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Tabata Training Does Not Improve Maximal Oxygen Uptake in Well-trained Women.

Master's thesis in Exercise Physiology
Supervisor: Professor Jan Helgerud
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Kunnskap for en bedre verden

Abstract

Purpose: To compare the effects of 8 x 20 seconds supramaximal Tabata training (TT) to 4 x 4 minutes of high-intensity interval training (HIIT) at 90 - 95% of HR_{max} on well-trained women.

Methods: 12 healthy, well-trained female subjects were randomized to either TT (N = 7) or HIIT (N = 5). The subjects conducted three weekly supervised sessions for eight weeks. Before and after the intervention, maximal oxygen uptake ($\dot{V}O_{2max}$), running economy (RE), lactate threshold (LT), anaerobic capacity (AC) and performance on 300m and 3000m were tested. All tests and training were done running on a treadmill, except the performance tests which was done on an indoor track field.

Results: $\dot{V}O_{2max}$ improved significantly more following HIIT than following TT. HIIT improved $\dot{V}O_{2max}$ from 54.1 $mL \cdot kg^{-1} \cdot min^{-1}$ to 58.9 $mL \cdot kg^{-1} \cdot min^{-1}$, while no significant change was observed following TT. Both 300m and 3000m performance improved in both groups with no difference between groups. RE and AC did not change from pre- to post training in neither of the groups. Velocity at LT increased in both groups, while $\dot{V}O_2$ at LT increased following TT only.

Conclusion: It is concluded that TT is an ineffective strategy for improving $\dot{V}O_{2max}$ in already well-trained women. Furthermore, the 4 x 4 minutes HIIT protocol improved $\dot{V}O_{2max}$ significantly more than TT, indicating that HIIT is more effective than TT.

Keywords: High-intensity interval training, Anaerobic capacity, Maximal accumulated oxygen deficit, Lactate threshold, Anaerobic threshold, Running economy, Work economy, Tabata, Sprint interval training, Running performance, Maximal oxygen uptake.

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Acknowledgements

Abbreviations

AC: Anaerobic capacity

a-vO$_2$diff: Arterial-venous oxygen difference

ATP: Adenosine triphosphate

CK: Creatine Kinase

CSA: Cross-sectional area

Hb: Hemoglobin

HIIT: High-intensity interval training

HR: Heart rate

LDH: Lactate dehydrogenase

LT: Lactate threshold

Mb: Myoglobin

MCT: Moderate intensity continuous training

MCT1: Monocarboxylate transporter 1

MCT4: Monocarboxylate transporter 4

NTNU: Norwegian University of Science and Technology

O$_2$ pulse: Oxygen pulse

PCr: Phosphocreatine

PFK: Phosphofructokinase

$\dot{Q}$: Cardiac output

RE: Running economy

RER: Respiratory exchange ratio

SD: Standard deviation

SEE: Standard error of the mean

SV: Stroke volume

TT: Tabata training

$\dot{V}_E$: Ventilation

$\dot{V}O_2$: Oxygen uptake

$[la^-]_b$: Blood lactate concentration

1. Introduction

Interval training is characterized by work intervals separated by rest periods. The intervals can range from a few seconds to several minutes, making "interval training" a broad term. The purpose of dividing a training session into several intervals is to be able to exercise at a higher intensity than what is possible during longer continuous training sessions. Interval training of supramaximal intensity, maximal intensity or 90 - 95% of maximal heart rate (HR_{max}) is generally shown to improve maximal oxygen uptake ($\dot{V}O_{2max}$) more than moderate intensity continuous training (MCT), according to recent meta-analyses (Milanovic, Sporis, & Weston, 2015; Williams et al., 2019). This is in agreement with the finding that exercise intensity is a key factor for eliciting improvements in $\dot{V}O_{2max}$ (Helgerud et al., 2007; Wenger & Bell, 1986). Since interval training generally is more effective than MCT, different interval training modalities should be compared in the pursuit of the most effective strategy for improving health and performance.

In the present thesis, high-intensity interval training (HIIT) refers to interval training at intensities equal to 85% - 95% of $\dot{V}O_{2max}$ where the intervals are lasting 2 minutes or more. Tabata training (TT) is a modality with short intervals, short rest periods, and supramaximal intensity. It typically refers to a workout consisting of 8 intervals of 20 seconds, carried out at an intensity of ~170% of $\dot{V}O_{2max}$, with 10 seconds rest between intervals (Tabata et al., 1996). In essence, the difference between TT and HIIT, in the present thesis, is the duration of both the work- and rest-periods and the intensity that it is maintained throughout the intervals.

In the present investigation, the 4 x 4 minutes HIIT protocol in Helgerud et al. (2007) will be compared to a treadmill version of the TT protocol in Tabata et al. (1996). An overview of the effects these two interval training modalities exert on endurance performance and the determinants of endurance performance will be presented in the introduction. Additionally, physiological differences between men and women will be presented, underlining the need for research conducted with female subjects.

1.1 Endurance running performance

Endurance running performance is commonly accepted to be dependent on $\dot{V}O_{2max}$, running economy (RE) and lactate threshold (LT), as described by Pate and Kriska (1984) and Joyner (1991). The importance of these factors in endurance performance is generally well accepted (Bassett & Howley, 2000; di Prampero, Atchou, Bruckner, & Moia, 1986; Helgerud, 1994;

Joyner & Coyle, 2008; Pollock, 1977; van der Zwaard et al., 2018). Additionally, anaerobic capacity (AC) influences performance in running events (Joyner & Coyle, 2008), especially in events associated with significant blood lactate accumulation, and is thus included as a determinant of running performance in the present thesis (Figure 1). The inferior performance of women compared to men, both at shorter and longer events (Weiss, Newman, Whitmore, & Weiss, 2016), must therefore be reflected by physiological dissimilarities in one or several of these determinants of endurance performance.

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┌─────────────────┐ ┌─────────────────┐ ┌─────────────────┐ ┌─────────────────┐
│ Maximal oxygen  │ │ Running economy-│ │ Lactate         │ │ Anaerobic       │
│ uptake - V̇O₂max │ │      RE         │ │ threshold - LT  │ │ capacity - AC   │
└─────────────────┘ └─────────────────┘ └─────────────────┘ └─────────────────┘
                    ┌─────────────────────────────────────────┐
                    │      Endurance running performance       │
                    └─────────────────────────────────────────┘
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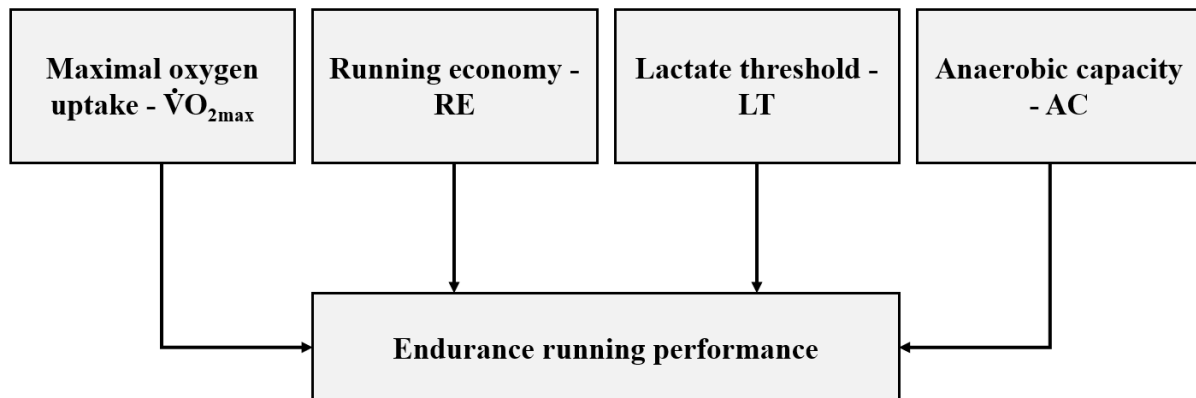
Figure 1: Determinants of endurance running performance, based on Joyner and Coyle (2008).

The factors determining running performance may play a larger or smaller part, depending on the duration it takes to complete the distance examined. $\dot{V}O_{2max}$ is more important in longer running events, and AC plays a more significant role in short running events. This may be exemplified by the relatively low $\dot{V}O_{2max}$ of speed- and power-trained subjects compared to endurance-trained subjects (do Nascimento Salvador et al., 2016; Kusy & Zielinski, 2014; Scott, Roby, Lohman, & Bunt, 1991), and the low AC in endurance-trained compared to sprint-trained subjects (Medbo & Burgers, 1990; Scott et al., 1991).

According to a recent review by Li, Niessen, Chen, and Hartmann (2015), the 50% - 50% point between anaerobically and aerobically derived Adenosine triphosphate (ATP) may be reached at ~75 seconds of maximal whole-body exercise. This finding is in agreement with a previous review on the topic (Gastin, 2001) and the estimate of 60 - 80 seconds presented by Medbo and Tabata (1989). This illustrates the large contribution from both aerobic and anaerobic energy sources during relatively short running events (e.g. 400m and 800m), and indicates that the rate of both aerobic and anaerobic energy release is important in running

performance. The 50% - 50% point is of course dependent on the intensity of the exercise being maximal (all-out).

