlled trials demonstrating dose related effects of physical activity on weight loss;[5] the more you do (duration or energy expended) the more weight is lost. Additionally, although Cochrane systematic reviews have also reported beneficial effects of exercise on weight loss independent of any dietary effect,[6] the view persists that being active does not contribute to weight control. In a recent editorial commentary in this journal, a headline title referred to ’...the myth of physical inactivity and obesity’ and the text categorically stated that ‘physical activity does not promote weight loss’.[7] Strongly argued articles refuting these claims [8 9] have attempted to prevent further damaging perceptions emanating from these claims.

For over two decades we have investigated the interactions between energy expenditure and energy intake.[10] We have demonstrated in several published studies that a programme of supervised and measured exercise in obese individuals leads to a significant reduction in body fat and a maintenance or increase in lean mass (fat-free mass) in both men and women.[11-13] These studies indicate that physical activity has the capacity to influence body fat in obese people. Recently we have used a sensitive validated wearable device (BodyMedia SenseWear armband (SWA)) to directly measure the amount of time people spend in sedentary behaviour and in light, moderate and vigorous activity.[14] We have quantified the amount of time (and energy expended) in sedentary and active behaviours, and related this to measures of body adiposity and validated traits reflecting dysregulated appetite control. We have used this methodology to directly test the myth of physical inactivity and body fatness (obesity). The study was designed to provide accurate and objective measures of the quantity of sedentary and active behaviours in habitual daily life, and to examine the relationships with measures of adiposity, energy expenditure, fitness and markers of health; and with psychological measures of the loss of control over appetite.

METHODS

Participants

Seventy-one participants (81.7% women) aged 37.4 years (±14) with a body mass index (BMI) of 29.9 kg/m^2 (±5.2) were recruited from the University of Leeds, UK, and surrounding area for this cross-sectional study. Sixty-eight of the 71 participants had valid SWA data (95.8% compliance) and all participants had valid body composition and appetite dysregulation data. Participants were males and females aged >18 years with no contraindications to exercise and not taking medication known to effect metabolism or appetite. All participants provided written informed consent before taking part in the study, and ethical approval was granted by the School of Psychology Ethics Board (14-0091).

Study design

Participants attended the research unit twice over the course of one week. Free-living PA and sedentary behaviour were measured continuously for a minimum of 7 days for >22 hours/day. Participants were fasted for a minimum of 12 hours and had abstained from exercise and alcohol for at least 24 hours before both laboratory visits.

On the morning of day one the following measures were taken: height, weight, waist and hip circumference, body composition and resting metabolism. Health markers including, fasting blood glucose diastolic and systolic blood pressure (BP) and resting heart rate (HR) were taken, along with measures of appetite dysregulation (Three-Factor Eating Questionnaire, Binge Eating Scale). Participants were provided with a PA diary and fitted with a SenseWear Mini Armband (BodyMedia, Inc., Pittsburgh, PA).

Anthropometrics

Height was measured using a stadiometer (Leicester height measure, SECA) and body composition was measured using air plethysmography (Bodpod, Concord, CA). Body weight was obtained from the BodPod whilst participants were wearing minimal clothing. BMI was calculated as weight in kg / height in m^2. Waist circumference was measured horizontally in line with the umbilicus and hip circumference was measured horizontally at the maximum circumference of the hip. Three measures were taken for each and averaged. The same researcher completed all measurements.

Resting metabolic rate and health markers

Resting metabolic rate (RMR) was measured using indirect calorimetry (GEM, NutrEn Technology Ltd, Cheshire, UK). Participants were instructed to remain awake but motionless in a supine position for 40 minutes, with RMR calculated from respiratory data averaged during the last 30 minutes of assessment. BP and resting HR were measured using an automatic sphygmomanometer (Omron) immediately after completion of the RMR procedure. Fasting glucose was obtained from a finger prick blood sample analyzed using a blood glucose analyzer (YSI 2300 STAT PLUS Glucose and Lactate Analyzer).

