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

Citation for published version (APA):

Saris, W. H., & Schrauwen, P. (2004). Substrate oxidation differences between high- and low-intensity exercise are compensated over 24 hours in obese men. *International Journal of Obesity*, 28(6), 759-65. <https://doi.org/10.1038/sj.ijo.0802631>

Document status and date:

Published: 01/01/2004

DOI:

[10.1038/sj.ijo.0802631](https://doi.org/10.1038/sj.ijo.0802631)

Document Version:

Publisher's PDF, also known as Version of record

Please check the document version of this publication:

- A submitted manuscript is the version of the article upon submission and before peer-review. There can be important differences between the submitted version and the official published version of record. People interested in the research are advised to contact the author for the final version of the publication, or visit the DOI to the publisher's website.
- The final author version and the galley proof are versions of the publication after peer review.
- The final published version features the final layout of the paper including the volume, issue and page numbers.

[Link to publication](#)

General rights

Copyright and moral rights for the publications made accessible in the public portal are retained by the authors and/or other copyright owners and it is a condition of accessing publications that users recognise and abide by the legal requirements associated with these rights.

- Users may download and print one copy of any publication from the public portal for the purpose of private study or research.
- You may not further distribute the material or use it for any profit-making activity or commercial gain
- You may freely distribute the URL identifying the publication in the public portal.

If the publication is distributed under the terms of Article 25fa of the Dutch Copyright Act, indicated by the "Taverne" license above, please follow below link for the End User Agreement:

www.umlib.nl/taverne-license

Take down policy

If you believe that this document breaches copyright please contact us at:

repository@maastrichtuniversity.nl

providing details and we will investigate your claim.

PAPER

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

WHM Saris^{1*} and P Schrauwen¹

¹Nutrition and Toxicology Research Institute Maastricht (NUTRIM), Maastricht University, Maastricht, The Netherlands

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, $W_{\max}$: 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% $W_{\max}$)) or LI (three times 60 min continuously at 38% $W_{\max}$) protocol with an equicaloric 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.

International Journal of Obesity (2004) **28**, 759–765. doi:10.1038/sj.ijo.0802631

Published online 30 March 2004

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.¹ 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.² 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.^{3,4} 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 focussed 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.⁵ 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

*Correspondence: Dr WHM Saris, Nutrition and Toxicology Research Institute Maastricht (NUTRIM), Maastricht University, P.O. Box 616, 6200 MD Maastricht, The Netherlands.
E-mail: W.Saris@hb.unimaas.nl

Received 16 September 2003; revised 6 January 2004; accepted 8 February 2004; published online 30 March 2004

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.⁶ 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 training⁷⁻⁹ whereas others found no effect.¹⁰⁻¹² 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,¹³⁻¹⁵ whereas others found no difference,^{16,17} 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.^{18,19} Therefore, when dealing with the optimal exercise intensity to prevent weight (re-)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.²⁰ In contrast, we and others have shown that a single exercise bout can rapidly increase fat oxidation for up to 36 h postexercise in both lean^{21,22} and obese subjects.²³ 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,²⁴ 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.²² 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,^{25,26} although an increased 24 h energy expenditure after HI exercise was found in the study of Treuth *et al.*²⁵ 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 equicaloric 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 (W_{max}) (HI) or three times 60 min at 38% W_{max} (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 (VO_{2max}) and maximal power output (W_{max}).

Table 1 Subjects' characteristics (n = 8)

	mean $\pm$ s.e.m.
Age (y)	38.3 $\pm$ 1.1
Height (m)	1.76 $\pm$ 0.02
Weight (kg)	96.9 $\pm$ 4.6
Percent body fat (%)	32.3 $\pm$ 1.3
BMI (kg/m ²)	31.2 $\pm$ 1.3
W_{max} (w)	235 $\pm$ 16
VO_{2max} (l/min)	3.3 $\pm$ 0.2

VO_{2max} : 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 Sormedics 2900 metabolic cart (Anaheim, USA). Heart rate was registered continuously using a Polar Sporttester (Kempele, Finland). In each individual $W_{\max}$ was calculated from

$$W_{\max} = W_{\text{out}} + (t/150) * \partial W$$

in which W_{out} is the highest workload completed by the subject, t is the time (in s) performed on the last workload and ∂W 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% $W_{\max}$. In the HI protocol subjects cycled for 30 min at alternatively 80 and 50% $W_{\max}$ for 2.5 min periods. During the HI protocol all exercise periods were preceded by 5 min warming up at 33% $W_{\max}$ and were ended by 5 minutes cooling down, also at 33% $W_{\max}$. 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 (Volugraph 2000, Mijndhardt). 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 m³ 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 70–80 l/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 O₂ analyser (Hartmann & Braun, type Magnos G6, Germany) and an infrared CO₂ 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 O₂ consumption and CO₂ 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 H₂SO₄ 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 am. 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 2 h after each exercise bout. Carbohydrate, fat and protein oxidation were calculated using O₂-consumption, CO₂-production and urinary nitrogen losses with the equations of Brouwer.³¹