TT vs. HIIT on endurance running performance

TT has been demonstrated to be equally effective as MCT in improving running time to exhaustion (Schaun, Pinto, Silva, Dolinski, & Alberton, 2018) and time to produce 500kcal on an ergometer cycle (Scribbans, Edgett, et al., 2014). To the author's awareness, no studies have investigated the effect of TT on running performance in a time trial or compared TT to other modalities than MCT. Additionally, potential endurance performance improvements following TT in female subjects have not been examined. On the other hand, endurance running performance have several times been studied and shown to improve following HIIT (Cicioni-Kolsky, Lorenzen, Williams, & Kemp, 2013; Denadai, Ortiz, Greco, & de Mello, 2006; Esfarjani & Laursen, 2007). In the study by Esfarjani and Laursen (2007), a 7% improved time on 3000m time trial was observed after HIIT, which was significantly more than after MCT. The same magnitude of improvement are seen following HIIT in Cicioni-Kolsky et al. (2013) (7.9% - 8.5% improvement), but this was not significantly different from MCT. Furthermore, Cicioni-Kolsky et al. (2013) did demonstrate that men and women improved 3000m performance to the same extent following HIIT.

1.2 Maximal oxygen uptake - $\dot{V}O_{2max}$

The superior $\dot{V}O_{2max}$ of runners compared to unathletic subjects was demonstrated in the 1930s by Robinson, Edwards, and Dill (1937). $\dot{V}O_{2max}$ can be defined as the maximal rate of oxygen uptake and utilization during exhausting exercise (Bassett & Howley, 2000). A relatively high $\dot{V}O_{2max}$ is a necessity to compete at a high level in endurance sports (Lucia, Hoyos, & Chicharro, 2001; Saltin & Astrand, 1967; Wagner, 2000), and it may be considered the single most important predictor of aerobic endurance performance, at least in groups with heterogenous performances (Pate & Kriska, 1984). For example, Costill, Thomason, and Roberts (1973) found a correlation of R = -0.91 between $\dot{V}O_{2max}$ and time to complete a 10-mile (16 km) race in a heterogenous group of runners, and Esfarjani and Laursen (2007) demonstrated a significant correlation of R = -0.77 between $\dot{V}O_{2max}$ and 3000m performance in averagely conditioned men. Furthermore, a recent investigation from our research group reported a correlation of R = -0.77 between $\dot{V}O_{2max}$ and 3000m time trial, with the standard

error of the estimate (SEE) being 7% (Trane, 2018). In addition to its relationship with performance, $\dot{V}O_{2max}$ is also a strong predictor of mortality (Kavanagh et al., 2002; Myers et al., 2002; Ross et al., 2016; Stelken et al., 1996). Therefore, an individual's $\dot{V}O_{2max}$ is of great interest, both in the light of endurance performance and health.

$\dot{V}O_2$, and thus $\dot{V}O_{2max}$, is described by the Fick equation (Acierno, 2000; Barrett-O'Keefe, Helgerud, Wagner, & Richardson, 2012):

$\dot{V}O_2$ = Cardiac output ($\dot{Q}$) x Arterial-venous oxygen difference (a-vO₂diff)

In healthy subjects and normoxic conditions, $\dot{Q}_{max}$ is the factor commonly accepted to be the major contributor to interindividual differences in $\dot{V}O_{2max}$, due to dissimilarities in maximal stroke volume (SV_{max}) (Bassett & Howley, 2000; Saltin & Calbet, 2006), which is highly trainable (Coyle et al., 1984; Helgerud et al., 2007; Helgerud, Wang, Mosti, Wiggen, & Hoff, 2009; Wang et al., 2014; Wisloff et al., 2007). Maximal heart rate (HR_{max}) is either unchanged or slightly decreased with endurance training (in healthy individuals), and is thus not considered to be an important factor contributing to altered $\dot{V}O_{2max}$ with training (Bassett & Howley, 2000; Helgerud et al., 2007; Storen et al., 2017; Wang et al., 2014). Maximal a-vO₂diff may increase with endurance training (Saltin, 1969). However, elite athletes may demonstrate attenuated maximal a-vO₂diff compared to moderately and well-trained controls, as indicated by the tendency in Zhou et al. (2001) and the calculations in Wang, Solli, Nyberg, Hoff, and Helgerud (2012). Reduced transit times in the muscle and/or pulmonary capillaries due to a very high $\dot{Q}_{max}$ may explain why reduced maximal a-vO₂diff sometimes is observed in athletes (Wang et al., 2012; Zhou et al., 2001).

It is important to view all the factors possibly limiting $\dot{V}O_{2max}$ as a whole, since the adaptations of one factor (e.g. SV) may affect other factors (e.g. muscle diffusion capacity) (Wagner, 2000). Wagner (2000) has postulated the importance of interplay between possible limiting factors, and convincingly argued that $\dot{V}O_{2max}$ must be viewed in the context of environmental conditions. The most important issues to grasp are probably that $\dot{V}O_{2max}$ is limited by different factors depending on the conditions and a person's fitness, and that the adaptations of one factor may influence and/or be influenced by other factors (Wagner, 2000). $\dot{V}O_{2max}$ is therefore not an absolute value, but changes with the conditions. For example, $\dot{V}O_{2max}$ is normally lower during cycling compared to running if the subject is not a trained cyclist (Glassford, Baycroft, Sedgwick, & Macnab, 1965; Hill & Vingren, 2014; Saltin & Astrand, 1967), and $\dot{V}O_{2max}$ is also reduced at high altitude (in supply-limited subjects) since

the reduced partial pressure of inspired oxygen lead to hypoxemia with increasing altitude, making less oxygen available for the organism (Wehrlin & Hallen, 2006).

$\dot{V}O_{2max}$ is without doubt trainable and is therefore a commonly used measurement of effect in endurance training studies. Women normally exhibit lower $\dot{V}O_{2max}$ than men, both when expressed as $L \cdot min^{-1}$, $mL \cdot kg^{-1} \cdot min^{-1}$, $mL \cdot kg^{-0.75} \cdot min^{-1}$ (range of exponent: -0.67 to -0.75) and $mL \cdot kg$ fat-free $mass^{-1} \cdot min^{-1}$ (Cureton et al., 1986; Helgerud, 1994; Ingjer, 1991; Jensen, Johansen, & Secher, 2001; A. Nevill et al., 2003; Ogawa et al., 1992; Pate & O'Neill, 2007; Saltin & Astrand, 1967; Sparling, 1980). The average $\dot{V}O_{2max}$ in a young (20-29 years old) and healthy Norwegian male and female population has been shown to be about 54.0 $mL \cdot kg^{-1} \cdot min^{-1}$ / 162.1 $mL \cdot kg^{-0.75} \cdot min^{-1}$ and 43.0 $mL \cdot kg^{-1} \cdot min^{-1}$ / 121.7 $mL \cdot kg^{-0.75} \cdot min^{-1}$, respectively (Aspenes et al., 2011; Loe, Rognmo, Saltin, & Wisloff, 2013). However, it should be noted that studies with less restrictive exclusion criteria report about 10% lower reference values (Edvardsen, Hansen, Holme, Dyrstad, & Anderssen, 2013). Although the reference values reported by Aspenes et al. (2011) and Loe et al. (2013) are quite high, probably due to exclusion of subjects with various illnesses, they are in the present thesis referred to as average $\dot{V}O_{2max}$. Across all age-groups in the study by Loe et al. (2013), men had a 19% and 23% higher $\dot{V}O_{2max}$ than women when expressed as $mL \cdot kg^{-1} \cdot min^{-1}$ and $mL \cdot kg^{-0.75} \cdot min^{-1}$, respectively. The reduced $\dot{V}O_{2max}$ relative to body mass in women is often explained by their higher percentage of body fat and lower concentration of hemoglobin (Hb) (Joyner, 2017).

Men are in general taller and heavier than women, which in turn makes their absolute $\dot{V}O_{2max}$ ($L \cdot min^{-1}$) greater. Expressing $\dot{V}O_{2max}$ in $mL \cdot kg^{-1} \cdot min^{-1}$ will overestimate light subjects compared to heavier subjects, since $\dot{V}O_2$ in theory is proportional to body mass raised to the power of 0.67 (Helgerud, 1994; Jensen et al., 2001; Von Dobeln, 1956). However, the empirical exponent in running is showed to be about 0.75 (Bergh, Sjodin, Forsberg, & Svedenhag, 1991; Helgerud, 1994; Jensen et al., 2001). Therefore, when comparing running $\dot{V}O_{2max}$ between individuals of varied body mass (e.g. men and women), it should be expressed as $mL \cdot kg^{-0.75} \cdot min^{-1}$.

Cureton et al. (1986) demonstrated that differences in Hb concentration between the sexes are unlikely to account for the entire dissimilarity in $\dot{V}O_{2max}$. Following blood withdrawal and plasma volume restoration in male subjects, Hb levels were identical between men and women. The males' $\dot{V}O_{2max}$ concomitantly decreased from 46.4 $mL \cdot kg^{-1} \cdot min^{-1}$ to 43.2 $mL \cdot kg^{-1} \cdot min^{-1}$, which was not different from the females' $\dot{V}O_{2max}$ of 41.6 $mL \cdot kg^{-1} \cdot min^{-1}$. The women in Cureton et al. (1986) may be viewed as relatively better trained than the men, since the sex

difference in baseline $\dot{V}O_{2max}$ was "only" ~12%. The findings of Cureton et al. (1986) lead to the conclusion that Hb concentration accounts for a significant but small portion of the sex difference in $\dot{V}O_{2max}$. Since the reduced Hb concentration results in women exerting a lower oxygen carrying capacity per volume of blood, an increased $\dot{Q}$ during submaximal exercise is necessary to meet the oxygen demand (Astrand, Cuddy, Saltin, & Stenberg, 1964). However, even though reduced blood viscosity is a possible upside of the lower Hb concentrations in women (Cureton et al., 1986), this sex difference cannot be compensated for with an increased $\dot{Q}_{max}$ (Astrand et al., 1964). This indicates that the reduced oxygen carrying capacity is a stronger disadvantage than the potential advantage of reduced blood viscosity. In addition, there is strong evidence that increasing the Hb concentration by manipulation increases $\dot{V}O_{2max}$ (Ekblom & Berglund, 1991; Heuberger et al., 2013; Schmidt & Prommer, 2010).

