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/m² (±5.2) were continuously monitored for 6-7 days 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 SenseWare 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/m² (±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 sphygmanometer (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 (\(\dot{V}O_2\max\)) 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 \(\dot{V}O_2\max\) 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).
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>
<thead>
<tr>
<th>Variable</th>
<th>Mean (SD)</th>
<th>Range</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)*</td>
<td>37.35 (14.01)</td>
<td>18.00 – 72.00</td>
</tr>
<tr>
<td>Height (m)*</td>
<td>1.66 (0.09)</td>
<td>1.49 – 1.91</td>
</tr>
<tr>
<td>Body mass (kg)*</td>
<td>82.24 (15.26)</td>
<td>44.90 – 113.90</td>
</tr>
<tr>
<td></td>
<td>Mean (SD)</td>
<td>Range</td>
</tr>
<tr>
<td>------------------------------</td>
<td>-------------</td>
<td>-------------</td>
</tr>
<tr>
<td><strong>BMI (kg/m²)</strong></td>
<td>29.94 (5.24)</td>
<td>19.10 – 39.90</td>
</tr>
<tr>
<td><strong>Fat mass (kg)</strong></td>
<td>31.79 (13.37)</td>
<td>5.00 – 60.40</td>
</tr>
<tr>
<td><strong>Lean mass (kg)</strong></td>
<td>50.44 (9.28)</td>
<td>32.10 – 81.40</td>
</tr>
<tr>
<td><strong>Waist circumference (cm)</strong></td>
<td>100.23 (12.83)</td>
<td>69.00 – 133.70</td>
</tr>
<tr>
<td><strong>Systolic blood pressure (mm Hg)</strong></td>
<td>118.17 (14.12)</td>
<td>87.00 – 162.00</td>
</tr>
<tr>
<td><strong>Diastolic blood pressure (mm Hg)</strong></td>
<td>77.80 (10.25)</td>
<td>61.00 – 77.80</td>
</tr>
<tr>
<td><strong>Resting heart rate (bpm)</strong></td>
<td>58.56 (9.71)</td>
<td>37.00 – 84.00</td>
</tr>
<tr>
<td><strong>Blood glucose (mmol/L)</strong></td>
<td>4.73 (0.69)</td>
<td>1.98 – 6.70</td>
</tr>
<tr>
<td><strong>Resting metabolic rate (kcal/d)</strong></td>
<td>1698.54 (296.86)</td>
<td>1070.90 – 2451.90</td>
</tr>
<tr>
<td><strong>Total energy expenditure (kcal/d)</strong></td>
<td>2708.07 (421.81)</td>
<td>1827.30 – 4256.60</td>
</tr>
<tr>
<td><strong>Cardiovascular fitness (ml/kg/min)</strong></td>
<td>40.99 (7.88)</td>
<td>29.60 – 54.93</td>
</tr>
<tr>
<td><strong>SWA wear time (hours/d)</strong></td>
<td>23.55 (0.26)</td>
<td>22.47 – 23.95</td>
</tr>
<tr>
<td><strong>Sedentary behaviour (hours/d)</strong></td>
<td>11.06 (1.72)</td>
<td>6.01 – 15.40</td>
</tr>
<tr>
<td><strong>Light PA (hours/d)</strong></td>
<td>3.26 (1.03)</td>
<td>1.35 – 6.05</td>
</tr>
<tr>
<td><strong>MVPA (hours/d)</strong></td>
<td>2.10 (1.40)</td>
<td>0.48 – 6.74</td>
</tr>
<tr>
<td><strong>Restraint</strong></td>
<td>8.21 (3.82)</td>
<td>0.00 – 17.00</td>
</tr>
<tr>
<td><strong>Disinhibition</strong></td>
<td>8.85 (3.88)</td>
<td>0.00 – 15.00</td>
</tr>
<tr>
<td><strong>Hunger</strong></td>
<td>6.00 (3.16)</td>
<td>0.00 – 13.00</td>
</tr>
<tr>
<td><strong>Binge Eating</strong></td>
<td>13.23 (7.31)</td>
<td>1.00 – 34.00</td>
</tr>
</tbody>
</table>

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.

