PAPER

Substrate oxidation differences between high- and low-intensity exercise are compensated over 24 hours in obese men

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OBJECTIVE: Exercise has been proposed as a tool for the prevention of obesity. Apart from an effect on energy expenditure, in particular low-intensity (LI) exercise might also influence substrate metabolism in favour of fat oxidation. It is however unclear what is the most beneficial exercise regime for obese people. We therefore studied the effect of either high-intensity (HI) or LI exercise on 24 h energy expenditure (24 h EE) and substrate metabolism.

METHODS: Eight healthy obese male volunteers (age: 38 ± 1 y, BMI: 31 ± 1 kg/m², Wmax: 235 ± 16 W) stayed in the respiration chamber for two nights and the day in-between. In the chamber they cycled either at a HI (three times 30 min in a interval protocol (2.5 min 80/50% Wmax)) or LI (three times 60 min continuously at 38% Wmax) protocol with an eucaloric energy expenditure. In the chamber subjects were fed in energy balance (37/48/15% of energy as fat/carbohydrate/protein).

RESULTS: The 24 h EE was not significantly different between protocols. In both protocols, sleeping metabolic rate (SMR) was elevated after the exercise (average ± 6.7%). The 24 h respiratory quotient (24 h RQ) was not different between protocols. During exercise, RQ was higher in the HI compared to the LI protocol (0.93 vs 0.91 resp., P < 0.05), whereas in the postexercise period RQ tended to be lower in the HI compared to the LI protocol (P = 0.06).

CONCLUSION: 24 h EE is not differently affected by HI or LI exercise in obese men. Similarly, the differences in HI and LI exercise, RQ are compensated postexercise leading to similar substrate oxidation patterns over 24 h independently of the level of exercise intensity.

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Keywords: exercise; exercise intensity; energy metabolism; fat oxidation

Introduction

Obesity is a well-recognised problem in our Western society and the development of obesity is characterised by a chronic imbalance between energy expenditure and energy intake. This imbalance can be due to both a reduced energy expenditure and/or an excess energy and fat intake. Therefore, weight-reducing programs often focus on both decreasing energy intake by dietary restrictions, as well as on increasing energy expenditure by means of exercise. Although the weight loss due to exercise alone is rather small in comparison to restriction of the energy intake, regular exercise appears to be one of the major factors determining long-term weight maintenance success. In relation to the energy balance equation, the factor that has the greatest impact on energy expenditure is volume of the work, which is a function of duration and intensity of the exercise. Most of the debate so far has been focused on the duration of the exercise. In a recent consensus meeting, the current activity guideline for adults of 30 min of moderate intense activity preferable all days of the week was challenged. There is compelling evidence that the prevention of weight regain in formerly obese individuals requires 60–90 min of moderate intensity activity or lesser amounts of vigorous intensity activity. Although definitive data are lacking, it seems likely that moderate intensity activity of approximately 45–60 min is required to prevent the transition to overweight or obesity.

Even less clear is the role of the intensity of the exercise in relation to body weight control. High-intensity (HI) exercise can favourably impact energy expenditure in a number of ways. First, exercise-related energy expenditure is of course
higher per time unit and this is enhanced because the exercise efficiency drops with increasing intensity. HI aerobic exercise may increase 24 h resting metabolic levels. Although it has been shown that long-term endurance training also increases resting metabolic rate, this effect is mainly due to an increase in fat-free mass. Whether endurance training influences resting metabolic rate after adjustment for body composition is still controversial. Some studies have shown an increase in resting metabolic rate, adjusted for fat-free mass and fat mass, in response to training whereas others found no effect. In contrast to these studies, not much research has been focused on establishing the most beneficial exercise intensity for increasing 24 h energy expenditure 24 h EE. Some studies have shown that HI exercise has a more pronounced effect on postexercise energy expenditure compared to LI exercise, whereas other found no difference, but these studies only measured energy expenditure for several hours after exercise. However, to determine the optimal exercise intensity for the prevention and/or treatment of obesity, 24 h EE should be considered. Moreover, exercise intensity might not only have an effect on energy metabolism but also on substrate oxidation. This is of particular interest in the prevention of obesity since it has been suggested that in the obese fat oxidation is impaired and that a diminished fat oxidation is a predictor for future weight gain and obesity. Therefore, when dealing with the optimal exercise intensity to prevent weight gain also the effects on 24 h substrate oxidation should be taken into account. We have shown that dietary effects on fat oxidation are slow: after the introduction of a high-fat diet, fat oxidation only gradually increased and equalled fat intake after 7 days. In contrast, we and others have shown that a single exercise bout can rapidly increase fat oxidation for up to 36 h postexercise in lean and obese subjects. Thus, exercise can be helpful in increasing fat oxidation capacity and thereby facilitating weight maintenance and possibly preventing obesity. However, it is still unknown what is the best option for the obese taken into account that people do not like HI exercise.