Women have a higher percentage of body fat compared to men, both in the untrained and trained state (Fleck, 1983; Helgerud, 1994; Ogawa et al., 1992; Sparling, 1980). The higher percentage of body fat in women likely contributes to their lower $\dot{V}O_{2max}$ ($mL \cdot kg^{-1} \cdot min^{-1}$ or $mL \cdot kg^{-0.75} \cdot min^{-1}$), since fat tissue does not contribute significantly to the uptake or utilization of oxygen, but certainly adds to the body mass (Pate & Kriska, 1984). Cureton and Sparling (1980) conducted an experiment where they added weight to their male subjects to make up for the higher body fat percentage in females. This approach reduced the sex difference in $\dot{V}O_{2max}$ ($mL \cdot kg^{-1} \cdot min^{-1}$) and performance on a treadmill test by 65% and ~30%, respectively, clearly illustrating that women's higher fat percentage is a handicap to their $\dot{V}O_{2max}$ and running endurance performance (Cureton & Sparling, 1980).

Furthermore, there are reports that the lungs of women are smaller in relation to their body size compared to men (American Thoracic Society, 1991). There is also evidence suggesting that women have a narrower trachea in relation to their lung size than men (Mead, 1980), and have more mechanically constrained lungs during exercise (McClaran, Harms, Pegelow, & Dempsey, 1998). These factors may contribute to the lower $\dot{V}O_{2max}$ in women by reducing their ability to saturate Hb during maximal exercise. In support of this, it has been reported that women more often than men demonstrate exercise-induced arterial hypoxemia, both in highly and moderately trained subjects (Dominelli et al., 2013; Harms et al., 1998; Richards, McKenzie, Warburton, Road, & Sheel, 2004).

The different phases of the menstrual cycle do not seem to affect endurance performance or $\dot{V}O_{2max}$ to a significant extent, although reviews on this topic report some equivocal results

between studies (Janse de Jonge, 2003; Oosthuyse & Bosch, 2010). Oral contraceptives, however, may be associated with a 5% - 15% decrease in $\dot{V}O_{2max}$ (Casazza, Suh, Miller, Navazio, & Brooks, 2002; Lebrun, Petit, McKenzie, Taunton, & Prior, 2003; Rechichi, Dawson, & Goodman, 2009), and different types of oral contraceptive probably affects $\dot{V}O_{2max}$ to different extents (Burrows & Peters, 2007). Therefore, use of oral contraceptives may influence between-sex comparisons of $\dot{V}O_{2max}$. Importantly, aerobic performance do probably not differ within an oral contraceptive cycle (i.e. 28 days of oral contraceptives use) (Rechichi et al., 2009). Taken together, the lack of fluctuations within a menstrual and oral contraceptive cycle suggests that these factors will not affect the results of training studies, as long as subjects do not start or stop using oral contraceptives during a study.

In addition to the abovementioned factors, there are several physiological traits that may differ between the sexes, and thus explain some of the dissimilarities in $\dot{V}O_{2max}$ between men and women. These factors include lower blood volume (Carrick-Ranson et al., 2013; Åstrand, 2003), lower SV at an absolute or relative intensity (Astrand et al., 1964; Zwiren, Cureton, & Hutchinson, 1983) and lower absolute heart volume (Kjelberg, Rudhe, & Sjostrand, 1949) in women. Blood volume, SV and heart volume may however vary between subjects depending on training status and body size, and these are major confounding factors which hampers between-sex comparisons. Additionally, these three factors should be scaled to body mass, and not reported in absolute values, when comparisons are made between subjects of different size. It is plausible that there are physiological sex differences in these factors, which all could affect $\dot{V}O_{2max}$, but this cannot be concluded based on the existing literature due to methodological difficulties. It should however be noted that Carrick-Ranson et al. (2013) reported larger total blood volume and SV_{max} relative to body mass in healthy males (N = 48) compared to healthy females (N = 47), factors that could be of major influence when explaining the sex difference in $\dot{V}O_{2max}$. In contrast to Carrick-Ranson et al. (2013), Howden et al. (2015) reported equal total blood volume relative to body mass between men (N = 7) and women (N = 5) both before and after training.

The effect of TT vs. HIIT on $\dot{V}O_{2max}$

In the original article, four cycling TT and one cycling MCT session per week for six weeks elicited improvements in $\dot{V}O_{2max}$ from 48 mL·kg^{-1}·min^{-1} to 55 mL·kg^{-1}·min^{-1} (15%) (Tabata et al., 1996). The MCT group in the same study also improved $\dot{V}O_{2max}$, from 53 mL·kg^{-1}·min^{-1} to

58 mL·kg^{-1}·min^{-1} (9%), without any significant difference between the groups. However, with about 15 times less time spent exercising compared to MCT (Tabata et al., 1996), TT have gained popularity in the general population (Viana, de Lira, et al., 2018).

Despite promising results in Tabata et al. (1996) favoring a four-minute protocol, the scientific foundation of TT is quite scarce. To the author's knowledge, eight studies have to this date followed up the investigation of TT's effect on $\dot{V}O_{2max}$ using cycling or running as the modality (Bonafiglia et al., 2017; Bonafiglia et al., 2016; Foster et al., 2015; Laird et al., 2016; Ravier, Dugue, Grappe, & Rouillon, 2009; Schaun et al., 2018; Scribbans, Edgett, et al., 2014; Scribbans, Ma, et al., 2014). Four of these studies compared TT to MCT, of which all four reported an increase in $\dot{V}O_{2max}$ elicited by both TT and MCT, with no difference between the modalities (Bonafiglia et al., 2016; Foster et al., 2015; Schaun et al., 2018; Scribbans, Edgett, et al., 2014).

The nine studies measuring $\dot{V}O_{2max}$ pre and post a cycling or running TT intervention have all reported increased $\dot{V}O_{2max}$ at post-test. The magnitude of change in $\dot{V}O_{2max}$ ranges from 4.6% in the male karate athletes in Ravier et al. (2009), to 18% in the relatively unfit male and female (65% women) subjects in Foster et al. (2015). Women are underrepresented in TT investigations, and only one study has been conducted exclusively on females (Laird et al., 2016). These female subjects had a poor baseline $\dot{V}O_{2max}$ (34 mL·kg^{-1}·min^{-1}) and the two groups did either concurrent TT and resistance training or resistance training only. Furthermore, none of the nine TT studies examining its effect on $\dot{V}O_{2max}$ have compared TT to endurance training modalities other than MCT. Since MCT in some studies are ineffective for subjects with average or above average baseline $\dot{V}O_{2max}$ (Esfarjani & Laursen, 2007; Helgerud et al., 2007), TT should be compared to HIIT to further investigate its potential for eliciting improvements in $\dot{V}O_{2max}$.

HIIT has been extensively studied during the past decades and is shown to induce impressive improvements in $\dot{V}O_{2max}$ in both well-trained subjects (Helgerud, Engen, Wisloff, & Hoff, 2001; Helgerud, Rodas, Kemi, & Hoff, 2011; Laursen, Shing, Peake, Coombes, & Jenkins, 2002, 2005), healthy subjects (Croft et al., 2009; Esfarjani & Laursen, 2007; Helgerud et al., 2007; Ni Cheilleachair, Harrison, & Warrington, 2017; Storen et al., 2017; Wang et al., 2014) and unhealthy/patients (Baekkerud et al., 2016; Rognmo, Hetland, Helgerud, Hoff, & Slordahl, 2004; Slordahl et al., 2005; Wisloff et al., 2007). To the author's knowledge, no study has shown HIIT to be inferior to other training modalities in improving $\dot{V}O_{2max}$, and HIIT commonly elicit significantly greater improvements in $\dot{V}O_{2max}$ than MCT (Baekkerud et

al., 2016; Esfarjani & Laursen, 2007; Helgerud et al., 2007; Rognmo et al., 2004; Wisloff et al., 2007). Additionally, HIIT are also shown effective in studies with exclusively female subjects (Bishop, Edge, Thomas, & Mercier, 2008; Burke, Thayer, & Belcamino, 1994; Edge, Bishop, & Goodman, 2006; Slordahl et al., 2004; Talanian, Galloway, Heigenhauser, Bonen, & Spriet, 2007; Walter, Smith, Kendall, Stout, & Cramer, 2010). However, many of the studies with only female subjects lack an exercising control group (Bishop et al., 2008; Slordahl et al., 2004; Talanian et al., 2007; Walter et al., 2010), and only one of the female HIIT studies mentioned above have used running as the exercise modality (Slordahl et al., 2004). To the author's awareness, the effect of HIIT on a sample of well-trained women (i.e. baseline $\dot{V}O_{2max} \geq 50$ mL·kg^{-1}·min^{-1}) have never been published.

Importantly, no studies have compared TT to HIIT, and this should therefore be conducted in order to compare the effects of TT to a more potent stimulus than MCT. A comparison of these two modalities' effects on $\dot{V}O_{2max}$ is also of interest for public health, since the modality that increases $\dot{V}O_{2max}$ the most may be recommended to the public with the purpose of reducing cardiovascular diseases and mortality (Myers et al., 2002; Ross et al., 2016).