protein oxidation (g/day) = 6.25 * N

fat oxidation (g/day) = 1.718 * VO₂ - 1.718 * VCO₂ - 0.315 * P

carbohydrate oxidation (g/day) = 4.17 * VCO₂ - 2.965 * VO₂ - 0.390 * 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 $\pm$ 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 $37 \pm 4\%$ of energy as fat, $43 \pm 4\%$ of energy as carbohydrate and $17 \pm 1\%$ of energy as protein. Maximal power output (W_{max}) was 235 ± 16 W, and maximal oxygen consumption (VO_{2max}) was 3.3 ± 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 was 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 24h 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 $2.9 \pm 1.5\%$. Physical activity index ($PAI = 24H$ EE/SMR) was not significantly different between the both protocols (Table 3).

The 24h 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 $2.1 \pm 1.8\%$. Respiration quotient during the exercise period was significantly lower during LI compared to HI

Table 2 The 24h-energy intake, energy expenditure and energy balance as measured in the respiration chamber $n=8$ (mean $\pm$ s.e.m)

Protocol	Intake (MJ)	Expenditure (MJ)	Balance (MJ)
LI	16.62 ± 0.53	16.69 ± 0.63	-0.07 ± 0.43
HI	16.62 ± 0.54	16.75 ± 0.62	-0.13 ± 0.21

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

Table 3 SMR (from 0000–0800) and PAI as measured in the respiration chamber (mean $\pm$ s.e.m)

Protocol	SMR night 1 (kJ/min)	SMR night 2 (kJ/min)	PAI ^a
LI	5.07 ± 0.21	$5.44 \pm 0.22^*$	2.22 ± 0.05
HI	5.20 ± 0.20	$5.53 \pm 0.21^*$	2.19 ± 0.06

^aPhysical activity index: 24h EE/24h SMR. * $P < 0.05$ compared to night 1.

Table 4 RQ as measured in the respiration chamber

Protocol	24h RQ	RQ night 1	RQ night 2	Exercise RQ	Postexercise RQ
LI	0.89 ± 0.01	0.85 ± 0.01	0.84 ± 0.01	$0.91 \pm 0.00^*$	$0.90 \pm 0.01^†$
HI	0.89 ± 0.01	0.83 ± 0.01	0.83 ± 0.01	0.93 ± 0.01	0.87 ± 0.02

* $P < 0.05$ compared to HI. $†P = 0.06$ compared to HI.

Figure 2 The 24h substrate balances (mean $\pm$ 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.³² 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 others 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 utilisation 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 PAI for both exercise intensities was around 2.2 which is at the upper extreme of the PAI 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.²⁵ 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.^{6,37} 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% $W_{\max}$.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 Flatt, 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 lean²² and obese subjects.²³ 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% $\text{VO}_{2\text{max}}$) 24 h substrate utilisation after an acute exercise load is not affected. Only at even higher intensity exercise (upto 100% $\text{VO}_{2\text{max}}$) and with glycogen depletion, an effect on substrate utilisation is observed, as shown by the studies of Schrauwen *et al.*^{22,23} and Treuth *et al.*²⁵ In this context, in our study in the HI exercise protocol subjects exercised only 50% of the exercised-time at 80% $\text{VO}_{2\text{max}}$, probably to little to result in changes in glycogen levels.

The negative results from this and other well-controlled 24 h EE studies²⁶ 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.*³⁹ showing a 40% increase in fat oxidation during exercise (but not at rest) after a LI (40% $\text{VO}_{2\text{max}}$) training protocol compared to a HI (70% $\text{VO}_{2\text{max}}$) 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,^{40,41} as well as on postprandial fat oxidation.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.² 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.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

We thank Marjolein van Gaalen and Sander van Vught for their assistance in collecting data.

