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ORIGINAL ARTICLE

Low-volume high-intensity swim training is superior to high-volume low-intensity training in relation to insulin sensitivity and glucose control in inactive middle-aged women

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Abstract

Purpose We tested the hypothesis that low-volume high-intensity swimming has a larger impact on insulin sensitivity and glucose control than high-volume low-intensity swimming in inactive premenopausal women with mild hypertension.

Methods Sixty-two untrained premenopausal women were randomised to an inactive control ($n = 20$; CON), a high-intensity low-volume ($n = 21$; HIT) or a low-intensity high-volume ($n = 21$; LIT) training group. During the 15-week intervention period, HIT performed 3 weekly 6–10 × 30-s all-out swimming intervals (average heart rate (HR) = 86 ± 3 % HR_{max}) interspersed by 2-min recovery periods and LIT swam continuously for 1 h at low intensity (average HR = 73 ± 3 % HR_{max}). Fasting blood samples were taken and an oral glucose tolerance test (OGTT) was conducted pre- and post-intervention.

Results After HIT, resting plasma [insulin] was lowered (17 ± 34 %; $P < 0.05$) but remained similar after LIT and CON. Following HIT, 60-min OGTT plasma [insulin] and [glucose] was lowered (24 ± 30 % and 10 ± 16 %; $P < 0.05$) but remained similar after LIT and CON. Total area under the curve for plasma [glucose] was lower ($P < 0.05$) after HIT than LIT (660 ± 141 vs. 860 ± 325 mmol min L^{-1}). Insulin sensitivity (HOMA-IR) had increased ($P < 0.05$) by 22 ± 34 % after HIT, with no significant change after LIT or CON, respectively. Plasma soluble intracellular cell adhesion molecule 1 was lowered ($P < 0.05$) by 4 ± 8 and 3 ± 9 % after HIT and CON, respectively, while plasma soluble vascular cell adhesion molecule 1 had decreased ($P < 0.05$) by 8 ± 23 % after HIT only.

Conclusions These findings suggest that low-volume high-intensity intermittent swimming is an effective and time-efficient training strategy for improving insulin sensitivity, glucose control and biomarkers of vascular function in inactive, middle-aged mildly hypertensive women.

Abbreviations

ANOVA	Analysis of variance
CON	Control
HIT	High-intensity training
HOMA-IR	Homeostatic model assessment of insulin resistance
HR	Heart rate
OGTT	Oral glucose tolerance test
LIT	Low-intensity training
SEM	Standard error of the mean
sICAM-1	Soluble intracellular cell adhesion molecule 1

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sVCAM-1	Soluble vascular cell adhesion molecule 1
T2DM	Type 2 diabetes mellitus
tAUC	Total area under the curve

Introduction

Lack of physical activity is considered a global pandemic and one of the most serious public health problems of the 21st century (Kohl et al. 2012). Physical inactivity is an independent risk factor for major chronic diseases and the annual global mortality attributable to physical inactivity is estimated at approximately 3.2 million (World Health Organization 2016). Type 2 diabetes mellitus (T2DM), an example of a pathological condition associated with an inactive lifestyle, shows an increasing growth rate, imposing a significant health and economic burden on the health care system. In the US alone, the incidence of T2DM increased 117 % from 1980 to 2011 (Al Tunaiji et al. 2014). The population attributable fraction of T2DM related to physical inactivity is reported to be higher in women than in men (Al Tunaiji et al. 2014).

Women experience a decline in health status in the years following the menopause (Appelman et al. 2015), including a higher prevalence of insulin resistance, which plays a major role in the pathophysiology of T2DM (Rizzoli et al. 2014; Shufelt et al. 2015). In addition, there is evidence that a relationship exists between insulin resistance and markers of endothelial dysfunction which also contributes to cardiovascular disease (Chen et al. 2000; Kim et al. 2006). Two markers of endothelial dysfunction that have been shown to predict cardiovascular disease include soluble intracellular cell adhesion molecule 1 (sICAM-1] and soluble vascular cell adhesion molecule 1 [sVCAM-1] (Weyer et al. 2002), which are mediators of platelet rolling and cell attachment (Marsh and Coombes 2005). It is well known that exercise training has a preventive effect on the development of lifestyle-related deficiencies (Chedraui and Perez-Lopez 2013); thus, exercise-interventions targeting an improvement in glucose control, insulin sensitivity and endothelial function are clearly warranted for inactive middle-aged women approaching the menopause period.