1.3 Running economy - RE

Running economy (RE) is regularly defined as the steady rate $\dot{V}O_2$ of running at a given velocity (Conley & Krahenbuhl, 1980) or the oxygen cost of running per meter (di Prampero et al., 1986). Interindividual differences in RE exists, and RE is a predictor of endurance performance independent of $\dot{V}O_{2max}$ and LT (Conley & Krahenbuhl, 1980; Helgerud, 1994). In Conley and Krahenbuhl (1980), variation in RE explained 65% of the variation in a 10-km race performance within a group with homogenous $\dot{V}O_{2max}$.

The mechanisms determining RE are not elucidated, but several factors are thought to affect RE (Barnes & Kilding, 2015; Joyner & Coyle, 2008; Pate & Kriska, 1984). These factors include fiber-type distribution (Bosco et al., 1987), elastic energy utilization (Bosco et al., 1987), mitochondrial and oxidative enzyme characteristics (Saunders, Pyne, Telford, & Hawley, 2004), biomechanics (Moore, Jones, & Dixon, 2012) and rate of force development (Storen, Helgerud, Stoa, & Hoff, 2008). RE is a trainable attribute, and can be improved both by strength training (Millet, Jaouen, Borrani, & Candau, 2002; Storen et al., 2008) and endurance training (Franch, Madsen, Djurhuus, & Pedersen, 1998; Helgerud et al., 2007).

Reports of whether RE expressed as $mL \cdot kg^{-1} \cdot min^{-1}$ differs between men and women are equivocal, with some indicative of males exhibiting superior RE compared with females (Bransford & Howley, 1977; Daniels & Daniels, 1992) and some indicative of equal RE between the sexes (Bunc & Heller, 1989; Daniels, Krahenbuhl, Foster, Gilbert, & Daniels, 1977; Davies & Thompson, 1979). However, just as with $\dot{V}O_{2max}$, RE should be allometrically scaled when comparing subjects of different body mass (Bergh et al., 1991; Helgerud, 1994). When body mass is raised to the power of 0.75, RE has been shown to be superior in trained women when compared to both performance matched men (Helgerud, 1994), and men with superior $\dot{V}O_{2max}$ and performance (Helgerud, Storen, & Hoff, 2010). It should be mentioned that in Helgerud (1994), the female subjects did report a higher training volume over the last 6 months compared to the males, which is a possible explanation for their superior RE. Interestingly, when expressed as $mL \cdot kg^{-1} \cdot m^{-1}$, mean RE was equal between the sexes in Helgerud et al. (2010), but the sex difference in RE when expressed as $mL \cdot kg^{-0.75} \cdot m^{-1}$ was about 10% (men = 0.755 $mL \cdot kg^{-0.75} \cdot m^{-1}$ vs. women = 0.680 $mL \cdot kg^{-0.75} \cdot m^{-1}$). This is an example of how lighter subjects are underestimated (assumed to have poorer RE) when comparing their RE to heavier subjects, expressed with body mass raised to the power of 1 (Bergh et al., 1991).

Slow-twitch type I fibers are commonly regarded as more efficient than fast-twitch type II fibers (Bosco et al., 1987; Conley, Jubrias, Cress, & Esselman, 2013), and there are large interindividual differences in the distribution of muscle fiber types (Simoneau & Bouchard, 1989). Equivocal results regarding potential sex differences in fiber-type distribution exists, with reports of a higher proportion of type I fibers in women (Miller, MacDougall, Tarnopolsky, & Sale, 1993; Simoneau & Bouchard, 1989), reports of no differences between the sexes (Costill et al., 1976; Staron et al., 2000) and reports of lower type I proportion in women (Essen-Gustavsson & Borges, 1986). Another aspect of potential skeletal muscle sex differences is the suggestion that female type I fibers have a larger cross-sectional area (CSA) than their type II fibers, which is the opposite of male muscle fibers (Staron et al., 2000). This possible difference would lead to a larger proportion of the total skeletal muscle CSA consisting of type I fibers in women compared to men. However, the possible sex differences in fiber-type distribution is not yet elucidated. One may speculate that a higher proportion of type I fibers or type I fiber CSA in women would be beneficial for RE, due to the superior efficiency of slow-twitch fibers compared to fast-twitch fibers.

The effect of TT vs HIIT on running economy

To the best of the author's awareness, only Schaun et al. (2018) have investigated the effect of TT on RE, including males only. Surprisingly, both the TT group and the MCT group in Schaun et al. (2018) displayed significantly worse RE following training. HIIT, on the other hand, frequently improves RE (or walking economy) to the same extent as MCT (Baekkerud et al., 2016; Helgerud et al., 2007; Slordahl et al., 2005) or even more than MCT (Wisloff et al., 2007), although improved RE is not a universal finding (Denadai et al., 2006; Impellizzeri et al., 2006). No study has to the author's awareness examined the effect of TT on RE in women.

1.4 Lactate threshold - LT

Lactate threshold (LT) may be defined as the intensity where blood lactate concentration ($[la^-]_b$) gradually starts to increase (Hagberg & Coyle, 1983). There is interindividual variance in LT expressed as percentage of $\dot{V}O_{2max}$ (%$\dot{V}O_{2max}$) (Coyle, Coggan, Hopper, & Walters, 1988; Farrell, Wilmore, Coyle, Billing, & Costill, 1993), and the fractional utilization of $\dot{V}O_{2max}$ during endurance events are linked to the LT (Bassett & Howley, 2000; Joyner, 1991, 1993). The velocity at LT (vLT) is a very strong predictor of endurance performance, as demonstrated by Hagberg and Coyle (1983) (R = 0.94) and Farrell et al. (1993) (R = 0.91), because this velocity depends on both LT (as %$\dot{V}O_{2max}$), $\dot{V}O_{2max}$ and RE (Bassett & Howley, 2000; Joyner, 1991). LT as %$\dot{V}O_{2max}$ is not affected by $\dot{V}O_{2max}$ and RE, and it is commonly reported that endurance-trained subjects have superior LT (%$\dot{V}O_{2max}$) compared to untrained subjects (Hurley et al., 1984; Joyner & Coyle, 2008; Saltin, 1969; Sjodin & Svedenhag, 1985).

Results from studies are equivocal regarding the trainability of LT as %$\dot{V}O_{2max}$, with some reporting an increase (Acevedo & Goldfarb, 1989; Hurley et al., 1984; Weltman et al., 1992), some reporting no change (Helgerud et al., 2001; Helgerud et al., 2007; Impellizzeri et al., 2006; Saltin, Hartley, Kilbom, & Astrand, 1969; Sjodin, Jacobs, & Svedenhag, 1982) and at least one study reporting a decrease (Philp, Macdonald, Carter, Watt, & Pringle, 2008). One explanation for the equivocal findings may be that LT as %$\dot{V}O_{2max}$ only commonly increases in poorly conditioned individuals (Sjodin & Svedenhag, 1985).

The factors responsible for interindividual differences in LT as %$\dot{V}O_{2max}$ are not elucidated, and the poor trainability of LT hampers investigations of potential underlying mechanisms.

Several factors may influence LT, such as lactate transport capacity, proportion of type I fibers, greater reliance on fat as fuel, and mitochondrial content and enzyme activity (Bishop, Granata, & Eynon, 2014; Hawley, 2002; Pilegaard, Bangsbo, Richter, & Juel, 1994). The capacity for lactate transport in skeletal muscle is probably dependent on density of the monocarboxylate transporters MCT1 and MCT4, of which MCT1 can increase following training (Juel, 2006). Furthermore, LT as $\%\dot{V}O_{2max}$ appears not to differ between performance matched men and women (Helgerud, 1994; Helgerud, Ingjer, & Stromme, 1990) or between well-trained men and women (Helgerud et al., 2010).

The effect of TT vs HIIT on lactate threshold

No reports of the effect of TT on LT (as $\dot{V}O_2$ or $\%\dot{V}O_{2max}$) exists, to the best of the author's awareness. As with other training modalities, LT ($\%\dot{V}O_{2max}$) do commonly not increase following HIIT (Helgerud et al., 2001; 2007).

1.5 Anaerobic capacity - AC

Anaerobic capacity (AC) can be defined as the maximal ATP formation by anaerobic processes, i.e. phosphocreatine (PCr) and anaerobic glycogen breakdown (Medbo et al., 1988). During submaximal exercise below LT, oxygen supply is abundant and the anaerobic energy system is not significantly taxed (Katz & Sahlin, 1988; Medbo & Tabata, 1989). However, with increasing intensity and decreasing duration, anaerobically derived ATP contributes increasingly to the total energy supply (Gastin, 2001; Joyner & Coyle, 2008; Medbo & Tabata, 1989).

It seems reasonable to suggest that there is a maximum of energy derived from anaerobic sources during exercise (Medbo & Tabata, 1989), since the PCr stores are limited (Hultman & Sjoholm, 1983) and the decreased pH associated with lactate accumulation is thought to contribute to muscular fatigue (Hostrup & Bangsbo, 2017; Sahlin & Henriksson, 1984). In support of the view that the AC exhibits a maximum, Medbo et al. (1988) showed that the accumulated oxygen deficit and peak $[la^-]_b$ reached a plateau after about 2 minutes of all-out treadmill running.

