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

Anna Myers, Catherine Gibbons, Graham Finlayson, John Blundell

ABSTRACT

Background There is considerable disagreement about the association between free-living physical activity (PA) and sedentary behaviour and obesity. Moreover studies frequently do not include measures that could mediate between PA 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 71 participants (81.7% women) aged 37.4 years (±14) with a body mass index of 29.9 kg/m² (±5.2) were continuously monitored for 6–7 days to track free-living PA (light 1.5–3 metabolic equivalents (METs), moderate 3–6 METs and vigorous >6 METs) and sedentary behaviour (<1.5 METs) 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 h/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 PA 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).

Conclusions This study demonstrated clear associations among objective measures of PA (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 bidirectional.

BACKGROUND

In recent years, the relative contributions of over-consumption of food and the underexpenditure 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. This argument has recently been extended to a global level. In contrast, it has been argued that the decline in work-related physical activity (PA; and therefore energy expenditure (EE)) over several decades has been sufficient to account for a positive energy balance and the rise in obesity in the USA. 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’. These headlines have appeared despite evidence from controlled trials demonstrating dose-related effects of PA on weight loss; 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, 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’. Strongly argued articles refuting these claims have attempted to prevent further damaging perceptions emanating from these claims.

For over two decades, we have investigated the interactions between EE and energy intake. 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. These studies indicate that PA 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 spent in sedentary behaviour and in light, moderate and vigorous activity. 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, EE, 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. Participants were males and females aged >18 years with no contraindications to exercise and not taking medication.

Study design
Participants attended the research unit twice over the course of 1 week. Free-living PA and sedentary behaviour were measured continuously for a minimum of 7 days for >22 h/day. Participants were fasted for a minimum of 12 h and had abstained from exercise and alcohol for at least 24 h before both laboratory visits.

On the morning of day 1, 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 (TFEQ), Binge Eating Scale). Participants were provided with a PA diary and fitted with a SenseWear Mini Armband (BodyMedia, Inc, Pittsburgh, Pennsylvania, USA).

Anthropometrics
Height was measured using a stadiometer (Leicester height measure, SECA) and body composition was measured using air plethysmography (BodPod, Concord, California, USA). Body weight was obtained from the BodPod while participants were wearing minimal clothing. BMI was calculated as weight in kg/height in m². 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 min, with RMR calculated from respiratory data averaged during the last 30 min 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 analysed using a blood glucose analyser (YSI 2300 STAT PLUS Glucose and Lactate Analyzer).

Appetite dysregulation
Participants completed the TFEQ, a 51-item questionnaire measuring restraint, disinhibition and hunger, and the Binge Eating Scale, a 16-item questionnaire measuring binge eating behaviour and cognitions indicative of eating disorders.

Free-living PA and EE
Free-living PA 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 h/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 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. 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 moderate-to-vigorous PA (MVPA) category to correspond with the guidelines for PA. The SWA has been shown to accurately estimate time in MVPA and EE at rest and during free-living light-intensity and moderate-intensity PA. For the SWA data to be valid, >22 h of data per day had to be recorded and at least six 24 h periods (midnight to midnight) including two weekend days. Participants completed a PA 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 laboratory 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 (VO₂max) was measured during an incremental treadmill test with expired air (Sensormedics Vmax29, Yorba Linda, California, USA) and HR (Polar RS400, Polar, Kempele, Finland) measured continuously. Attainment of true VO₂max was determined by a plateau in VO₂ with an increase in workload, a respiratory quotient 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, USA, V21). For reasons of scientific rigour and to reduce the likelihood of false positives, we only regarded relationships 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 among 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 23.5 ± 2.06 h/day (98 ± 1.2%). Participants were sedentary for an average of 11.06 ± 1.72 h/day (excluding sleep) and recorded...
Associations between sedentary behaviour, PA 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)=−0.65, 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 correlations 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 correlations 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 participants, the correlation remained positive and significant (r(62)=0.34, p<0.01).

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

<table>
<thead>
<tr>
<th>Variable</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†</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†</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‡</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‡</td>
<td>−0.38‡</td>
<td>−0.57‡</td>
<td>−0.63‡</td>
<td>−0.55‡</td>
<td>0.24</td>
</tr>
</tbody>
</table>

Bold figures indicate correlations that are statistically significant.

N=68; data are Pearson correlation (r).

* † p<0.001.
†† Controlled for MVPA (min).
†‡ Controlled for sedentary behaviour (min).
§p<0.01.
‡BMI, body mass index; MVPA, moderate-to-vigorous physical activity.

3.26±1.03 h/day in light PA and 2.10±1.40 h/day in MVPA (see figure 1). Participants mean age was 37.35±14.01 and their average total EE was 2708.07±421.81 kcal/day.