Appetite dysregulation

Participants completed the Three Factor Eating Questionnaire, a 51 item questionnaire measuring restraint, disinhibition and hunger[15] and the Binge Eating Scale, a 16 item questionnaire measuring binge eating behaviour and cognitions indicative of eating disorders.[16]

Free living PA and EE

Free-living physical activity and sedentary behaviour was measured objectively using the SWA. Participants wore the armband on the posterior surface of their upper non-dominant arm for a minimum of 22 hours per day for 7-8 days (except for the time spent showering, bathing or swimming). This data collection allowed for the calculation of daily averages for each activity category. The SWA measures motion (triaxial accelerometer), galvanic skin response, skin temperature and heat flux. Proprietary algorithms available in the accompanying software calculate energy expenditure (EE) and classify the intensity of activity. Sedentary behaviour was classified as <1.5 METs, light 1.6-2.9 METs, moderate 3-5.9 METs and vigorous >6 METs.[17] Sedentary behaviour and PA variables were calculated as a percentage of total awake time over the wear period of 6-7 days, for example, total sedentary minutes were divided by total awake minutes to give the proportion of awake time spent sedentary over the total wear period. Moderate and vigorous PA was grouped together to form one MVPA category to correspond with the guidelines for PA.[18] The SWA has been shown to accurately estimate time in MVPA and EE at rest and during free-living light and moderate intensity PA.[19-22] For the SWA data to be valid >22 hours of data per day had to be recorded and at least six 24 hour periods (midnight to midnight) including 2 weekend days. Participants completed a physical activity diary to coincide with the PA monitoring period detailing the intensity, duration and type of activity performed along with details on removal of the SWA

Participants returned to the lab on day 7 or 8 to return the activity monitors and completed PA diary. Cardiovascular fitness was also measured.

Maximal aerobic capacity

Maximal aerobic capacity ($\dot{V}O_2max$) was measured during an incremental treadmill test with expired air (Sensormedics Vmax29, Yorba Linda, USA) and heart rate (Polar RS400, Polar, Kempele, Finland) measured continuously. Attainment of true $\dot{V}O_2max$ was determined by a plateau in $\dot{V}O_2$ with an increase in workload, a respiratory quotient (RQ) of >1 and a HR within 20 beats of age predicted maximum HR (220-age).

Statistical analysis

Data are reported as mean ± SD throughout. Statistical analysis was performed using IBM SPSS for Windows (Chicago, Illinois, Version 21). For reasons of scientific rigour and to reduce the likelihood of false positives, we only regarded relationship as meaningful with a p value < 0.01. Characteristics of the study population were summarised using descriptive statistics. Pearson correlations were performed to examine the associations amongst sedentary and active behaviour, body composition and appetite dysregulation. In addition partial correlations were also carried out to separate the effects of a third variable acting concurrently on two variables; this involved controlling for body fat percentage, sedentary behaviour and MVPA in different analyses.

RESULTS

Participant Characteristics

Study sample characteristics are displayed in table 1. Of the 71 participants who took part in the study 68 provided ≥6 days of valid armband data. Average wear time of the armband was 23.55±0.26 hours/day (98±1.2%). Participants were sedentary for an average of 11.06±1.72 hours/day (excluding sleep) and recorded 3.26±1.03 hours/day in light PA and 2.10±1.40 hours/day in MVPA (see figure 1). Participants mean age was 37.35±14.01 and their average total energy expenditure was 2708.07±421.81 kcal/d.

Table 1. Descriptive statistics of study sample

Variable	Mean (SD)	Range
Age (years)*	37.35 (14.01)	18.00 – 72.00
Height (m)*	1.66 (0.09)	1.49 – 1.91
Body mass (kg)*	82.24 (15.26)	44.90 – 113.90