Since there are no direct methods for measuring AC, indirect measures are commonly used. The maximal accumulated oxygen deficit (MAOD), re-introdused by Medbo et al. (1988), is, although not flawless, probably the most used method to determine AC (Noordhof, de

Koning, & Foster, 2010). AC, measured as MAOD, may increase with training, and sprint-trained subjects are shown to exhibit higher MAOD than untrained and endurance-trained subjects (Medbo & Burgers, 1990; Ravier et al., 2009; Scott et al., 1991; Tabata et al., 1996). The Wingate test or 30 seconds all-out running are other commonly used performance tests associated with anaerobic performance, and also these methods indicates the trainability of the anaerobic system (Astorino, Allen, Roberson, & Jurancich, 2012; Bangsbo, Gunnarsson, Wendell, Nybo, & Thomassen, 2009; Burgomaster et al., 2008; Iaia et al., 2008).

The intramuscular energy stores of PCr and glycogen are limited, and while the PCr stores probably will deplete during a two-minute long MAOD-test (Bogdanis, Nevill, Boobis, & Lakomy, 1996; Hultman & Sjoholm, 1983), the magnitude of glycogen stored are unlikely to affect performance in such a test (Bangsbo, Graham, Kiens, & Saltin, 1992). The amount of energy derived from stored high-energy phosphates (ATP and PCr) and O_2 bound to Hb and myoglobin (Mb) equates for about 20% - 35% of the total accumulated oxygen deficit during a 2-3-minute bout until exhaustion (Bangsbo et al., 1992; Medbo et al., 1988). The concentration of PCr in skeletal muscle is commonly reported to be unaffected by sprint-training, e.g. in the reviews by Ross and Leveritt (2001) and Iaia and Bangsbo (2010). Thus, despite some reports of elevated PCr stores following exercise training (Parra, Cadefau, Rodas, Amigo, & Cusso, 2000), it seems reasonable to suggest that the amount of stored PCr does not contribute significantly to the increased AC observed following training.

Considering that the amount of stored glycogen and high-energy phosphates probably do not account for the improved AC following training, other mechanisms must account for this improvement. These mechanisms likely contribute to an improved capacity to derive ATP from glycolysis, as this probably is the main adaption causing improved AC following training (Ramsbottom, Nevill, Seager, & Hazeldine, 2001; Weber & Schneider, 2002).

Several enzymes are hypothesized to contribute to increased AC and performance, such as phosphofructokinase (PFK), lactate dehydrogenase (LDH) and creatine kinase (CK) (MacDougall et al., 1998; Roberts, Billeter, & Howald, 1982; Rodas, Ventura, Cadefau, Cusso, & Parra, 2000). However, the activity of PFK, LDH and CK does not always increase despite improvements in anaerobic performance (Iaia & Bangsbo, 2010). It is suggested that these differences between studies are, at least partial, due to the different baseline fitness of the subjects studied (Iaia & Bangsbo, 2010), meaning that well-trained subjects may have abundant activity of these enzymes.

The expression of Na^+, K^+ pump α1 and/or α2 subunits is shown to increase after supramaximal training (Bangsbo et al., 2009; Iaia et al., 2008; Nielsen et al., 2004; Thomassen, Christensen, Gunnarsson, Nybo, & Bangsbo, 2010). This augmentation may increase the number of working ion pumps, resulting in reduced net loss of intracellular K^+ (probably due to increased re-uptake) during intense exercise, and thus reduced interstitial K^+ accumulation (Iaia & Bangsbo, 2010). A high concentration of interstitial K^+ is associated with decreased membrane potential and fatigue (Nielsen et al., 2004). However, even though K^+ concentrations vary between individuals and probably plays a role in the development of fatigue, the magnitude of its impact is uncertain (Nielsen & de Paoli, 2007).

There may be a close interplay between factors causing fatigue and factors associated with AC, since fatigueing mechanisms is likely to have detrimental effects on performance during the short 2-3-minute bouts used to measure AC. A more thorough overview of the potential mechanisms thought to contribute to fatigue during intense exercise, and how these may be enhanced with training, is beyond the scope of this thesis. Some possible factors are dicussed, but other factors, such as Ca^{2+} handling and H^+ regulation, are not. The mechanisms underlying fatigue and AC are complex, possibly affected by the training status of subjects, probably multifactorial, and clearly not yet elucidated (Hostrup & Bangsbo, 2017).

Without doubt, some of the inter- and intraindividual differences in AC (e.g. between men and women or between running and cycling) can be attributed to the amount of active muscle mass during the tests (Hill & Vingren, 2011; Maud & Shultz, 1986; Weyand, Cureton, Conley, & Higbie, 1993). Hill and Vingren (2014) compared the AC of men (N = 104 running and N = 110 cycling) and women (N = 119 running and N = 106 cycling), and revealed that men had a 32% greater MAOD than women, expressed as $mL·kg^{-1}$. The superior AC of men is in agreement with other studies (Ramsbottom, Nevill, Nevill, & Hazeldine, 1997; Weber & Schneider, 2000; Weyand et al., 1993). Some of this sex difference can probably be explained by the greater percentage of body fat commonly observed in women, since fat mass do not contribute to the anaerobic metabolism. However, when AC is expressed per kg fat-free mass or per kg estimated active muscle mass, women are still inferior to men (Weber & Schneider, 2000; Weyand et al., 1993). Importantly, MAOD is a measure of the total volume of oxygen deficit, and not per time unit (which commonly is the case for $\dot{V}O_{2max}$). Consequently, it should in theory be reported as O_2 volume per kg body mass (e.g. $mL·kg^{-1}$) when comparing subjects of different size.

The reports of lower AC in women compared to men when scaled for fat-free mass or active muscle mass are interesting because they suggest qualitative differences in maximal anaerobic metabolism between the sexes. One potential explanation for this difference is that women may generally exhibit a lower percentage of type II fibers compared to men (Miller et al., 1993; Simoneau & Bouchard, 1989), and fast-twitch fibers are known to have a larger potential for anaerobic energy release than slow-twitch fibers (Picard, Hepple, & Burelle, 2012). However, as mentioned earlier, this potential sex difference in fiber-type distribution is not yet elucidated. Male skeletal muscle generally have a larger absolute CSA of both slow- and fast-twitch fibers compared to female skeletal muscle (Simoneau & Bouchard, 1989; Staron et al., 2000). Additionally, a higher concentration of glycolytic enzymes in male skeletal muscle may also contribute to the sex difference in AC (Simoneau & Bouchard, 1989). An interesting finding in Staron et al. (2000) is that type II CSA amounted for 41% of the total CSA in the males biopsies, while the same proportion in females was only 34%. This possible dissimilarity in relative CSA of fast-twitch fibers may contribute to the lower AC in women, also when scaled for fat-free mass or active muscle mass.

Furthermore, some studies report that the trainability of the AC is equal between men and women (Ramsbottom et al., 2001; Weber & Schneider, 2002), while Medbo and Burgers (1990) suggested that women may have reduced trainability of the AC compared to men. None of these three studies did however reestablish the relationship between $\dot{V}O_2$ and velocity/power after training and are therefore in risk of overestimating MAOD at post-tests. Thus, it remains to be elucidated whether a sex difference in the trainability of AC exists.

The effect of TT vs HIIT on anaerobic capacity

In the original article by Tabata et al. (1996), TT elicited a 28% improvement in AC, measured as MAOD, while MCT did not affect this parameter. Importantly, in Tabata et al. (1996), the relationship between steady-state $\dot{V}O_2$ and power was not reestablished after the training interventions, a methodological flaw possibly leading to overestimation of MAOD post-training. To the best of the author's knowledge, only Ravier et al. (2009) have followed up the investigation of TT's effect on MAOD, reporting a 10% increase. Neither the study by Tabata et al. (1996) or Ravier et al. (2009) included female subjects.

To the author's awareness, no study has investigated the effect of HIIT on AC measured as MAOD. When aiming for anaerobic improvements, intensity is typically higher than during

HIIT, resulting in a larger relative contribution from anaerobic energy sources during the supramaximal intervals. However, it is plausible that HIIT may improve AC, since lactate accumulates during the intervals (Storen et al., 2017). This rise in $[la^-]_b$ leads to the necessity for breaks interspersing the intervals and are evidence of some anaerobic contribution. In the study by Medbo and Burgers (1990), a HIIT-like protocol consisting of 3 x 2 minutes supramaximal intervals of 116% of $\dot{V}O_{2max}$ conducted 3 times per week for 6 weeks increased AC by 10%.

Considering the finding by Medbo and Burgers (1990) and the lactate accumulation associated with the frequently used 4 x 4 minutes protocol, it should be investigated whether HIIT induces sufficient stimulus to elicit improvements in AC. In such an investigation, HIIT should preferably be compared with supramaximal training, e.g. TT, which are previously shown to improve AC.

1.6 Aim and hypothesis

The aim of the present study is to compare the effects of HIIT and TT on running performance, $\dot{V}O_{2max}$, RE, LT and AC, with exclusively well-trained female subjects included. Specifically, the 4 x 4 minutes HIIT protocol described in Helgerud et al. (2007) will be compared to a TT protocol consisting of 7-9 intervals of 20 seconds separated by 10 seconds of rest. Women generally seems underrepresented in interval training research, and investigations of well-trained females are especially rare. Even though male and female physiology have some dissimilarities, there is no strong evidence indicative of different adaptations to training in women than what is normally seen in comparable male samples. It is hypothesized that HIIT will improve $\dot{V}O_{2max}$ and 3000m more than TT, and that TT will increase AC more than HIIT.