During LI exercise energy is primarily supplied by fat, whereas during HI exercise the main energy supplier is carbohydrate oxidation, suggesting that LI exercise would be most beneficial for increasing fat oxidation. On the other hand, HI exercise will deplete glycogen stores, which will lead to increased rates of fat oxidation in the postexercise period. So far, only two studies have examined the long-term effect of exercise intensity on 24 h fat oxidation and these studies failed to find a positive effect of HI exercise on 24 h fat oxidation, although an increased 24 h energy expenditure after HI exercise was found in the study of Treuth et al. To the best of our knowledge, no controlled studies have been performed that examined the acute effect of exercise intensity on 24 h energy and substrate metabolism in obese subjects, which is of particular interest since obese subjects are characterised by an impaired fat oxidation capacity. Therefore, the aim of the present study was to study the acute effects of either HI or LI equivalent exercise on both 24 h energy expenditure and substrate oxidation in obese men.

**Methods**

**Subjects**

Eight male obese volunteers participated in the study. Their characteristics are shown in Table 1. All subjects were untrained (less than 1 h/week participating in sport activities) and obese (BMI > 30 kg/m²) males. A medical examination excluded subjects with cardiovascular diseases, diabetes mellitus, asthma or other metabolic diseases. The study was reviewed by the Medical Ethical Committee of the University of Maastricht and all subjects gave their written informed consent.

**Experimental design**

Each subject followed two different protocols in random order with 2 weeks in-between. Prior to each protocol subjects were given a diet for consumption at home for 3 days. Each protocol consisted of a 36 h stay in the respiration chamber. Subjects came to the laboratory on the evening of day 0 (2000) and left the respiration chamber on the morning of day 2 at 0830. While in the respiration chamber, subjects cycled three times 30 min at alternatively bouts of 2.5 min at 80 and 50% of their maximal work output (Wmax) (HI) or three times 60 min at 38% Wmax (LI). These exercise bouts were calculated to achieve an similar energy expenditure during both HI and LI protocol, assuming similar mechanical efficiency. On the morning of day 2, after the first visit in the respiration chamber, body composition was determined using underwater weighing.

**Maximal power output and exercise tests**

At 1 week before the experimental protocol each subject performed an incremental exhaustive exercise test on an electronically braked cycle ergometer (Lode Excalibur, Groningen, The Netherlands) to determine maximal oxygen consumption (VO2max) and maximal power output (Wmax).

<table>
<thead>
<tr>
<th>Table 1</th>
<th>Subjects' characteristics (n = 8)</th>
<th>mean ± s.e.m.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (y)</td>
<td>38.3 ± 1.1</td>
<td></td>
</tr>
<tr>
<td>Height (m)</td>
<td>1.76 ± 0.02</td>
<td></td>
</tr>
<tr>
<td>Weight (kg)</td>
<td>96.9 ± 4.6</td>
<td></td>
</tr>
<tr>
<td>Percent body fat (%)</td>
<td>32.3 ± 1.3</td>
<td></td>
</tr>
<tr>
<td>BMI (kg/m²)</td>
<td>31.2 ± 1.3</td>
<td></td>
</tr>
<tr>
<td>Wmax (w)</td>
<td>235 ± 16</td>
<td></td>
</tr>
<tr>
<td>VO2max (l/min)</td>
<td>3.3 ± 0.2</td>
<td></td>
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</table>

VO2max: maximal oxygen consumption.
Exercise was performed until voluntary exhaustion or until the subject could no longer maintain a pedal rate of more than 60 rpm. Subjects started at 100 W and work load was increased by 50 W every 2.5 min. When subjects were approaching exhaustion, as indicated by heart rate and subjective scoring, the increment was reduced to 25 W. In practice, this meant that the last one to three load increments were 25 W. Oxygen consumption and carbon dioxide production were measured continuously using a Sensormedics 2900 metabolic cart (Anaheim, USA). Heart rate was registered continuously using a Polar Sporttester (Kempele, Finland). In each individual $W_{\text{max}}$ was calculated from

\[ W_{\text{max}} = W_{\text{out}} + (t/150) \times 8W \]

in which $W_{\text{out}}$ is the highest workload completed by the subject, $t$ is the time (in s) performed on the last workload and $8W$ is the final uncompleted load increment.