High-intensity interval training protocols have garnered attention in recent years as a time-efficient exercise option for improving metabolic health, and they may be particularly effective for improving blood glucose homeostasis in individuals with, or at risk of, T2DM (Little and Francois 2014). In a recent study by Little and Francois (2014), high-intensity interval cycling induced greater and longer-lasting effects on postprandial blood glucose control than continuous moderate-intensity exercise in obese middle-aged adults. Moreover, 10×1 min of high-intensity intermittent cycling caused a reduction in insulin resistance

measured using the homeostatic model assessment of insulin resistance (HOMA-IR) in inactive women (Durrer et al. 2015), which is supported by studies on patients with T2DM (Shaban et al. 2014) and non-diabetic subjects (Adams 2013). These studies, and the vast majority of studies on the health effects of high-intensity training regimes have deployed running or cycling exercise, while a limited number of studies have deployed exercise modalities such as swimming, which involves a significant taxation of the upper body.

Swimming is a popular physical leisure activity among the female population and may be considered a good choice of exercise training for obese, middle-aged and elderly individuals due to the minimal weight-bearing stress involved. In addition, swimming highly engages the upper-body muscles, where the adaptive capacity of the skeletal muscle has shown to be higher than the lower limbs due to the bipendular nature of humans (Nordsborg et al. 2015). For example, low-intensity swimming has been shown to be more efficient for improving insulin resistance than walking at a matched intensity in 50- to 70-year-old sedentary women (Cox et al. 2010). In addition, in obese mice (Motta et al. 2015) and rats (Song et al. 2014) swimming has been demonstrated to facilitate glucose control and insulin sensitivity. Moreover, 1 year of participation in low-intensity swimming has been demonstrated to increase insulin sensitivity in young hypertensive patients (Chen et al. 2010). However, limited information exists on the impact of high-intensity intermittent swimming, and, as far as we know, no studies have compared the effects of different swimming protocols on insulin resistance and blood glucose control in middle-aged, inactive women.

Thus, the objective of the present study was to investigate the effects of low-volume high-intensity swimming compared to high-volume low-intensity swimming, on insulin sensitivity and blood glucose control in middle-aged, inactive women with mild hypertension. It was hypothesised that short-term high-intensity interval swimming is a more efficient training method than prolonged, continuous low-intensity swimming for improving metabolic fitness in untrained middle-aged mildly hypertensive women.

Materials and methods

Participants

Sixty-two inactive premenopausal women with mild to moderate arterial hypertension were recruited for the study. The subjects were selected from 262 volunteers based on training history, medication, blood pressure and body mass index. A total of 83 participants were recruited

in the original sample. Sixty-two took part in the present study, for which the cardiovascular data are presented in Mohr et al. (2014b) and 21 were randomly assigned to a football group as part of another study (Mohr et al. 2014a). Data for the adaptive capacity of skeletal muscle for the above-mentioned participants were also presented in Nordsborg et al. (2015). In addition, the control group (20 participants) in the present study was also the controls in the above-mentioned studies (Mohr et al. 2014a, b; Nordsborg et al. 2015) The study was approved by the ethical committee of the Faroe Islands as well as the Sport and Health Sciences Research Ethics Committee at the University of Exeter, Exeter, United Kingdom, and conducted in accordance with the Declaration of Helsinki (1964). After being informed verbally and in writing of the experimental procedures and associated risks, all participants gave their written consent to take part in the study.