BMI (kg/m^2)*	29.94 (5.24)	19.10 – 39.90
Fat mass (kg)*	31.79 (13.37)	5.00 – 60.40
Lean mass (kg)*	50.44 (9.28)	32.10 – 81.40
Waist circumference (cm)*	100.23 (12.83)	69.00 – 133.70
Systolic blood pressure (mm Hg)*	118.17 (14.12)	87.00 – 162.00
Diastolic blood pressure (mm Hg)*	77.80 (10.25)	61.00 – 77.80
Resting heart rate (bpm)*	58.56 (9.71)	37.00 – 84.00
Blood glucose (mmol/L)**	4.73 (0.69)	1.98 – 6.70
Resting metabolic rate (kcal/d)†	1698.54 (296.86)	1070.90 – 2451.90
Total energy expenditure (kcal/d)^	2708.07 (421.81)	1827.30 – 4256.60
Cardiovascular fitness (ml/kg/min)^	40.99 (7.88)	29.60 – 54.93
SWA wear time (hours/d)^	23.55 (0.26)	22.47 – 23.95
Sedentary behaviour (hours/d)^	11.06 (1.72)	6.01 – 15.40
Light PA (hours/d)^	3.26 (1.03)	1.35 – 6.05
MVPA (hours/d)^	2.10 (1.40)	0.48 – 6.74
Restraint*	8.21 (3.82)	0.00 – 17.00
Disinhibition*	8.85 (3.88)	0.00 – 15.00
Hunger*	6.00 (3.16)	0.00 – 13.00
Binge Eating*	13.23 (7.31)	1.00 – 34.00

SWA, SenseWear armband; MVPA, moderate-to-vigorous physical activity; * n=71; ** n=69; † n=70; ^ n=68

Figure 1 near here

Association between sedentary behaviour and different categories of physical activity

Sedentary behaviour was negatively associated with light (r(66)=-0.39, p=0.001), moderate (r(66)=-0.76, p<0.001) and vigorous (r(66)=-0.44, p<0.001) PA. Light PA was also negatively associated with vigorous PA (r(66)=-0.33, p<0.01). Moderate and vigorous PA were positively correlated (r(66)=0.65, p<0.001).

Associations between sedentary behaviour, physical activity and body composition

Sedentary behaviour was positively correlated with multiple indices of adiposity including body mass (r(66)=0.44, p<0.001), BMI (r(66)=0.50, p<0.001), fat mass (r(66)=0.50, p<0.001) and waist circumference (r(66)=0.45, p<0.001) as shown in Table 2. On the other hand, MVPA was negatively associated with body mass (r(66)=-0.55, p<0.001), BMI (r(66)=-0.71, p<0.001), fat mass (r(66)=-0.72, p<0.001) and waist circumference (r(66)=0.45, p<0.001).

Partial correlations were performed to identify the independent effects of sedentary behaviour (controlled for MVPA), light PA (controlled for MVPA and sedentary behaviour, separately) and MVPA (controlled for sedentary behaviour) on body composition. After controlling for MVPA the magnitude of the correlation between sedentary behaviour and adiposity were markedly weakened. However, when the correlations between MVPA and adiposity were adjusted for sedentary behaviour all correlations remained significant (body mass (r(65)=-0.38, p=0.001), BMI (r(65)=-0.57, p<0.001) fat mass (r(65)=-0.63, p<0.001) and waist circumference (r(65)=-0.55, p<0.001)). Controlling the correlation between body composition and light PA for sedentary behaviour resulted in significant positive correlation for body mass, BMI, fat mass, body fat percentage and waist circumference.

The graphical relationships between fat mass and the percentage time spent sedentary and in MVPA categories are shown in Figure 2.

Figure 2 near here

It is noticeable in Figure 2a that four participants have low amounts of sedentary behaviour and it was possible that these values were unduly influencing the correlation. When the statistical test was repeated excluding these subjects the correlation remained positive and significant (r(62)=0.31, p=0.01).

Table 2. Correlation between sedentary and active behaviours and body composition

	Body mass	BMI	Fat mass	Waist circumference	Lean mass
Sedentary behaviour	**0.44****	**0.50****	**0.50****	**0.45****	-0.01
Light PA	0.06	0.18	0.19	0.17	-0.18
MVPA	**-0.55****	**-0.71****	**-0.72****	**-0.65****	0.14
Sedentary behaviour[1]	-0.001	-0.14	-0.16	-0.13	0.18
Light PA[1]	0.01	0.16	0.18	0.15	-0.16
Light PA[2]	**0.32†**	**0.54****	**0.52****	**0.45****	-0.19
MVPA[2]	**-0.38****	**-0.57****	**-0.63****	**-0.55****	0.24

n=68; Data are Pearson correlation (r). [1] Controlled for MVPA (minutes); [2] Controlled for sedentary behaviour (minutes). ** p<0.001; † p<0.01. BMI, body mass index.