References

- Schrauwen P, Westerterp KR. The role of high-fat diets and physical activity in the regulation of body weight. *Br J Nutr* 2000; 84: 417–427.
- Saris WHM. Physical activity and body weight regulation. In: Bouchard C, Bray GA (eds) *Regulation of body weight: Biological and behavioral mechanisms*. John Wiley & Sons Ltd: New York; 1996. pp 135–148.
- Ballor DL, Keeseey RE. A meta-analysis of the factors affecting exercise-induced changes in body mass, fat mass and fat-free mass in males and females. *Int J Obes Relat Metab Disord* 1991; 15: 717–726.
- van Baak MA, van Mil E, Astrup AV, Finer N, Van Gaal LF, Hilsted J, Kopelman PG, Rossner S, James WP, Saris WH, STORM Study Group. Leisure-time activity is an important determinant of longterm weight maintenance after weightloss in the sibutramine trial on obesity reduction and maintenance (Strom trial). *Am J Clin Nutr* 2003; 78: 209–214.
- Saris WHM, Blair SN, van Baak MA, Eaton SB, Davies PSW, Di Pietro L, Fogelholm M, Rissanen A, Schoeller D, Swinburn B, Tremblay A, Westerterp KR, Wyatt H. How much physical activity is enough to prevent unhealthy weight gain? Outcome of the IASO 1st Stock Conference and Consensus Statement. *Obes Rev* 2003; 4: 101–114.
- Hunter GR, Weinsier RL, Bamman MM, Larson DE. A role for high intensity exercise on energy balance and weight control. *Int J Obes Relat Metab Disord* 1998; 22: 489–493.
- Poehlman ET, Melby CL, Bradylak SF, Calles J. Resting metabolic rate and postprandial thermogenesis in highly trained and untrained males. *Am J Clin Nutr* 1988; 47: 793–798.
- Tremblay A, Fontaine E, Poehlman ET, Mitchell D, Perron L, Bouchard C. The effect of exercise-training on resting metabolic rate in lean and obese moderately obese individuals. *Int J Obes Relat Metab Disord* 1986; 10: 511–517.
- Pratley R, Nicklas B, Rubin M, Miller J, Smith A, Smith M, Hurley B, Goldberg A. Strength training increases resting metabolic rate and norepinephrine levels in healthy 50- to 65-yr-old men. *J Appl Physiol* 1994; 76: 133–137.
- Schulz LO, Nyomba BL, Alger S, Anderson TE, Ravussin E. Effect of endurance training on sedentary energy expenditure measured in a respiratory chamber. *Am J Physiol Endocrinol Metab* 1991; 260: E257–E261.
- Davis JR, Tagliaferro AR, Kertzer R, Gerardo T, Nichols J, Wheeler J. Variations in dietary-induced thermogenesis and body fatness with aerobic capacity. *Eur J Appl Physiol* 1983; 50: 319–329.
- Westerterp KR, Meijer GAL, Schoffelen P, Janssen E. Body mass, body composition and sleeping metabolic rate before, during and after endurance training. *Eur J Appl Physiol* 1994; 69: 203–208.
- Gore CJ, Whithier RT. The effect of exercise intensity and duration on the oxygen deficit and excess post-exercise oxygen consumption. *Eur J Appl Physiol* 1990; 60: 169–174.
- Phelain JF, Reinke E, Harris MA, Melby CL. Postexercise energy expenditure and substrate oxidation in young women resulting from exercise bouts of different intensity. *J Am Coll Nutr* 1997; 16: 140–146.
- Smith J, Naughton L. The effects of intensity of exercise on excess postexercise oxygen consumption and energy expenditure in moderately trained men and women. *Eur J Appl Physiol* 1993; 67: 420–425.
- Sedlock DA, Fissinger JA, Melby CL. Effect of exercise intensity and duration on postexercise energy expenditure. *Med Sci Sports Exerc* 1989; 21: 662–666.