Experiment design

The study was designed as a randomised controlled trial. After initial screening of the 262 volunteers, 62 participants were enrolled in the present study based on the selection criteria: an inactive lifestyle for the last 2 years; mild to moderate hypertension (mean arterial pressure 96–110 mmHg); and body mass index >25. Participants undergoing treatment with adrenergic beta-antagonists were excluded. Participants using diuretics and ACE inhibitors ($n = 4$) were not excluded from the study, but none of the four subjects changed their medication during the intervention period. The participants were randomised into a high-intensity intermittent swim-training group (HIT: age; 44 ± 5 ($\pm$SD) (range 36–49) years, height; 164 ± 6 cm, weight; 76.5 ± 8.8 kg; $n = 21$), a low-intensity continuous swimming group (LIT: age; 46 ± 4 (38–48) years, height; 165 ± 5 cm, weight; 83.8 ± 18.8 kg; $n = 21$) and a control group (CON: age; 45 ± 4 (35–48) years, height; 166 ± 6 cm, weight; 77.3 ± 10.4 kg; $n = 20$). The randomization process first separated the participants in groups and thereafter determined the type of intervention. Both steps were carried out in random and blinded conditions. The training groups took part in two types of swim training involving three training sessions per week for 15 weeks, while CON had no training or lifestyle changes in the same period. There were no drop-outs from the study, but one subject in the LIT group suffered aquatic phobia and was therefore moved to CON after the randomization process. The participants also performed an oral glucose tolerance test (OGTT) and had a resting blood sample taken. Dietary intake was not controlled during the training period and the testing periods were not timed in relation to the menstrual cycle.

Training intervention

The HIT participants completed a total of 44 ± 1 (39–50) training sessions over the 15-week intervention period, corresponding to 2.9 ± 0.1 (2.6–3.3) sessions per week. Each session lasted ~15–25 min (3–5 min of effective swimming) and consisted of 6–10 30-s all-out free-style swimming (front crawl) intervals interspersed by 2 min of passive recovery according to training principles previously described (Iaia et al. 2009; Mohr et al. 2007). In the first 6 weeks of training each comprised 6 intervals, the following 6 weeks comprised 8 intervals and the final 3 weeks comprised 10 intervals. The LIT group completed a total of 43 ± 1 (37–49) training sessions over 15 weeks, corresponding to 2.9 ± 0.1 (2.5–3.3) training sessions per week. All LIT training sessions lasted 1 h and consisted of continuous front-crawl swimming, with the participants encouraged to swim as far as possible in every session. Five trained swimming coaches were present during all training sessions to give technical advice, control the intensity and duration of the training, and secure a safe training environment. There were no injuries from the swim-training during the 15 wks. Heart rate was measured during one training session in week 1 and one session in week 15 of the training intervention, and the swimming distance was noted in every session. Average mean and peak HR during HIT training in the first and last weeks of the intervention was 158 ± 5 and 176 ± 2 bpm, respectively, corresponding to 86 ± 3 and 96 ± 1 % HR_{max}, respectively, which was higher ($P < 0.05$) than average values in LIT (132 ± 4 and 144 ± 3 bpm equivalent to 73 ± 3 and 79 ± 1 % HR_{max}).

Resting blood sampling and OGTT testing

On the day of testing, the participants reported to the laboratory after transport by car or bus. No training was performed 48-72 h prior to the testing and participants were instructed to avoid physical activity the day prior to the OGTT tests. A resting blood sample was collected under standardised conditions from an antecubital vein between 7 and 8 a.m. after an overnight fast using venipuncture technique. The blood was rapidly centrifuged for 30 s and the plasma was collected and analysed by an automatic analyzer (Cobas Fara, Roche, France) using enzymatic kits (Roche Diagnostics, Germany) to determine the relevant variables. Furthermore, 100 µl of plasma was used to determine concentrations of sICAM-1 and sVCAM-1 by immunoassays (Elisa, R&D Systems, Minneapolis, MN) using an Emax precision microplate reader (Molecular Devices, Sunnyvale, CA, USA). The intra-assay coefficient of variations for sICAM-1 and sVCAM-1 are 4.9 ± 3.5 and 5.1 ± 5.9 %, respectively.