In the respiration chamber subjects cycled three times (0900, 1300 and 1930). In the LI protocol subjects cycled for 60 min at 38% $W_{\text{max}}$. In the HI protocol subjects cycled for 30 min at alternatively 60 and 50% $W_{\text{max}}$ for 2.5 min periods. During the HI protocol all exercise periods were preceded by 5 min warming up at 33% $W_{\text{max}}$ and were ended by 5 minutes cooling down, also at 33% $W_{\text{max}}$. In this way total amount of work accomplished in both intensity groups was calculated to be identical.

Diets
Food intake diaries were filled in during 3 days (2 weekdays and one weekend day). Subjects habitual energy and macronutrient intake was determined using 3 days food intake records. Prior to each protocol subjects were given a diet for consumption at home for 3 days. Subjects were given a fixed amount of food (based on their food intake record) and ad libitum access to snacks. Energy requirement on the first day in the respiration chamber was set at 1.55 times sleeping metabolic rate (SMR) as measured during the first night plus an amount of energy equal to the amount of energy expended during the exercise tests. The latter was calculated by assuming a mechanical efficiency of 20%. At the second stay in the respiration chamber energy intake was fixed at the same level as during the first stay. All food was consumed as breakfast, lunch, dinner and two or more snacks per day. Diets consisted of 47% of energy as carbohydrate, 37% of energy as fat and 15% of energy as protein. Energy intake and macronutrient composition of the diets were calculated using the Dutch food composition table.\(^{27}\)

Procedures

**Body composition.** On the morning of day 2 whole-body density was determined by underwater weighing in the fasted state, directly after subjects left the respiration chamber. Body weight was measured with a digital balance accurate to 0.01 kg (Sauter, type E1200). Lung volume was measured simultaneously with the helium dilution technique using a spirometer (Vulograph 2000, Mijnhardt). Percent body fat was calculated using the equations of Siri.\(^{28}\)

**Indirect calorimetry and physical activity.** Oxygen consumption and carbon dioxide production was measured in a whole-room indirect calorimeter, which was described previously.\(^{29}\) The respiration chamber is a 14 m\(^3\) room furnished with a bed, chair, television, radio, telephone, intercom, wash bowl, and toilet. The room is ventilated with fresh air at a rate of 70-801/min. The ventilation rate was measured with a dry gas meter (Schlumberger, type G6, The Netherlands). The concentration of oxygen and carbon dioxide was measured using a paramagnetic O\(_2\) analyser (Hartmann & Braun, type Magnos G6, Germany) and an infrared CO\(_2\) analyser (Hartmann & Braun, type Uras 3G, Germany). Ingoing air was analysed every 15 min and outgoing air once every 5 min. The gas sample to be measured was selected by a computer that also stored and processed the data. Energy expenditure was calculated from O\(_2\) consumption and CO\(_2\) production according to the method of Weir.\(^{30}\) In the daytime, outside the exercise periods, no sleeping or other exercise was allowed during the stay in the respiration chamber. Therefore, the subjects mainly watched television, read or did some 'bench work'. Spontaneous physical activity of the subjects was monitored by means of a radar system based on the Doppler principle.

**Urinary nitrogen excretion.** During the stay in the respiration chamber 24 h urine was collected from 0800 to 0800. Subjects had to empty the bladder at 0800 so urine produced during the night could be included with the urine sample of the previous day. Samples were collected in containers with 10 ml H\(_2\)SO\(_4\) to prevent nitrogen loss through evaporation; volume and nitrogen concentration were measured, the latter using a nitrogen analyser (Heraeus, type CHN-O-Rapid).