- 17 Sedlock DA. Effect of exercise intensity on postexercise energy expenditure in women. *Br J Sports Med* 1991; 25: 38–40.
- 18 Astrup A, Buemann B, Christensen NJ, Toubro S. Failure to increase lipid oxidation in response to increasing dietary fat content in formerly obese women. *Am J Physiol* 1994; 266: E592–E599.
- 19 Blaak EE, Baak MAV, Kemerink GJ, Pakbiers MTW, Heidendal GAK, Saris WHM. β -Adrenergic stimulation of energy expenditure and forearm skeletal muscle metabolism in lean and obese men. *Am J Physiol Endocrinol Metab* 1994; 267: E306–E315.
- 20 Schrauwen P, van Marken Lichtenbelt WD, Saris WH, Westerterp KR. Changes in fat oxidation in response to a high-fat diet. *Am J Clin Nutr* 1997; 66: 276–282.
- 21 Bielinski R, Schutz Y, Jéquier E. Energy metabolism during the postexercise recovery in man. *Am J Clin Nutr* 1985; 42: 69–82.
- 22 Schrauwen P, van Marken Lichtenbelt WD, Saris WH, Westerterp KR. Role of glycogen-lowering exercise in the change of fat oxidation in response to a high-fat diet. *Am J Physiol* 1997; 273: E623–E629.
- 23 Schrauwen P, Lichtenbelt WD, Saris WH, Westerterp KR. Fat balance in obese subjects: role of glycogen stores. *Am J Physiol* 1998; 274: E1027–1033.
- 24 Romijn JA, Coyle EF, Sidossis LS, Gastaldelli A, Horowitz JF, Enderit E, Wolfe RR. Regulation of endogenous fat and carbohydrate metabolism in relation to exercise intensity and duration. *Am J Physiol Endocrinol Metab* 1993; 265: E380–E391.
- 25 Treuth MS, Hunter GR, Williams M. Effects of exercise intensity on 24h-energy expenditure and substrate oxidation. *Med Sci Sports Exerc* 1996; 28: 1138–1144.
- 26 Melanson EL, Sharp TA, Seagle HM, Horton TJ, Donahoo WT, Grunwald GK, Hamilton JT, Hill JO. Effect of exercise intensity on 24-h energy expenditure and nutrient oxidation. *J Appl Physiol* 2002; 92: 1045–1052.
- 27 Stichting-Nederlands-Voedingsstoffenbestand. *NEVO Tabel*, Voorlichtingsbureau voor de voeding, Den Haag, 1993. [in Dutch].
- 28 Siri WE. The gross composition of the body. *Adv Biol Med Physiol* 1956; 4: 239–280.
- 29 Schoffelen PF, Westerterp KR, Saris WH, Ten Hoor F. A dual-respiration chamber system with automated calibration. *J Appl Physiol* 1997; 83: 2064–2072.
- 30 Weir JB. New methods for calculating metabolic rate with special reference to protein metabolism. *J Physiol* 1949; 109: 1–9.
- 31 Brouwer E. On simple formulae for calculating the heat expenditure and the quantities of carbohydrate and fat oxidized in metabolism of men and animals, from gaseous exchange (oxygen intake and carbonic acid output) and urine-n. *Acta Physiol Pharmacol Neerlandica* 1957; 6: 795–802.
- 32 Saris WHM. Physical inactivity and metabolic factors as predictors of weight gain. *Nutr Rev* 1996; 54: S110–S115.
- 33 Treuth MS, Hunter GR, Weinsier RL, Kell SH. Energy expenditure and substrate utilization in older women after strength training: 24-h calorimeter results. *J Appl Physiol* 1995; 78: 2140–2146.
- 34 Bahr R, Ingnes I, Vaage O, Sejersted OM, Newsholme EA. Effect of duration of exercise on excess postexercise O_2 consumption. *J Appl Physiol* 1987; 62: 485–490.
- 35 Ravussin E, Lilloja S, Knowler WC, Christin L, Freymond D, Abbott WG, Boyce V, Howard BV, Bogardus C. Reduced rate of energy expenditure as a risk factor for body-weight gain. *N Engl J Med* 1988; 318: 467–472.
- 36 Westerterp KR. Alterations in energy balance with exercise. *Am J Clin Nutr* 1998; 68: 970S–974S.
- 37 Gaesser GA, Brooks GA. Muscular efficiency during steady-state exercise: effects of speed and work rate. *J Appl Physiol* 1975; 38: 1132–1139.
- 38 Flatt JP. The difference in the storage capacities for carbohydrate and for fat, and its implications in the regulation of body weight. *Ann N Y Acad Sci* 1987; 499: 104–123.
- 39 Van Aggel-Leijssen DP, Saris WH, Wagenmakers AJ, Senden JM, Van Baak MA. Effect of exercise training at different intensities on fat metabolism of obese men. *J Appl Physiol* 2002; 92: 1300–1309.
- 40 Tremblay A, Despres JP, Leblanc C, Craig CL, Ferris B, Stephens T, Bouchard C. Effect of intensity of physical activity on body fatness and fat distribution. *Am J Clin Nutr* 1990; 51: 153–157.
- 41 Dionne I, Almeras N, Bouchard C, Tremblay A. The association between vigorous physical activities and fat deposition in male adolescents. *Med Sci Sports Exerc* 2000; 32: 392–395.
- 42 Yoshioka M, Doucet E, St-Pierre S, Almeras N, Richard D, Labrie A, Despres JP, Bouchard C, Tremblay A. Impact of high-intensity exercise on energy expenditure, lipid oxidation and body fatness. *Int J Obes Relat Metab Disord* 2001; 25: 332–339.
- 43 Wing RR, Jackicic JM. Changing lifestyle: moving from sedentary to active. In: Bouchard C (ed) *Physical activity and obesity*. Human Kinetics: Champaign, IL; 2000. pp 372–389.