Also, an OGTT test was performed as previously described (Durrer et al. 2015), with blood samples taken after 0, 30, 60 and 120 min and analysed for plasma [glucose] using enzymatic kits (Roche Diagnostics, Germany) and [insulin]. Insulin sensitivity was evaluated for all subjects using the homeostatic model assessment method (HOMA-IR); (Yokoyama et al. 2003). Changes in plasma [glucose] and [insulin] during the OGTT were quantified using total area under the curve (tAUC) analyses employing the trapezium rule (GraphPad Prism, San Diego, CA, USA). The intra-assay coefficient of variations for glucose and insulin are 3.0 ± 3.5 and 4.8 ± 3.6 %, respectively.

Statistical analysis

Data are presented as mean $\pm$ SD. Statistical analyses were performed using SPSS v.22. A two-factor mixed ANOVA design with the between factor 'group' (HIT versus LIT versus CON) and repeated factor 'condition' (pre-training versus post-training) was used to analyse all data apart from the 2-h OGTT. Responses to the OGTT were analysed using a three-factor mixed ANOVA design, with the factors 'group' (HIT versus LIT versus CON), 'condition' (pre-training versus post-training) and 'time' (0, 30, 60 and 120 min). When significant interactions or main effects were detected, data were subsequently analysed using Bonferroni post hoc t tests. The significance level was $P < 0.05$.

Results

Plasma [glucose] and [insulin]

Pre-intervention fasting plasma [glucose] was 4.9 ± 0.3, 5.8 ± 2.3, and 4.9 ± 0.5 mmol L^{-1} in HIT, LIT and CON, respectively, with a tendency for higher ($P = 0.07$) values in LIT than HIT. Fasting plasma [glucose] was unaltered after the intervention period (Fig. 1). Plasma [glucose] increased (main effect for time, $P < 0.05$) in all three groups after 30 and 60 min during the OGTT, but was normalised after 120 min pre- and post-training. In HIT, plasma [glucose] was 10 ± 16 % lower after 60 min post- compared to pre-intervention, with the concentration being lower (condition x time x group interaction, $P < 0.05$) than the corresponding concentration in LIT and tending ($P = 0.054$) to be lower than in CON (Fig. 1). For plasma [glucose] tAUC, there were no significant differences between groups prior to the training intervention (HIT: 700 ± 155, LIT: 833 ± 324 and CON: 764 ± 186 mmol min L^{-1}), respectively. However, following the training intervention HIT tAUC (660 ± 141 mmol min L^{-1}) was significantly lower than LIT tAUC (860 ± 325 mmol min L^{-1}, group–condition interaction, $P < 0.05$).
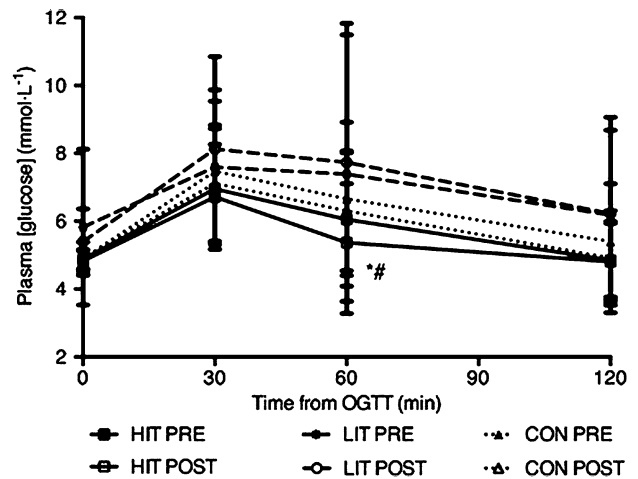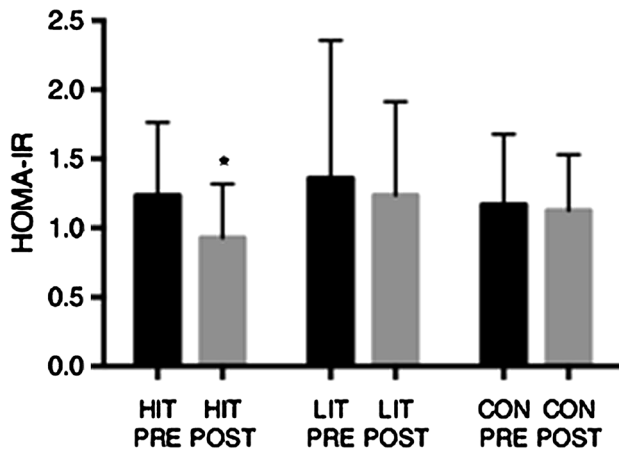