Associations between sedentary behaviour, physical activity and markers of appetite dysregulation

There were no significant correlations between sedentary behaviour and any of the indices of appetite dysregulation; Restraint (r(66)=-0.13, p=0.30), Disinhibition (r(66)=0.16, p=0.19), Hunger (r(66)=-0.02, p=0.88), and Binge Eating (r(66)=0.14, p=0.25).

However, light PA and MVPA showed some relationship to the questionnaire scores, but these were no longer apparent when partial correlations were performed controlling for the amount of body fat (see Table 3).

Table 3. Correlations between sedentary and active behaviours and appetite dysregulation

	Sedentary behaviour	Light PA	MVPA	Sedentary behaviour[1]	Light PA[1]	MVPA[1]
Restraint	-0.13	0.14	0.05	-0.15	0.15	0.08
Disinhibition	0.16	**0.36†**	**-0.44****	-0.13	0.25	-0.06
Hunger	-0.02	0.24	-0.15	-0.05	0.23	-0.16
Binge Eating	0.14	**0.24***	**-0.34†**	-0.05	0.15	-0.07

n=68; Data are Pearson correlation (r). [1] Controlled for body fat percentage. ** p<0.001; † p<0.01. MVPA, moderate-to-vigorous physical activity.

Associations among physical activity, sedentary behaviour and energy expenditure

In order to investigate whether the relationship between behaviour and adiposity was accounted for by energy expenditure, Activity Energy Expenditure (AEE) was calculated as the difference between Total EE (Armband) and RMR (directly measured by indirect calorimetry). The AEE was positively correlated with MVPA (r(66)=0.48, p<0.0001) and negatively related to time spent in sedentary behaviour (r=(66)0.57, p <0.0001).

Associations between markers of appetite dysregulation and body composition

TFEQ Disinhibition and Binge Eating were positively associated with body mass (r(69)=0.51 and r(69)=0.49, respectively, p<0.001), BMI (r(69)=0.59 and r(69)0.45, respectively, p<0.001), fat mass (r(69=0.58 and r(69)=0.47, respectively, p<0.001) and waist circumference (r(69)=0.56 and r(69)=0.48, respectively, p<0.001). Fat free mass was not significantly associated with any of the measures of appetite dysregulation nor were there any associations between any of the measures of body composition and Restraint or Hunger (see table 4).

Table 4. Correlations between body composition and appetite dysregulation

	Body mass	BMI	Fat mass	Waist circumference	Lean mass
Restraint	-0.20	-0.05	-0.07	-0.14	-0.23
Disinhibition	**0.51****	**0.59****	**0.58****	**0.56****	0.00
Hunger	0.18	0.12	0.10	0.12	0.15
Binge Eating	**0.49****	**0.45****	**0.47****	**0.48****	0.12

n=71; Data are Pearson correlation (r). ** p<0.001. BMI, body mass index.

DISCUSSION

The aim of the present study was to examine the associations amongst objectively measured free-living sedentary and active behaviours, body composition and appetite dysregulation, and to throw light upon the potential link between physical (in)activity and obesity.

Free-living sedentary and active behaviour and adiposity

Our data show sedentary behaviour and light PA was associated with higher adiposity. However, after controlling for MVPA the magnitude of the correlation between sedentary behaviour and body fat percentage was weakened and the correlation between light PA and body fat percentage was strengthened. Previous research assessing the relationship between sedentary behaviour and adiposity has yielded mixed results. Lynch et al,[23] reported an association between sedentary time and waist circumference and BMI in breast cancer survivors, furthermore after controlling for MVPA the associations were attenuated. Similarly, when lean and obese individuals were compared the obese group spent around 2 hours/day longer in sedentary behaviours.[24 25] Longitudinal studies have also demonstrated an association between sedentary behaviour and adiposity. Ekelund et al.[26] found that those who gained weight over a 5 to 6 year period performed significantly more sedentary behaviour than those who lost weight at follow-up.