**The 24 h EE and substrate oxidation.** The 24 h EE and 24 h respiratory quotient (RQ) were calculated from 0800 am to 0800 pm. SMR was defined as the lowest mean energy expenditure during three subsequent hours between 0000 and 0800. Energy expenditure during the exercise period was defined as the total energy expended during the three exercise bouts. The postexercise energy expenditure was defined as the total energy expended during the 2h after each exercise bout. Carbohydrate, fat and protein oxidation were calculated using O\(_2\)-consumption, CO\(_2\)-production and urinary nitrogen losses with the equations of Brouwer.\(^{31}\)

\[
\text{protein oxidation (g/day)} = 6.25 \times N
\]

\[
\text{fat oxidation (g/day)} = 1.718 \times V\text{O}_2 - 1.718 \times V\text{CO}_2 - 0.315 \times P
\]

\[
\text{carbohydrate oxidation (g/day)} = 4.17 \times V\text{CO}_2 - 2.955 \times V\text{O}_2 - 0.390 \times P
\]
where \( N \) is the total nitrogen excreted in urine (g/day), \( VO_2 \) is the oxygen consumption (l/day), \( VCO_2 \) is the carbon dioxide production (l/day), \( P \) is protein oxidation (g/day).

**Statistical analysis.** Paired t-test were used to test differences in measured variables between protocols. All data are presented as mean±s.e.m. and \( P<0.05 \) was considered as statistical significant.

**Results**

The habitual energy intake was 11.7±1.4 MJ/day. This was achieved by 37±4% of energy as fat, 43±4% of energy as carbohydrate and 17±1% of energy as protein. Maximal power output (\( W_{\text{max}} \)) was 235±16 W, and maximal oxygen consumption (\( VO_2\text{max} \)) was 3.5±0.21/min.

The 24H EE was not significantly different between the LI and HI protocol (Table 2, Figure 1). As there were no differences in energy intake, energy balance was not significantly different between the LI and HI protocol. More important, subjects were in energy balance during both protocols (-0.07±0.4 vs -0.13±0.2 MJ/day for HI and LI respectively, (Table 2). As designed, energy expenditure during exercise was equal between HI and LI (6.17±0.91 and 6.22±0.92 MJ respectively). Energy expended during exercise accounted for approximately 38% of 24 h EE. Energy expended in the postexercise period was also not significantly different between HI and LI (9.6±0.4 vs 9.8±0.5 KJ/min respectively). SMR was significantly higher during the second night compared to the first night in both protocols (\( P<0.05 \), Table 3). The within-subject coefficient of variation for SMR was 2.9±1.5%. Physical activity index (\( PAI=24\text{H EE}/\text{SMR} \)) was not significantly different between the two protocols (Table 3).

The 24 h RQ was not significantly different between HI and LI (Table 4). No significant differences in RQ between HI and LI and between night 1 and 2 were found (Table 4). For this period the within-subject coefficient of variation for RQ was 2.1±1.8%. Respiration quotient during the exercise period was significantly lower during LI compared to HI.

<table>
<thead>
<tr>
<th>Table 2</th>
<th>The 24-h energy intake, energy expenditure and energy balance as measured in the respiration chamber ( n=8 ) (mean±s.e.m)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Protocol</td>
<td>Intake (MJ)</td>
</tr>
<tr>
<td>LI</td>
<td>16.62±0.53</td>
</tr>
<tr>
<td>HI</td>
<td>16.62±0.54</td>
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</tbody>
</table>

<table>
<thead>
<tr>
<th>Table 3</th>
<th>SMR (from 0000–0800) and PAI as measured in the respiration chamber (mean±s.e.m)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Protocol</td>
<td>SMR night 1 (kJ/min)</td>
</tr>
<tr>
<td>LI</td>
<td>5.07±0.21</td>
</tr>
<tr>
<td>HI</td>
<td>5.20±0.20</td>
</tr>
</tbody>
</table>

*Physical activity index: 24 h EE/24 h SMR. *\( P<0.05 \) compared to night 1.

<table>
<thead>
<tr>
<th>Table 4</th>
<th>RQ as measured in the respiration chamber</th>
</tr>
</thead>
<tbody>
<tr>
<td>Protocol</td>
<td>24 h RQ</td>
</tr>
<tr>
<td>LI</td>
<td>0.89±0.01</td>
</tr>
<tr>
<td>HI</td>
<td>0.89±0.01</td>
</tr>
</tbody>
</table>

*\( P<0.05 \) compared to HI. *\( P=0.06 \) compared to HI.