Fig. 1 Plasma [glucose] response following the OGTT displayed over time in HIT, LIT and CON before and after the 15-wk intervention. Data are mean $\pm$ SD. *Asterisk* denotes significant difference from pre intervention. *Hash* denotes significant difference from LIT. $P < 0.05$
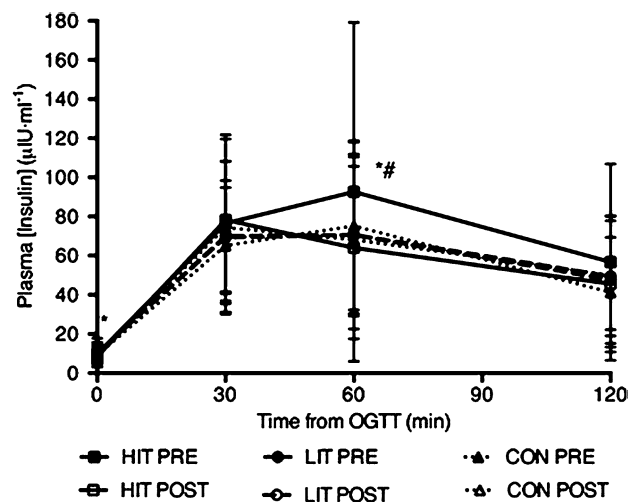
Fig. 2 Plasma [insulin] response following the OGTT displayed over time in HIT, LIT and CON before and after the 15-week intervention. Data are mean $\pm$ SD. *Asterisk* denotes significant difference from pre intervention. *Hash* denotes significant difference from LIT. $P < 0.05$

Pre intervention fasting plasma [insulin] was 9.6 ± 4.2, 10.2 ± 7.3 and 9.9 ± 4.6 µIU·ml^{-1} in HIT, LIT and CON, respectively, with no significant differences between groups (Fig. 2). A training-induced decrease of 17 ± 34 % in baseline plasma [insulin] occurred following HIT (group–condition interaction, $P < 0.05$), while levels remained constant after LIT and CON. Plasma [insulin] increased in all three groups during the OGTT, being higher than at baseline at 30-, 60- and 120-min pre- and post-intervention (main effect for time, $P < 0.05$, Fig. 2). However, plasma [insulin] was only lower (24 ± 30 %) at 60 min into the

Fig. 3 Insulin sensitivity (HOMA-IR) in HIT, LIT and CON before and after the 15-week intervention. Data are mean ± SD. *Asterisk* denotes significant difference from pre intervention. $P < 0.05$

OGTT following HIT which was lower than the corresponding value for LIT (condition x time x group interaction, $P < 0.05$). There was also a tendency ($P = 0.09$) for a lower concentration following HIT at the 120 min time point (Fig. 2). There were no differences between pre- and post-plasma [insulin] during the OGTT in LIT and CON (Fig. 2). For plasma [insulin] tAUC, there were no significant differences between groups prior to (HIT: 9121 ± 7606, LIT: 6879 ± 3621, and CON: 6947 ± 3116 μIU ml min^{-1}, respectively) and following (HIT: 6634 ± 3449, LIT: 6809 ± 2973, and CON: 6712 ± 2999 μIU ml min^{-1}, respectively) the training intervention. However, there was a tendency ($P = 0.09$) for plasma [insulin] tAUC to be lower after HIT.