The relationship between sedentary behaviour, light PA and adiposity has important implications given that sedentary behaviour and light PA accounts for the majority of the waking day.[27] In the current sample participants spent just over 11 hours of their waking day in sedentary activities and over 3 hours in light PA. Similar values have been observed in previous studies,[28 29] however, some studies report less sedentary time and more light intensity PA perhaps due to variations in measurement techniques.[30 31] Important to note are the correlations between light intensity PA and all markers of adiposity after controlling for sedentary behaviour. Under these circumstances light PA is associated with increased body mass, BMI, fat mass, body fat percentage and waist circumference and becomes a marker for sedentary behaviour. We have noted the inverse association between light and vigorous PA this means that the protective effect of exercise on adiposity is threshold based, and needs to be at least moderate intensity to produce any benefit.

Our data confirm the association between MVPA and adiposity previously demonstrated.[23 31-34] MVPA was inversely associated with body mass, BMI, fat mass, body fat percentage and waist circumference independent of sedentary behaviour. The positive association between MVPA and total energy expenditure observed in our data (data not presented) provides one possible explanation for the relationship with adiposity; PA results in increased energy expenditure. Healy et al,[34] also demonstrated an inverse association between MVPA and adiposity independent of sedentary behaviour. After controlling for MVPA only body fat percentage remained significantly correlated with sedentary behaviour but all correlations remained significant between MVPA and indices of adiposity when controlled for sedentary behaviour. This suggests that the absence of MVPA could be more important than the presence of sedentary behaviour in the accumulation of fat mass. Recommendation to displace sedentary time with light PA may not be sufficient for weight management and to accrue any benefit PA must be at least moderate intensity in line with current PA guidelines.[35]

Free-living sedentary and active behaviour, appetite dysregulation and adiposity

There were no correlations between sedentary behaviour and any of the measures of appetite dysregulation. MVPA was associated with higher Disinhibition and Binge Eating but these relationships were no longer significant after controlling for body fat percentage. Our analysis has shown a strong relationship between measures of adiposity and questionnaire measures of eating that imply a loss of control over appetite in the environment. This association is supported by many studies in the literature.[36 37] This outcome suggests that any observed relationship between sedentary behaviour and trait measures of poor appetite control may be mediated indirectly via mechanisms involved in adipose tissue dynamics.

Conclusion

This study has examined the relationship between objective measures of physical activity (from sedentary to vigorous) and measures of adiposity under conditions of daily habitual living. The outcome has shown that the level of physical activity is associated with body fatness and is likely to be relevant for obesity.

The outcome measures were based on systematic measures taken under natural conditions without any specific intervention. The analysis was derived from correlations (and partial correlations) and the interpretation informed by logic and plausibility. We are aware that correlations are not proof of causation, but they certainly do not rule out the possibility of causal relationships. This study has shown strong and statistically significant links between bodily activity and adiposity; this provides presumptive evidence that sedentary behaviour itself and a low level of physical activity is relevant for obesity. Our interpretation is that bidirectional causality can account for this link. Therefore, low levels of physical activity involving low energy expenditure will lead to a positive energy balance and favour the gain of body fat. In turn a greater degree of adiposity (caused by low activity or by high energy intake) will serve as a disincentive to perform physical activity and will favour a positive energy balance. However, these comments are one interpretation of the data and should be clarified with further investigation.

Importantly, the relevance of physical activity for obesity is corroborated by intervention studies. It has been demonstrated that taking people from an inactive to an active state by means of a regime of supervised daily exercise leads to a significant loss of fat tissue and a gain (or maintenance) of lean mass.[11 13] In contrast when people are shifted from an active to a sedentary state, there is no down-regulation of food intake thereby resulting in a positive energy balance and the potential for weight gain.[38] It is important to recognise that evidence and arguments indicating the importance of low physical activity in adiposity, does not deny the contribution of food intake to obesity. Indeed there is abundant evidence that overconsumption of food is a major cause of a positive energy balance and increased body fatness.[39] Interestingly the dynamic effects of fatness itself exacerbate the energy imbalance; while increasing adiposity serves as a disincentive to perform physical activity, it does not deter food consumption.