**Figure 1.** Mean 24 h EE in the respiration chamber from 0800 on day 1 to 0730 on day 2 for the LI and HI protocol (\( n=8 \)).

**Figure 2.** The 24 h substrate balances (mean±s.e.m.) as measured in the respiration chamber from 0800 on day 1 to 0730 on day 2 for the LI and HI protocol.
(P < 0.05). In the postexercise period RQ tended to be higher during LI compared to HI (P = 0.06).

24 h fat oxidation was not significantly different between HI and LI. Fat oxidation was significantly lower than fat intake, resulting in positive fat balances of 33.2 ± 13.6 and 30.1 ± 8.2 g/day for HI and LI respectively (Figure 2). The 24 h carbohydrate oxidation was not significantly different between HI and LI. Carbohydrate oxidation was significantly higher than carbohydrate intake, resulting in negative carbohydrate balances of −120.1 ± 32.6 and −112.2 ± 25.6 g/day for HI and LI respectively (Figure 2). The 24 h protein oxidation was also not significantly different between HI and LI and protein oxidation was significantly lower than protein intake. This resulted in positive protein balances of 41.3 ± 7.3 and 46.3 ± 4.9 g/day for HI and LI respectively (Figure 2).

Discussion

The results of the present study demonstrate that both 24 h EE and substrate oxidation are not differently affected by equicaloric HI or LI exercise in obese men. During the LI exercise periods RQ was significantly lower indicating a higher fat oxidation. However, postexercise RQ tended to be lower after the HI exercise periods compared to the LI exercise, indicating a compensating effect on fat oxidation leading to a 24 h similar substrate oxidation profile.

Since obesity develops due to small differences between energy intake and energy expenditure over prolonged periods of time, physical inactivity has been suggested as an important predictor of weight gain.1,2 When considering the treatment of obesity and in particular body weight regain, regular physical activity can overcome the decrease in energy expenditure, which is associated with an initial lower body mass.3 Exercise not only increases total energy expenditure due to the extra energy expended during the exercise but also due to an elevated postexercise energy expenditure. Some studies have shown that HI exercise has a more pronounced effect on postexercise energy expenditure compared to LI exercise,13-15 whereas other found no difference.16,17 However, these studies only measured energy expenditure for several hours after the exercise bout. Therefore, we used the respiration chamber to study the effect of LI or HI exercise on energy and substrate metabolism, while subjects were fed in perfect energy balance. The latter is of crucial importance since both energy expenditure and especially substrate utilization can be easily influenced by a positive or negative energy balance, as was observed in the studies of Treuth et al.25,33 We found that under energy balance conditions three times of either LI or HI exercise with the same total workload resulted in an increase in SMR during the night following the exercise day. The magnitude of this elevation in SMR was similar after the LI and HI exercise and averaged 0.35 kJ/min (6.7%). These data are in accordance with the findings of others, showing that an acute bout of exercise increases resting (or sleeping) metabolic rate.21,34 Although this elevation in metabolic rate seems to be only minor, the development of obesity is often also characterised by minimal deviations between energy expenditure and energy intake over prolonged periods of time. Furthermore, it has been shown that a 10% lower SMR is a predictor for future weight gain.35 Therefore, the effect of exercise on sleeping metabolic rate might be significant in the prevention and/or treatment of obesity, although the amount of exercise in this protocol has to be taken into account. The PAt for both exercise intensities was around 2.2 which is at the upper extreme of the PAt distribution.36 This relatively high level of exercise for sedentary obese subjects could certainly have contributed to the positive effect on SMR compared to some of the negative studies in the past, which were at a lower total workload or in trained subjects.

The 24 h EE was not significantly affected by exercise intensity. This is in contrast to the finding of Treuth et al.,25 who showed an increased 24 h EE with HI compared to LI exercise. However, in their study, energy expended during exercise was higher with the HI protocol, even though similar amounts of work were performed, indicating a reduced mechanical efficiency.35 It has indeed been shown that with increasing exercise intensity, mechanical efficiency is reduced, probably due to the increased dependence on inefficient fast-twitch fibres.3,37 In our study, in obese subjects, we did not find any difference in total energy expenditure during the exercise bouts, indicating no differences in energy efficiency between the HI and LI exercise. Furthermore, we found no effect of exercise intensity on postexercise energy expenditure in the present study. Therefore, in accordance with the study of Melanson,26 our study shows that exercise intensity does not influence 24 h EE.