2. Methods

2.1 Subjects

23 healthy non-smoking females volunteered to participate in this study. The majority were recruited through written and verbal advertisement around different campuses at NTNU, Trondheim. A $\dot{V}O_{2max}$ between 45 and 58 mL·kg^{-1}·min^{-1} and endurance training at least once per week or other activities three times per week were set as inclusion criteria. A history of heart, coronary or chronic lung disease were set as exclusion criteria. Use of hormonal contraceptives was not an exclusion criteria, even though it may affect a subjects' $\dot{V}O_{2max}$ (Janse de Jonge, 2003; Oosthuyse & Bosch, 2010; Rechichi et al., 2009). Participation in 20 - 24 ($\geq 83.3\%$) supervised training sessions within an 8-week period were required for inclusion in the post-tests. The study was carried out in accordance with the recommendation of Norwegian Data Protection Center and Declaration of Helsinki. The participants were informed with a written consent. The Institutional Review Board of NTNU approved the protocol.

Table 1. Subject descriptive data

	HIIT (N = 5)	Tabata (N = 7)
Age (year)	25 ± 2	22 ± 2
Height (cm)	167 ± 6	173 ± 7
Body mass (kg)	59.5 ± 5.4	65.7 ± 7.3
$\dot{V}O_{2max}$		
$\quad$ L · min^{-1}	3.22 ± 0.38	3.51 ± 0.42
$\quad$ mL · kg^{-1} · min^{-1}	54.1 ± 1.9	53.4 ± 3.3
$\quad$ mL · kg$^{-0.75}$ · min^{-1}	150.2 ± 8.3	151.9 ± 9.8

Data are presented as mean ± SD.

2.2 Testing

Within two weeks before and after the training period, the subjects had to meet twice in an exercise lab and once at an indoor track and field. All tests described was conducted both before and after the intervention. The testing and training were administered by two master students who was not blinded to the subjects' test results or intervention. All subjects had at least one day of rest preceding each test. All tests in the lab were conducted on a motorized treadmill (Woodway PPS 55 Sport, Waukesha, Germany). Cortex Metamax II portable test-system (Cortex Biophysik GmbH, Leipzig, Germany) were used for all measurements of pulmonary oxygen uptake, and this system has been validated against the Douglas bag method (Larsson, Wadell, Jakobsson, Burlin, & Henriksson-Larsen, 2004). For HR measurements during the tests, a Polar heart rate monitor and watch (Polar F11, polar Electro Oy, Kempele, Finland) were used.

$\dot{V}O_{2max}$, RE and LT

$\dot{V}O_{2max}$, RE and LT were tested on a motorized treadmill continuously and without exception set at 5.3% inclination. Following a 10-minute warm-up at about 60% of estimated $\dot{V}O_{2max}$, five-minute stages of running at increasing velocities to determine the LT began.

In order to determine the LT, a 20 μL sample of capillary blood from a fingertip was collected directly following warm-up and each five-minute stage. LT was defined as the $\dot{V}O_2$, HR or velocity were $[la^-]_b$ equaled 1.5 mM above the lowest measured $[la^-]_b$ (normally following the warm-up) (Helgerud et al., 2007). The intensity of the five-minute stages increased with 1 $km \cdot h^{-1}$ from one stage to the next, separated by a short break for measuring $[la^-]_b$. All lactate samples were analyzed using a Biosen C-line lactate analyzer (EKF-diagnostic GmbH, Leipzig, Germany).

RE at a standardized intensity was in the present study analyzed at 7 $km \cdot h^{-1}$ for all subjects. Additionally, all subjects had to complete at least three five-minute stages during the LT-protocol, since the relationship between submaximal $\dot{V}O_2$ and velocity needed to be established before the MAOD-test (see below).

Following the LT and RE procedure, subjects were allowed to walk for about five minutes before proceeding to the $\dot{V}O_{2max}$-test. The $\dot{V}O_{2max}$-test started at an intensity above LT, and the velocity was increased by 1 $km \cdot h^{-1}$ every minute until exhaustion, lasting 4-7 minutes. Strong verbal encouragement was given during the last minutes of the $\dot{V}O_{2max}$-test. A capillary blood sample was drawn from a fingertip within the first 2 minutes after exhaustion to determine the $[la^-]_b$. The highest 30-second average $\dot{V}O_2$ was given as the subjects $\dot{V}O_{2max}$. The presence of a plateau in $\dot{V}O_2$ despite increased velocity or ventilation, combined with either a $[la^-]_b$ above 8 mM and/or a respiratory exchange ratio (RER) above 1.05 was used as criteria for accepting the $\dot{V}O_{2max}$ (Helgerud et al., 2007; Helgerud et al., 2010). The MAOD-protocol (see below) was regarded as a method to verify the $\dot{V}O_{2max}$-test (Poole & Jones, 2017). The highest recorded HR during the $\dot{V}O_{2max}$-test was taken as HR_{max}. HR_{max} and $\dot{V}O_{2max}$ was used to calculate maximal O_2 pulse, a parameter which reflects SV (Crisafulli et al., 2007). O_2 pulse ($mL \cdot beat^{-1}$) is given by $\dot{V}O_{2max}$ ($mL \cdot min^{-1}$) divided by HR_{max} ($beat \cdot min^{-1}$).

MAOD

MAOD was tested at least two days after LT, RE and $\dot{V}O_{2max}$, using a motorized treadmill set at 5.3% inclination throughout the test. In the present study, the simplified procedure with a Y-intercept of 5 mL·kg^{-1}·min^{-1} was used due to a low number (< 10) of submaximal stages (simplified procedure nr. 3 in Medbo et al. (1988)). The $\dot{V}O_2$ at several (at least three) submaximal velocities and the Y-intercept of 5 mL·kg^{-1}·min^{-1} was used to establish a linear relationship between $\dot{V}O_2$ and velocity for each subject. For each individual, v$\dot{V}O_{2max}$ was calculated from their $\dot{V}O_{2max}$ and the relationship between $\dot{V}O_2$ and velocity (see Figure 2). The subjects ran at an intensity corresponding to 110% - 130% of v$\dot{V}O_{2max}$ until exhaustion during the MAOD-test. This intensity was set with the purpose of exhausting the subjects after 2 minutes, a method which has been demonstrated to give high and plateauing values of MAOD (Medbo et al., 1988). The original procedure in Medbo et al. (1988), including 10 submaximal bouts, had a total methodological error of 3 mL·kg^{-1} (4%) in determining MAOD. The simplified procedure used in the present investigation has been demonstrated to have a mean absolute difference of 2.0 ± 0.8 mL·kg^{-1} from the original procedure, with individual differences in MAOD never exceeding 3 mL·kg^{-1} (Medbo et al., 1988). Importantly, the results from the procedure used in the present study was not statistically different from the control procedure in the study by Medbo et al. (1988).

A 15-minute warm-up at about 60% of $\dot{V}O_{2max}$ and 10 minutes of rest preceded the MAOD-test. During the warm-up, subjects performed 1-2 bouts of ~10 seconds at the same intensity as the upcoming MAOD-test. This was done to prepare the subjects for the high intensity of the test without significant risk of lactate accumulation. During the 10 minutes of rest between warm-up and the test, $[la^-]_b$ was measured to make sure the subjects started the test with resting values of $[la^-]_b$. The subjects had been verbally prepared to run as long as possible at this intensity, and they all received strong encouragement during the test. A capillary blood sample was drawn within the first 2 minutes after exhaustion to determine the $[la^-]_b$.