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CONTRIBUTION

AM, CG, GF and JB designed research; AM conducted research; AM, CG, GF and JB analysed data; AM, CG, GF and JB wrote manuscript. All authors discussed results/interpretation and approved the final manuscript. No authors declare a conflict of interest.

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REFERENCES

1. Swinburn B, Sacks G, Ravussin E. Increased food energy supply is more than sufficient to explain the US epidemic of obesity. The American journal of clinical nutrition 2009;**90**(6):1453-56

2. Vandevijvere S, Chow CC, Hall KD, et al. Increased food energy supply as a major driver of the obesity epidemic: a global analysis. Bulletin of the World Health Organization 2015;**93**(7):446-56

3. Church TS, Thomas DM, Tudor-Locke C, et al. Trends over 5 decades in US occupation-related physical activity and their associations with obesity. PloS one 2011;**6**(5):e19657

4. Telegraph D. Health warning - Exercise makes you fat. Secondary Health warning - Exercise makes you fat 2009. <http://www.telegraph.co.uk/science/6083234/Health-warning-exercise-makes-you-fat.html>; .

5. Donnelly JE, Smith BK. Is exercise effective for weight loss with ad libitum diet? Energy balance, compensation, and gender differences. Exercise and sport sciences reviews 2005;**33**(4):169-74

6. Shaw K, Gennat H, O'Rourke P, et al. Exercise for overweight or obesity. Cochrane Database Syst Rev 2006;**4**(4)

7. Malhotra A, Noakes T, Phinney S. It is time to bust the myth of physical inactivity and obesity: you cannot outrun a bad diet. British journal of sports medicine 2015:bjsports-2015-094911

8. Blair SN. Physical inactivity and obesity is not a myth: Dr Steven Blair comments on Dr Aseem Malhotra's editorial. British journal of sports medicine 2015;**49**(15):968-69

9. Mahtani KR, McManus J, Nunan D. Physical activity and obesity editorial: is exercise pointless or was it a pointless exercise? British journal of sports medicine 2015:bjsports-2015-095005

10. King NA, Hopkins M, Caudwell P, et al. Individual variability following 12 weeks of supervised exercise: identification and characterization of compensation for exercise-induced weight loss. International Journal of Obesity 2008;**32**(1):177-84

11. King NA, Caudwell PP, Hopkins M, et al. Dual-process action of exercise on appetite control: increase in orexigenic drive but improvement in meal-induced satiety. The American journal of clinical nutrition 2009;**90**(4):921-27

12. Caudwell P, Gibbons C, Hopkins M, et al. No sex difference in body fat in response to supervised and measured exercise. Medicine & Science in Sports & Exercise 2013;**45**(2):351-58

13. Caudwell P, Gibbons C, Finlayson G, et al. Exercise and weight loss: No sex differences in body weight response to exercise. Exercise and sport sciences reviews 2014;**42**(3):92-101

14. Myers A, Finlayson G, Blundell J, et al. Investigating the impact of sedentariness on appetite control. A multilevel platform linking energy intake and energy expenditure. Appetite 2015(87):390

15. Stunkard AJ, Messick S. The three-factor eating questionnaire to measure dietary restraint, disinhibition and hunger. Journal of psychosomatic research 1985;**29**(1):71-83

16. Gormally J, Black S, Daston S, et al. The assessment of binge eating severity among obese persons. Addictive behaviors 1982;**7**(1):47-55

17. Ainsworth BE, Haskell WL, Herrmann SD, et al. 2011 Compendium of Physical Activities: a second update of codes and MET values. Medicine and science in sports and exercise 2011;**43**(8):1575-81

18. Department of Health. Start active, stay active: a report on physical activity from the four home countries' Chief Medical Officers. Secondary Start active, stay active: a report on physical activity from the four home countries' Chief Medical Officers 2011. https://www.gov.uk/government/publications/start-active-stay-active-a-report-on-physical-activity-from-the-four-home-countries-chief-medical-officers.