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

<table>
<thead>
<tr>
<th></th>
<th>Body mass</th>
<th>BMI</th>
<th>Fat mass</th>
<th>Waist circumference</th>
<th>Lean mass</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sedentary behaviour</td>
<td>0.44**</td>
<td>0.50**</td>
<td>0.50**</td>
<td>0.45**</td>
<td>-0.01</td>
</tr>
<tr>
<td>Light PA</td>
<td>0.06</td>
<td>0.18</td>
<td>0.19</td>
<td>0.17</td>
<td>-0.18</td>
</tr>
<tr>
<td>MVPA</td>
<td>-0.55**</td>
<td>-0.71**</td>
<td>-0.72**</td>
<td>-0.65**</td>
<td>0.14</td>
</tr>
<tr>
<td>Sedentary behaviour$^1$</td>
<td>-0.001</td>
<td>-0.14</td>
<td>-0.16</td>
<td>-0.13</td>
<td>0.18</td>
</tr>
<tr>
<td>Light PA$^1$</td>
<td>0.01</td>
<td>0.16</td>
<td>0.18</td>
<td>0.15</td>
<td>-0.16</td>
</tr>
<tr>
<td>Light PA$^2$</td>
<td>0.32†</td>
<td>0.54**</td>
<td>0.52**</td>
<td>0.45**</td>
<td>-0.19</td>
</tr>
<tr>
<td>MVPA$^2$</td>
<td>-0.38**</td>
<td>-0.57**</td>
<td>-0.63**</td>
<td>-0.55**</td>
<td>0.24</td>
</tr>
</tbody>
</table>

$n=68$; 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).
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 ($r(66)=0.48$, $p<0.001$) and negatively related to time spent in sedentary behaviour ($r=0.57$, $p<0.001$).

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 3. Correlations between sedentary and active behaviours and appetite dysregulation

<table>
<thead>
<tr>
<th></th>
<th>Sedentary behaviour</th>
<th>Light PA</th>
<th>MVPA</th>
<th>Sedentary behaviour&lt;sup&gt;1&lt;/sup&gt;</th>
<th>Light PA&lt;sup&gt;1&lt;/sup&gt;</th>
<th>MVPA&lt;sup&gt;1&lt;/sup&gt;</th>
</tr>
</thead>
<tbody>
<tr>
<td>Restraint</td>
<td>-0.13</td>
<td>0.14</td>
<td>0.05</td>
<td>-0.15</td>
<td>0.15</td>
<td>0.08</td>
</tr>
<tr>
<td>Disinhibition</td>
<td>0.16</td>
<td><strong>0.36†</strong></td>
<td><strong>-0.44</strong></td>
<td>-0.13</td>
<td>0.25</td>
<td>-0.06</td>
</tr>
<tr>
<td>Hunger</td>
<td>-0.02</td>
<td>0.24</td>
<td>-0.15</td>
<td>-0.05</td>
<td>0.23</td>
<td>-0.16</td>
</tr>
<tr>
<td>Binge Eating</td>
<td>0.14</td>
<td><strong>0.24</strong></td>
<td><strong>-0.34†</strong></td>
<td>-0.05</td>
<td>0.15</td>
<td>-0.07</td>
</tr>
</tbody>
</table>

n=68; Data are Pearson correlation ($r$). <sup>1</sup> Controlled for body fat percentage. <sup>**</sup> $p<0.001$; † $p<0.01$.

MVPA, moderate-to-vigorous physical activity.
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

Table 4. Correlations between body composition and appetite dysregulation

<table>
<thead>
<tr>
<th></th>
<th>Body mass</th>
<th>BMI</th>
<th>Fat mass</th>
<th>Waist circumference</th>
<th>Lean mass</th>
</tr>
</thead>
<tbody>
<tr>
<td>Restraint</td>
<td>-0.20</td>
<td>-0.05</td>
<td>-0.07</td>
<td>-0.14</td>
<td>-0.23</td>
</tr>
<tr>
<td>Disinhibition</td>
<td>0.51**</td>
<td>0.59**</td>
<td>0.58**</td>
<td>0.56**</td>
<td>0.00</td>
</tr>
<tr>
<td>Hunger</td>
<td>0.18</td>
<td>0.12</td>
<td>0.10</td>
<td>0.12</td>
<td>0.15</td>
</tr>
<tr>
<td>Binge Eating</td>
<td>0.49**</td>
<td>0.45**</td>
<td>0.47**</td>
<td>0.48**</td>
<td>0.12</td>
</tr>
</tbody>
</table>

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