The O_2 demand of the supramaximal bout was estimated by extrapolating the relationship between submaximal $\dot{V}O_2$ and velocity to the supramaximal intensity of the test. To calculate MAOD, the accumulated VO_2 during the test was subtracted from the theoretical accumulated O_2 demand, which is given by the intensity and time to exhaustion (Medbo et al., 1988) (Figure 3). Stored O_2 bound to Mb and Hb was not corrected for when calculating MAOD.
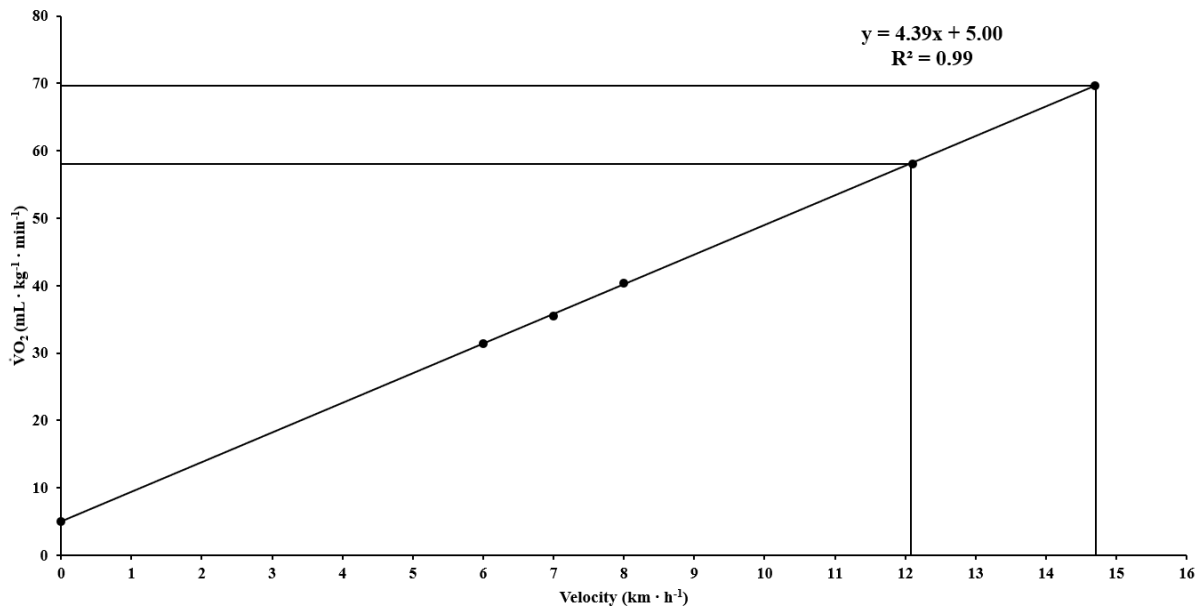
Figure 2. Linear relationship between $\dot{V}O_2$ and velocity. The linear regression for this female subject is based on three submaximal workloads at 6, 7 and 8 km·h^{-1} and a Y-intercept of 5.0 mL·kg^{-1}·min^{-1}. This subjects' $\dot{V}O_{2max}$ (58.1 mL·kg^{-1}·min^{-1}) and linear relationship between $\dot{V}O_2$ and velocity gives a v$\dot{V}O_{2max}$ of 12.1 km·h^{-1}. The velocity during the MAOD-test was 14.7 km·h^{-1}, which equals 120% of 12.1 km·h^{-1} (v$\dot{V}O_{2max}$).
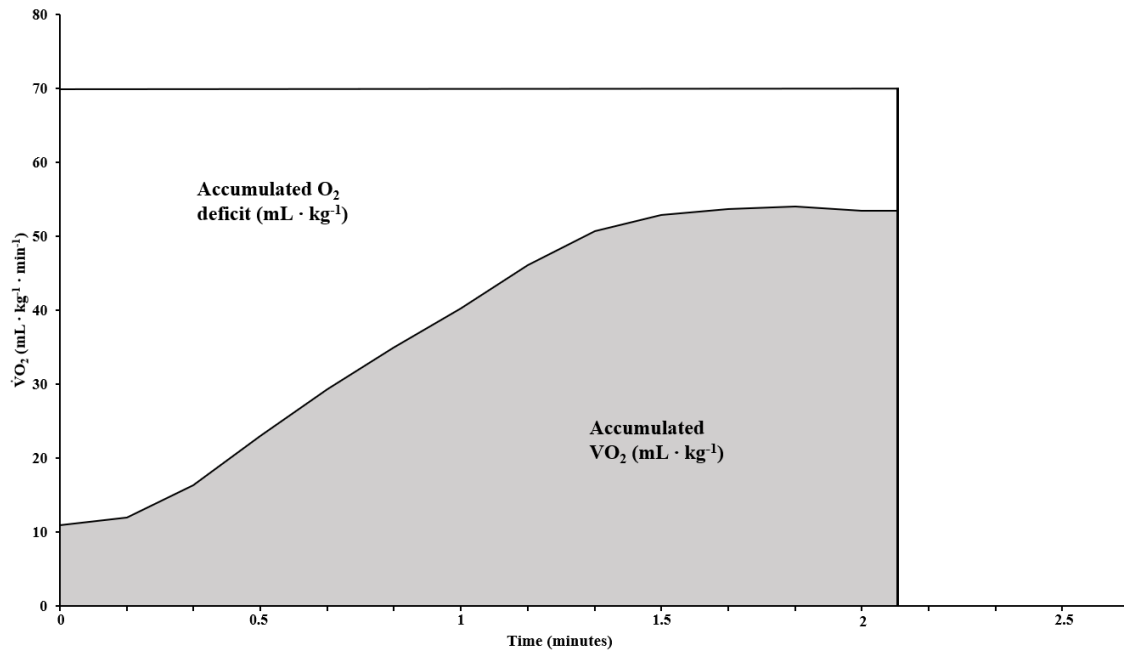

Figure 3. Illustration of the calculation of MAOD for a female subject with a $\dot{V}O_{2max}$ of 58.1 mL·kg^{-1}·min^{-1}. The subject ran at 14.7 km·h^{-1}, corresponding to 120% of v$\dot{V}O_{2max}$ and a calculated O_2 demand of 69.7 mL·kg^{-1}·min^{-1}. During the time to exhaustion of 124 seconds, the total accumulated O_2 demand (white and grey area combined) was calculated to equal 144.0 mL·kg^{-1}. The accumulated $\dot{V}O_2$ (grey area) during the 124 seconds was 75.9 mL·kg^{-1}, giving an accumulated O_2 deficit (white area) of 68.1 mL·kg^{-1}.

Running performance on 300m and 3000m

The performance tests were conducted on a banked 200-meter indoor track and field. The subjects completed 10 minutes of individual warm-up before running the 300m. Following the 300m, the subjects waited 30 minutes before the 3000m start, of which the last 10 minutes was dedicated to another warm-up. The 300m was interval start in a random order, while the 3000m was mass start with up to 10 participants. Time was measured manually using a watch and rounded to the nearest tenth of a second for 300m and to the nearest second for 3000m. Both during 300m and 3000m, the subjects received verbal encouragement.

2.3 Training

The subjects were randomized to either a HIIT or TT group. Both interventions consisted of three weekly supervised training sessions over an 8-week period. The subjects were asked to refrain from other high-intensity endurance training during the period between pre- and post-tests. However, subjects were encouraged to continue with their other normal activities in addition to the training intervention, for example soccer, dancing and badminton. Both interventions were conducted running on a treadmill (Gymsport TX200, Trondheim, Norway) at 5.5% inclination, and both interventions had a 10-minute warm-up at about 70% of HR_{max}.

HIIT

The HIIT group performed 4 intervals of 4 minutes duration (4 x 4) at 90% - 95% of HR_{max} (Helgerud et al., 2007). The intervals were separated by 3 minutes of active rest at about 70% of HR_{max}, and 3 minutes of cool-down at $\leq$ 70% of HR_{max} ended the sessions. All intervals were supervised throughout the entire training period to ensure that the actual intensity was 90% - 95% of HR_{max}. Throughout the intervention period, the treadmill velocity was regularly adjusted in order to reach the target intensity, and HR was recorded after 3 minutes during all intervals. Including warm-up and cool-down, the total duration of the 4 x 4 protocol was 38 minutes. An illustration of a 4 x 4 session is presented in Figure 4.
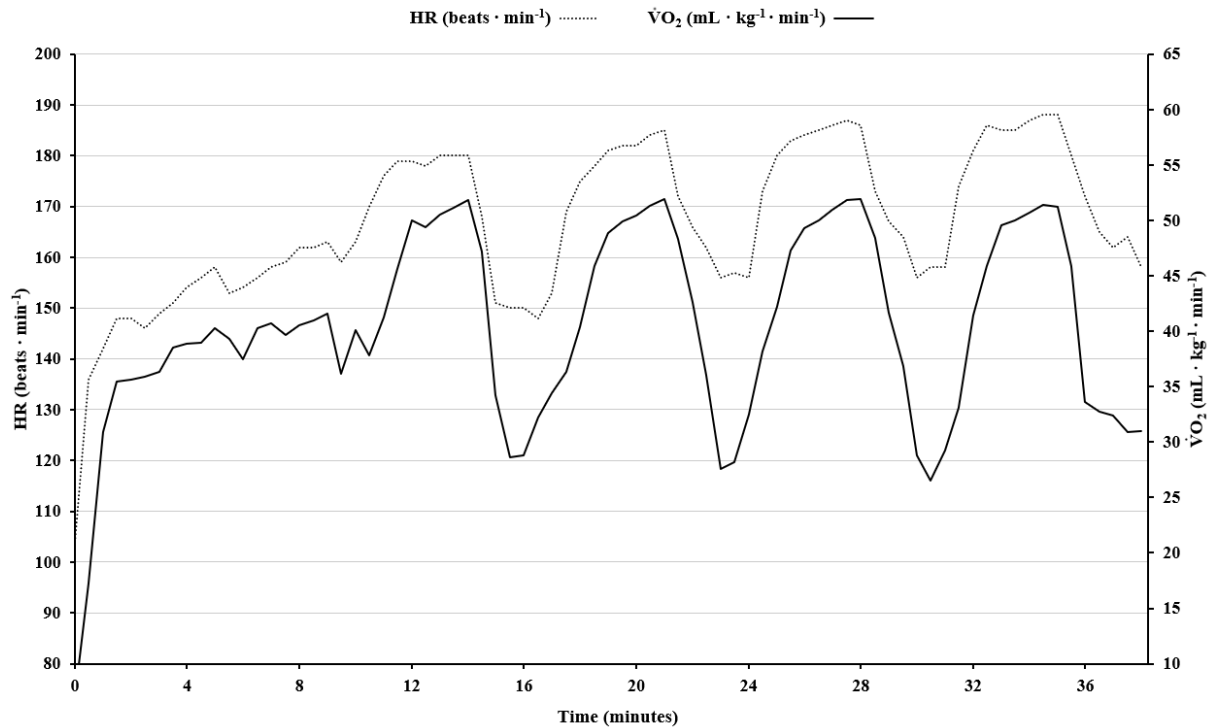
Figure 4. Example of a HIIT (4 x 4 minutes) session for a female subject with a $\dot{V}O_{2max}$ of 55.7 mL·kg^{-1}·min^{-1} and a HR$_{max}$ of 199 beats·min^{-1}. Immediately following the first and fourth interval, [la$^-$]$_b$ was measured to 6.3 mM and 7.0 mM, respectively.