Perhaps more important, apart from an effect on energy metabolism, is the effect of exercise on substrate oxidation. We hypothesised that exercise intensity would differentially influence 24 h substrate oxidation. It is well known that at low exercise intensities, fat oxidation is responsible for the major part of the energy demands and that with increasing exercise intensity the contribution of carbohydrate oxidation increases. It has been shown that maximal fat oxidation is achieved between 40 and 60% Wmax.24 Therefore, LI exercise has been considered the most beneficial for the treatment of obesity. However, LI exercise only has minor influence on postexercise fat oxidation. According to the two-compartment model of Fiati, fat oxidation can be increased by (1) expansion of the adipose tissue mass or (2) maintaining glycogen stores in a lower range.38 Regarding the latter, it is well known that HI exercise will lead to reduction of the glycogen stores, and would thus allow for a higher fat oxidation postexercise. Indeed, we recently showed that 24 h fat oxidation could be increased 1.5-fold by performing glycogen lowering exercise in both lean25 and obese subjects.33 In these studies, we found a negative
correlation between carbohydrate balance during the first 12 h in a respiration chamber and the subsequent 24 h fat oxidation. These results illustrate the impact of the glycogen stores on 24 h fat oxidation. In the present study, we were just underpowered to detect a significant effect on a lower postexercise RQ after HI exercise. However, the subsequent night RQ as well as the 24 h RQ were not different suggesting that the decrease in muscle glycogen in the present study was insufficient to trigger an increase in 24 h fat oxidation. Therefore, the results indicate that over a relative large range of exercise intensities (up to 80% VO\textsubscript{2max}) 24 h substrate utilisation after an acute exercise load is not affected. Only at even higher intensity exercise (upto 100% VO\textsubscript{2max}) and with glycogen depletion, an effect on substrate utilisation is observed, as shown by the studies of Schrauben et al.\textsuperscript{22,23} and Treuth et al.\textsuperscript{25} In this context, in our study in the HI exercise protocol subjects exercised only 50% of the exercised-time at 80% VO\textsubscript{2max} probably to little to result in changes in glycogen levels.

The negative results from this and other well-controlled 24 h EE studies\textsuperscript{26} of the effects of acute HI or LI exercise bouts on substrate utilisation seems to be in contrast to the recently published training study of van Aggel et al.\textsuperscript{29} showing a 40% increase in fat oxidation during exercise (but not at rest) after a LI (40% VO\textsubscript{2max}) training protocol compared to a HI (70% VO\textsubscript{2max}) training protocol. Case-controls studies examining the effect of participation in vigorous physical activities consistently showed a favouring effect of HI exercise on body fatness.\textsuperscript{40,41} as well as on postprandial fat oxidation.\textsuperscript{42} One possible explanation why in the 24 h respiration chamber studies, in contrast to training and case-control studies, no effect of exercise intensity on substrate metabolism is observed, might be the acute character of the exercise in the chamber studies. In the 24 h EE studies, nontrained lean or obese subjects, not habituated to perform such exercise bouts, were studied. Based on the similar results coming out of these studies it seems that the intensity level of an acute exercise bout as such does not contribute to the 24 h substrate oxidation pattern. Only regular training or daily vigorous physical activity does have an effect on muscle metabolism leading to a higher reliance on fat as energy substrate at a similar level of exercise intensity.\textsuperscript{5} This change in substrate preference is only observed during exercise, most likely since at rest, muscle metabolism already almost completely relies on fat as a substrate. This is an important argument for maintaining exercise adherence over time.\textsuperscript{43} Further well-controlled respiration chamber studies are needed to test whether regular exercising or physical activity at HI or LI affects 24 h substrate utilisation.

In conclusion in obese men, both LI and HI exercise increased SMR but no difference in 24 h EE was observed. Similarly, the differences in HI and LI exercise RQ was compensated postexercise leading to similar substrate oxidation patterns over 24 h independently of the level of exercise intensity.

Acknowledgements
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