Insulin sensitivity: HOMA-IR

Insulin sensitivity data are shown in Fig. 3. There were no significant differences in insulin sensitivity between HIT, LIT and CON pre intervention (HIT: 1.3 ± 0.5, LIT: 1.4 ± 1.0, and CON: 1.2 ± 0.5). Following the training-intervention, there was a 22 ± 34 % improvement in insulin sensitivity after HIT (group–condition interaction, $P < 0.05$), with no change in LIT and CON.

Plasma [sICAM-1] and [sVCAM-1]

There were no significant differences in plasma [sICAM-1] between HIT, LIT and CON groups pre intervention (146 ± 68, 142 ± 56 and 134 ± 67 ng ml^{-1}), but it was lowered (group–condition interaction, $P < 0.05$) by 4 ± 8 and 3 ± 9 % post-intervention after HIT and CON only, respectively (Fig. 4a). There were no significant differences
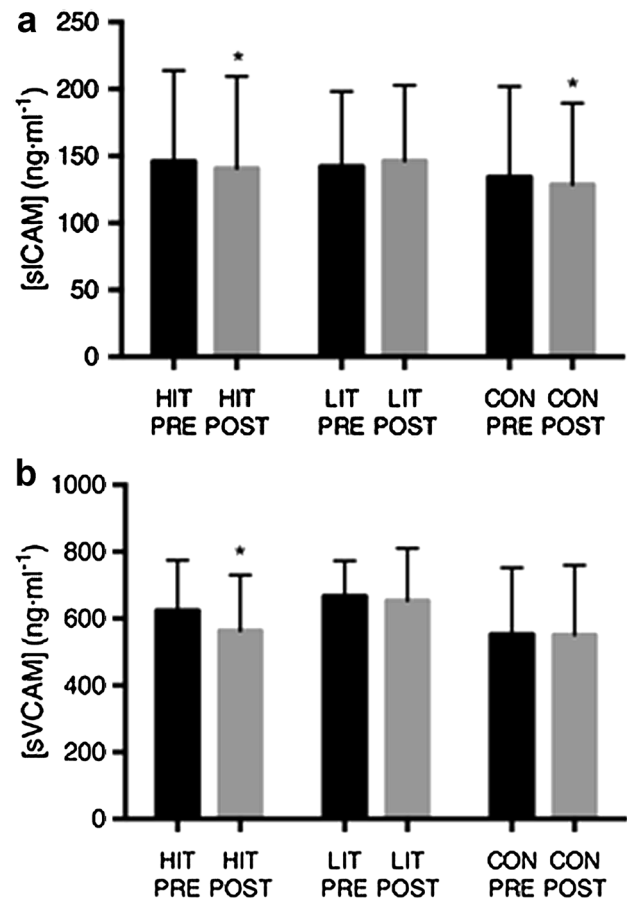
Fig. 4 Fasting plasma [sICAM-1] (**a**) and [sVCAM-1] (**b**) in HIT, LIT and CON before and after the 15-week intervention. Data are mean ± SD. *Asterisk* denotes significant difference from pre-intervention. $P < 0.05$

in plasma [sVCAM-1] between groups pre intervention (624 ± 154, 667 ± 106 and 553 ± 198 ng ml^{-1} in HIT, LIT and CON, respectively; Fig. 4b). However, plasma [sVCAM-1] was lower (group–condition interaction, $P < 0.05$) by 8 ± 23 % after HIT (Fig. 4b), with no change after LIT and CON.

Discussion

For the first time, the effects of low-volume high-intensity versus high-volume low-intensity swim training on insulin sensitivity and blood glucose control have been studied in middle-aged, inactive women. The major findings were that short-term high-intensity intermittent swim training lowered the fasting plasma [insulin], as well as plasma [glucose] and [insulin] during an oral glucose tolerance test, demonstrating an improvement in insulin sensitivity. In contrast, no changes occurred in those parameters

after prolonged low-intensity swimming despite a ~12-fold longer efficient swimming time and ~fivefold longer swimming distance covered during training.