**Table 1** Descriptive statistics of study sample

<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>BMI (kg/m²)†</td>
<td>29.94 (5.24)</td>
<td>19.10–39.90</td>
</tr>
<tr>
<td>Fat mass (kg)*</td>
<td>31.79 (13.37)</td>
<td>5.00–60.40</td>
</tr>
<tr>
<td>Lean mass (kg)*</td>
<td>50.44 (9.28)</td>
<td>32.10–81.40</td>
</tr>
<tr>
<td>Sedentary behaviour (h/day)</td>
<td>11.06 (1.72)</td>
<td>6.01–23.95</td>
</tr>
<tr>
<td>SWA wear time (h/day)§</td>
<td>23.55 (0.26)</td>
<td>22.47–23.95</td>
</tr>
<tr>
<td>Total energy expenditure (kcal/day)§</td>
<td>2708.07 (421.81)</td>
<td>1827.30–4256.60</td>
</tr>
<tr>
<td>Cardiovascular fitness (mL/kg/min)§</td>
<td>40.99 (7.88)</td>
<td>29.60–54.93</td>
</tr>
<tr>
<td>SWA wear time (h/day)§</td>
<td>23.55 (0.26)</td>
<td>22.47–23.95</td>
</tr>
<tr>
<td>Sedentary behaviour (h/day)§</td>
<td>11.06 (1.72)</td>
<td>6.01–15.40</td>
</tr>
<tr>
<td>MVPA (h/day)§</td>
<td>3.26 (1.03)</td>
<td>1.35–6.05</td>
</tr>
<tr>
<td>Restraint*</td>
<td>8.21 (3.82)</td>
<td>0.00–17.00</td>
</tr>
<tr>
<td>Disinhibition*</td>
<td>8.85 (3.88)</td>
<td>0.00–15.00</td>
</tr>
<tr>
<td>Hunger*</td>
<td>5.32 (3.16)</td>
<td>0.00–13.00</td>
</tr>
<tr>
<td>Binge eating*</td>
<td>19.89 (7.31)</td>
<td>1.00–34.00</td>
</tr>
</tbody>
</table>

BMI, body mass index; MVPA, moderate-to-vigorous physical activity; SWA, SenseWear armband.

**Figure 1** The proportion of waking time spent in sedentary, light physical activity (PA) and moderate-to-vigorous PA (MVPA). Data presented as percentage of awake time and total minutes.
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).

### Associations among PA, sedentary behaviour and EE

In order to investigate whether the relationship between behaviour and adiposity was accounted for by EE, activity EE (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 $r(69)=0.47$, (see figure 3) 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).

### DISCUSSION

The aim of the present study was to examine the associations among objectively measured free-living sedentary and active behaviours, body composition and appetite dysregulation, and to throw light on 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. 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 h/day longer in sedentary behaviours. Longitudinal studies have also demonstrated an association between sedentary behaviour and adiposity. Ekelund et al. found that those who gained weight over a 5–6-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.

### 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†</th>
<th>Light PA†</th>
<th>MVPA†</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>

Bold figures indicate correlations that are statistically significant. N=68; data are Pearson correlation ($r$).

**$p<0.001$.**  
†$p<0.01$.  
‡Controlled for body fat percentage.  
MVPA, moderate-to-vigorous physical activity.
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.33–36 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 EE observed in our data provides one possible explanation for the relationship with adiposity: PA results in increased EE. Healy et al.37 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. Recommendations 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.33

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 or active 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 PA (from sedentary to vigorous) and measures of adiposity under conditions of daily habitual living. The outcome has shown that the level of PA 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 PA is relevant for obesity. Our interpretation is that bidirectional causality can account for this link. Therefore, low levels of PA involving low EE 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 PA 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 PA 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.31–33 In contrast, when people are shifted from an active to a sedentary state, there is no downregulation of food intake thereby resulting in a positive energy balance and the potential for weight gain.33 It is important to recognise that evidence and arguments indicating the importance of low PA 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.37 Interestingly, the dynamic effects of fatness itself exacerbate the energy imbalance; while increasing adiposity serves as a disincentive to perform PA, it does not deter food consumption.

Correction notice This paper has been amended since it was published Online First. Under the section heading ‘Association between sedentary behaviour and different categories of PA’ the ‘<’ signs were incorrectly added. These have been replaced with the ‘=’ symbol. In the section titled ‘Associations among PA, sedentary behaviour and EE’, the significance value was also missing from one of the reported correlations and this has now been amended.

Twitter Follow https://twitter.com/aceb_leeds

Acknowledgements The research leading to these results has received funding from EU projects under grant agreement n° 610440 (DAPHNE). The authors are grateful to Dr Nicola Buckland for her contribution to the logistics of the study.

Contributors AM, CG, GF and JB designed research; AM conducted research; AM, CG, GF and JB analysed the data and wrote the manuscript. All authors discussed results/interpretation and approved the final manuscript.

Competing interests None declared.

Provenance and peer review Not commissioned; externally peer reviewed.

REFERENCES


Associations among sedentary and active behaviours, body fat and appetite dysregulation: investigating the myth of physical inactivity and obesity
Anna Myers, Catherine Gibbons, Graham Finlayson and John Blundell

doi: 10.1136/bjsports-2015-095640

Updated information and services can be found at:
http://bjsm.bmj.com/content/51/21/1540

These include:

**References**

This article cites 35 articles, 13 of which you can access for free at:
http://bjsm.bmj.com/content/51/21/1540#BIBL

**Email alerting service**

Receive free email alerts when new articles cite this article. Sign up in the box at the top right corner of the online article.

**Topic Collections**

Articles on similar topics can be found in the following collections

- Health education (481)
- Obesity (nutrition) (120)
- Obesity (public health) (120)

**Notes**

To request permissions go to:
http://group.bmj.com/group/rights-licensing/permissions

To order reprints go to:
http://journals.bmj.com/cgi/reprintform

To subscribe to BMJ go to:
http://group.bmj.com/subscribe/