19. Berntsen S, Hageberg R, Aandstad A, et al. Validity of physical activity monitors in adults participating in free-living activities. British Journal of Sports Medicine 2010;**44**(9):657-64

20. Calabró MA, Lee J-M, Saint-Maurice PF, et al. Validity of physical activity monitors for assessing lower intensity activity in adults. International Journal of Behavioral Nutrition and Physical Activity 2014;**11**(1):119

21. Malavolti M, Pietrobelli A, Dugoni M, et al. A new device for measuring resting energy expenditure (REE) in healthy subjects. Nutrition, metabolism and cardiovascular diseases 2007;**17**(5):338-43

22. St-Onge M, Mignault D, Allison DB, et al. Evaluation of a portable device to measure daily energy expenditure in free-living adults. The American journal of clinical nutrition 2007;**85**(3):742-49

23. Lynch BM, Dunstan DW, Healy GN, et al. Objectively measured physical activity and sedentary time of breast cancer survivors, and associations with adiposity: findings from NHANES (2003–2006). Cancer Causes & Control 2010;**21**(2):283-88

24. Johannsen DL, Welk GJ, Sharp RL, et al. Differences in daily energy expenditure in lean and obese women: the role of posture allocation. Obesity 2008;**16**(1):34-39

25. Levine JA, Lanningham-Foster LM, McCrady SK, et al. Interindividual variation in posture allocation: possible role in human obesity. Science 2005;**307**(5709):584-86

26. Ekelund U, Brage S, Besson H, et al. Time spent being sedentary and weight gain in healthy adults: reverse or bidirectional causality? The American journal of clinical nutrition 2008;**88**(3):612-17

27. Dempsey PC, Owen N, Biddle SJ, et al. Managing sedentary behavior to reduce the risk of diabetes and cardiovascular disease. Current diabetes reports 2014;**14**(9):1-11

28. Smith L, Thomas EL, Bell JD, et al. The association between objectively measured sitting and standing with body composition: a pilot study using MRI. BMJ open 2014;**4**(6):e005476

29. Hamer M, Venuraju SM, Urbanova L, et al. Physical activity, sedentary time, and pericardial fat in healthy older adults. Obesity 2012;**20**(10):2113-17

30. Golubic R, Wijndaele K, Sharp S, et al. Physical activity, sedentary time and gain in overall and central body fat: 7-year follow-up of the ProActive trial cohort. International Journal of Obesity 2014;**39**(1):142-8

31. McGuire KA, Ross R. Incidental physical activity and sedentary behavior are not associated with abdominal adipose tissue in inactive adults. Obesity 2012;**20**(3):576-82

32. Murabito JM, Pedley A, Massaro JM, et al. Moderate-to-Vigorous Physical Activity With Accelerometry is Associated With Visceral Adipose Tissue in Adults. Journal of the American Heart Association 2015;**4**(3):e001379

33. Larsen BA, Allison MA, Kang E, et al. Associations of physical activity and sedentary behavior with regional fat deposition. Medicine and science in sports and exercise 2014;**46**(3):520-28

34. Healy GN, Dunstan DW, Salmon J, et al. Breaks in sedentary time beneficial associations with metabolic risk. Diabetes care 2008;**31**(4):661-66

35. Department of Health. UK Physical Activity Guidelines. Secondary UK Physical Activity Guidelines 2011. https://www.gov.uk/government/publications/uk-physical-activity-guidelines.

36. Bryant EJ, King N, Blundell JE. Disinhibition: its effects on appetite and weight regulation. Obesity Reviews 2008;**9**(5):409-19

37. Bellisle F, Clément K, Barzic M, et al. The Eating Inventory and body adiposity from leanness to massive obesity: a study of 2509 adults. Obesity research 2004;**12**(12):2023-30

38. Stubbs RJ, Hughes DA, Johnstone AM, et al. A decrease in physical activity affects appetite, energy, and nutrient balance in lean men feeding ad libitum. The American journal of clinical nutrition 2004;**79**(1):62-69

39. Hall KD, Sacks G, Chandramohan D, et al. Quantification of the effect of energy imbalance on bodyweight. The Lancet 2011;**378**(9793):826-37

LEGENDS

Figure 1. The proportion of waking time spent sedentary, in light PA and MVPA. Data presented as percentage of awake time and total minutes.

Figure 2. Correlation between proportion of awake time spent sedentary and in MVPA and fat mass.

Figure 3. Correlation between fat mass and binge eating.