Although previous literature on high-intensity swim training is lacking, Chen et al. (2010), Nualnim et al. (2012), and Cox et al. (2010) have implemented 3-12 months of moderate-intensity swim training for a combination of hypertensive and normotensive young (21 years), and older adults (>50 years) in which there were no significant improvements in insulin sensitivity, fasting [glucose], [insulin] or [glucose] AUC. In contrast the present study displayed a 22 % improvement in insulin sensitivity after HIT. However, it should be noted that fasting values for Nualnim et al. (2012) and the present study were already within the normal range. Interestingly, HIT in the present study decreased [glucose] values by 10 % at 60 min during the OGTT. Thus, HIT swimming may represent an attractive alternative to moderate-intensity swimming.

In the present study, 17 % decrease in fasting [insulin] was also reported for HIT along with a 24 % improvement at 60 min of the OGTT. The present findings are comparable with effects on metabolic health from other forms of high-intensity exercise interventions. For example, Little et al. (2011) demonstrated that 10×60 s of high intensity cycling over only 2 weaks reduced hyperglycemia and improved muscle mitochondrial capacity in patients with type 2 diabetes. Ciolac et al. (2010) found that 16 wks of high-intensity aerobic interval running 3 times per week (1 min running interspersed with 2 min walking for 40 min) for young females (20–30 years) at risk of hypertension resulted in a 35 % improvement in fasting [insulin]. In addition, Trapp et al. (2008) conducted 15 wks of high-intensity intermittent cycling 3 times per week (8-s all-out interspersed with 12 s of slow cycling for 60 repetitions) for inactive women (18–30 years) which resulted in a 31 % and 33 % improvement in fasting [insulin] and HOMA-IR score whereas the steady-state exercise group only resulted in a 9 % and 11 % improvement. The slightly lower improvement in fasting [insulin] in the present study compared to Ciolac et al. (2010) and Trapp et al. (2008) may be due to the exercise modality given that swimming differs in medium, position, breathing pattern and muscle groups used, providing a different training stimulus to cardiovascular parameters due to differences in ventricular volumes (Lazar et al. 2013; Sundstedt et al. 2003). It may also be speculated that the less pronounced systemic adaptation may be due to the smaller muscle mass activated during swimming compared to other forms of exercise (Nordsborg et al. 2015). Although the positive effect of high-intensity training on insulin sensitivity has been confirmed in numerous studies, the additional knowledge provided by this study is that high-intensity swimming provides a useful alterative for inactive middle-aged women compared to other modes of exercise.

On the other hand, not all high-intensity training interventions have reported insulin improvements. For example, Arad et al. (2015), Gillen et al. (2013) and Keating et al. (2015) found no change in fasting [insulin] and [glucose] and insulin sensitivity following 8–16 weeks of cycling interval training in overweight females (27–40 years). One reason for the discrepancy between these studies and the present study may be due to this study applying front-crawl training primarily taxing the upper-body muscles, which have a lower baseline training status than the legs and, hence, a higher improvement potential (Nordsborg et al. 2015).

Studies of swim training in rats have reported increased upregulation of molecules related to insulin signalling transduction in the skeletal muscle. For example, Fujimoto et al. (2010) previously reported that 5 days of non-exhaustive high-intensity intermittent swimming in male Sprague–Dawley rats provided an excellent stimulus for improving GLUT4 expression in skeletal muscle. In addition, in a study by Terada et al. (2001), male Sprague–Dawley rats performed short-term bouts of extremely high, relatively high and low intensity swimming over an 8-day period. GLUT4 content in the three training groups was increased following the exercise period, with no differences between groups. In support of these changes, Burgomaster et al. (2007) found increased muscle GLUT4 content after 1 week of sprint interval training, which remained higher during detraining of young (22 years) active men, which is supported by findings by Little et al. (2011). GLUT4 has been shown to be responsible for insulin-stimulated and contraction-induced glucose uptake (Daugaard and Richter 2001). As shown by Nordsborg et al. (2015), muscle glycogen in the deltoideus muscle of the study's participants increased by 63 % following HIT, which could strengthen the notion that muscular uptake of glucose increased via GLUT4 transporters.

While little research is available on [sICAM-1] and [sVCAM-1] following high-intensity swimming in middle-aged women, the improvement in vascular function as indicated by the 4 and 8 % reductions in [sICAM-1] and [sVCAM-1] following HIT is of interest as endothelial dysfunction is known to initiate not only the process of atherosclerosis, but also the development of insulin resistance (Glowinska et al. 2005). The present findings are not as notable as the improvements presented by Nyberg et al. (2014) following a 12-week floorball training intervention for premenopausal women ([sVCAM-1] pre: 674 and post: 535 ng ml^{-1}; [sICAM-1] pre: 166 and post: 122 ng ml^{-1}, however, this may be due to the exercise modality. On the other hand, the swimming data of the present study appears to be more promising than those of cycling based training, for example, Sabatier et al. (2008) failed to show a significant improvement in [sICAM-1] following a 14 week

cycling exercise intervention in healthy premenopausal women with a baseline [sICAM-1] of 169 ng ml^{-1}. In addition, although not significantly correlated (data not shown), the reductions in [sICAM-1] and [sVCAM-1] after HIT were also accompanied by improvements in glucose handling which was not the case following LIT, which is in agreement with previous studies incorporating continuous aerobic training (Padilla et al. 2008). As shear stress on endothelial cells lining conduit blood vessels increases in direct proportion to intensity (Padilla et al. 2008), it may be speculated that HIT has greater potential for improving endothelial function compared to LIT (Wisloff et al. 2009). The improvement in glucose handling following HIT may have been enhanced by vascular actions of insulin in endothelium leading to an increased stimulation of the production of nitric oxide (NO). It has previously been reported that the NO-dependent increase in vasodilatation and skeletal muscle blood flow accounts for 25–40 % of the increase in glucose uptake in response to insulin stimulation (Kim et al. 2006). It may therefore be postulated that to induce changes in [sVCAM-1] and [sICAM-1] improvements are likely to occur quicker when exercising at high intensity and quite possibly without the need to change the individual's diet.

Despite HIT only exercising for one-third of the time of LIT, similar changes in whole-body fat and lean body mass occurred, although leg lean mass increased by 6 % after HIT with no changes after LIT (data not shown). As the speed of swimming increases, drag forces increase exponentially, reducing the mechanical efficiency of front crawl by 5–9.5 % (Lazar et al. 2013). This may have increased the energy requirements of the lower limbs in HIT and, in turn, increased leg lean mass. It is also possible that the number of muscles recruited during HIT and LIT swimming differ, possibly causing a faster depletion of glycogen during HIT, and, together with increased circulating catecholamines, this may contribute to an increase in glucose utilisation and an improved insulin response (Vollestad and Blom 1985). The favourable change in body composition may also have been brought about by an increased capacity for fat oxidation. Previous research has shown that endurance exercise (Holloszy and Booth 1976) and high-intensity exercise (Talanian et al. 2007) enhance the capacity for fat oxidation and mitochondrial enzyme activity. This has been shown to improve the potential for muscle to utilise lipids as a substrate for energy and is also associated with improved insulin sensitivity (Goodpaster et al. 2003). It is interesting to note that, in this study, HIT participants of this study increased mitochondrial enzyme activity more than LIT (data published in Nordsborg et al. (2015)). If the training had continued for a longer period (6 months to 1 year), it is possible that the decrease in [insulin] would have led to higher fat oxidation and fat loss in the long

term (Ferrannini et al. 1997). However, it cannot be ruled out that changes in body composition could have been due to unreported changes in diet, as diet was not directly controlled. As stated by Chen et al. (2010), interventions that enhance insulin sensitivity can alleviate hypertension, and the findings of the present study, together with those of Nordsborg et al. (2015), may help explain the reduction in blood pressure in the present participants shown by Mohr et al. (2014b).

Conclusions

In conclusion, 15 weeks of low-volume high-intensity intermittent, but not prolonged low intensity high-volume, swim training improves insulin sensitivity and reduces biomarkers of endothelial activation in inactive premenopausal women with mild hypertension. Consequently, high-intensity intermittent swim training appears to be an appealing non-weight bearing training regime with a very low risk of injury.

Compliance with ethical standards

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