Tabata training

TT consisted of 20-second intervals separated by 10 seconds of rest. The intensity during TT was set by the subject's performance on the previous training session, and the velocity during the first session was 140% of $v\dot{V}O_{2max}$. If a subject completed 9 intervals, the velocity was increased at the next training session, aiming to exhaust the subjects during the eight or ninth interval. Excluding the rest periods, a session consisted of 140 - 180 seconds of supramaximal intensity. During all intervals, every subject had one-to-one follow-up and strong verbal encouragement, ensuring that the intensity was all-out. Including the warm-up and a 10-minute cool-down at ≤ 70% of HR$_{max}$, the total duration of a TT session was 25 minutes. An illustration of a TT session is presented in Figure 5.
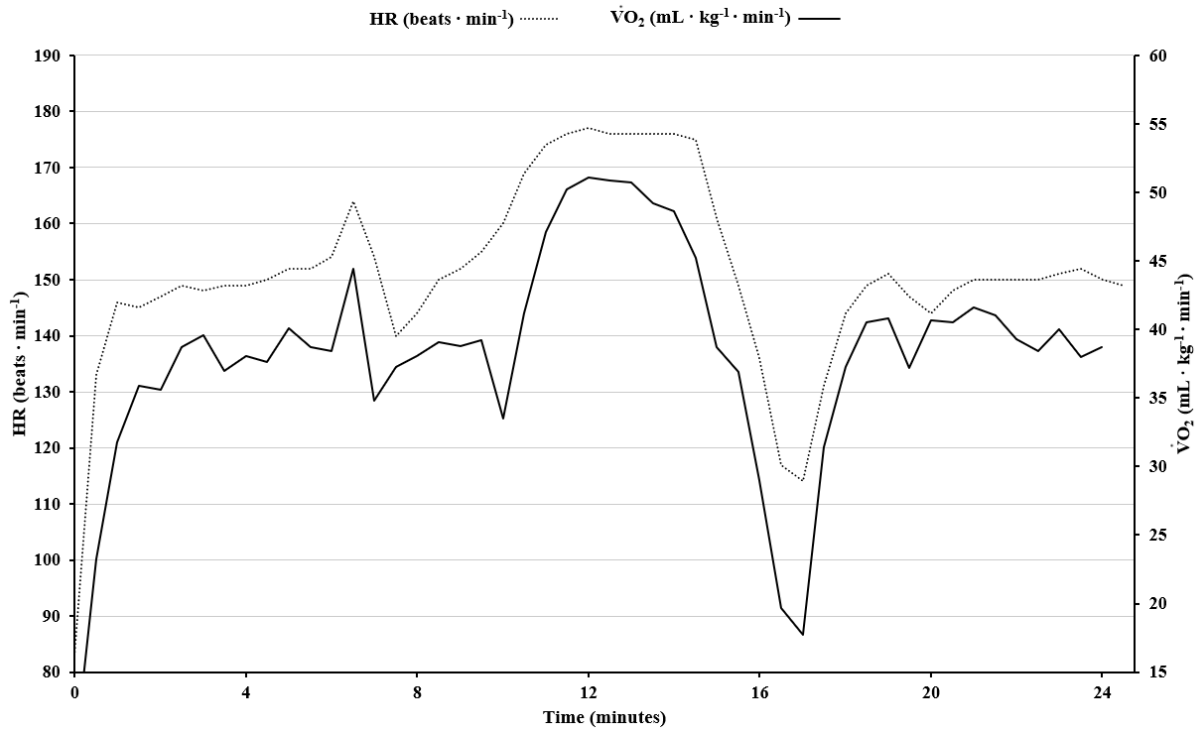
Figure 5. Example of a Tabata training session for a female subject with a $\dot{V}O_{2max}$ of 55.8 mL·kg⁻¹·min⁻¹ and a HR_{max} of 182 beats·min⁻¹. The treadmill incline was 5.5% and the velocity was 15.8 km·h⁻¹. In this example session, the subject was exhausted during the ninth interval, and her post-training $[la^-]_b$ was 15.4 mM. During the warm-up, one 12-second bout at 15.8 km·h⁻¹ was performed.

2.4 Statistical analysis

All statistical analyses were conducted using IBM SPSS Statistics 25 (Armonk, NY: IBM Corp.) software. Due to small sample sizes, Wilcoxon signed-ranks test (within group) and Mann-Whitney U test (between groups) were used to analyze significance levels within and between groups. Correlations were analyzed with Spearman's rank-order correlation test, using pooled data from pre- and post-tests. Level of significance was without exception set to $P \leq 0.05$. Instead of median and interquartile range, mean and standard deviation are presented in text, tables and figures for descriptive purposes and to facilitate comparison with other studies.

3. Results

Of the 23 women randomized to either HIIT or TT, 12 subjects were included in the analysis (Figure 6). Six subjects dropped out during the intervention period, one due to relocation to another city and five due to illness unrelated to the study. After completion of the post-tests, four subjects were excluded due to unreliable $\dot{V}O_{2max}$-tests and one was excluded because of illness during the 3000m.
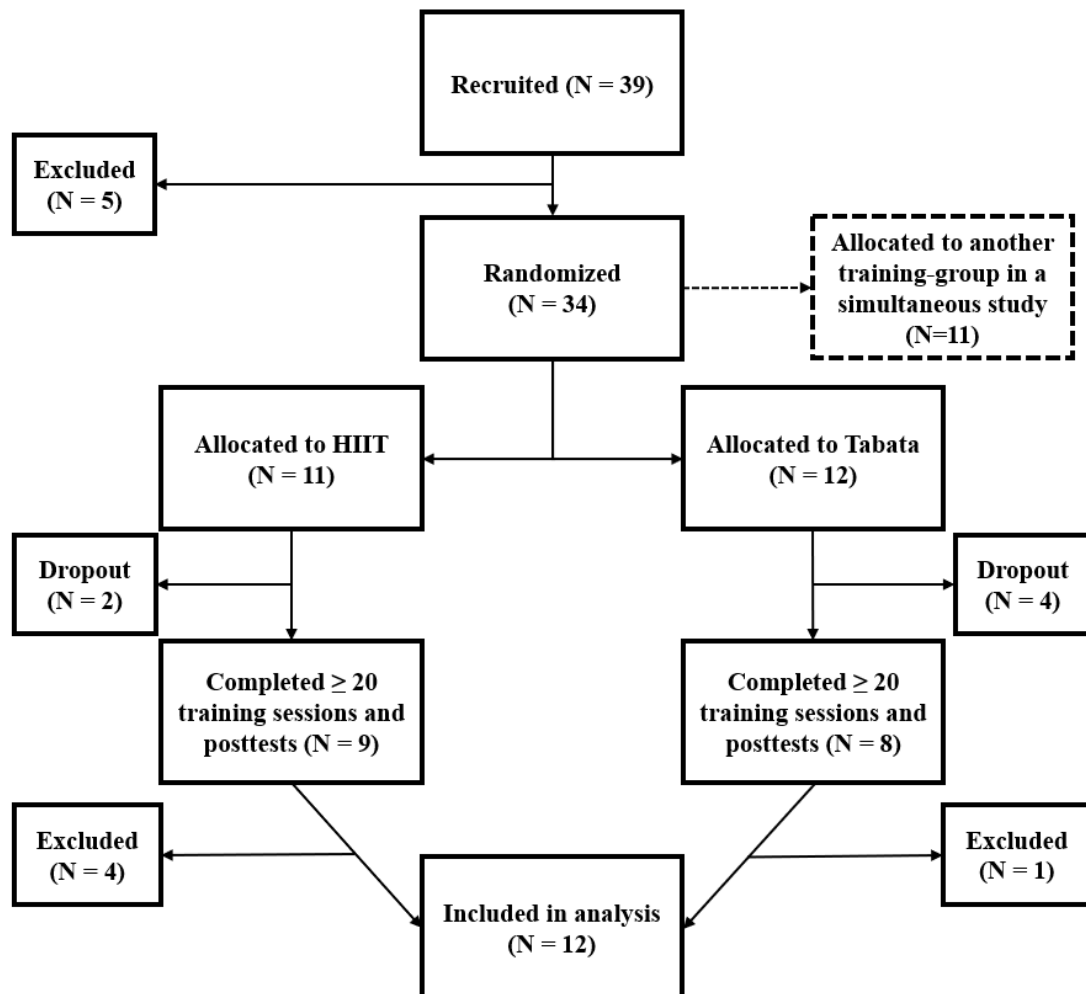

Figure 6. Flow chart of study design.

Table 2. Compliance.

	HIIT (N = 5)	Tabata (N = 7)
Number of training sessions	23 ± 1	22 ± 2
% training sessions (of 24)	98 ± 4	93 ± 8
Avg % HR_{max} after 3min	94 ± 1	-
Avg total time of intervals (sec)	-	156 ± 7
Avg number of intervals	-	7.8 ± 0.4

Data are presented as mean ± SD. HR, heart rate.

Both the HIIT and TT sessions were conducted according to the protocol, and there was no significant difference in compliance between groups (Table 2). At baseline, no parameter was significantly different between groups (Table 1, Table 3 and Table 4).

3.1 $\dot{V}O_{2max}$, RE and LT

HIIT improved $\dot{V}O_{2max}$ and maximal O_2 pulse significantly more than TT (Table 3). The HIIT group significantly increased $\dot{V}O_{2max}$ by 7.9%, 8.9% and 8.6% expressed as $L \cdot min^{-1}$, $mL \cdot kg^{-1} \cdot min^{-1}$ and $mL \cdot kg^{-0.75} \cdot min^{-1}$ respectively, while there was no change in $\dot{V}O_{2max}$ in the TT group (Figure 7). Maximal O_2 pulse increased by 9.3% following HIIT, while no significant change was seen following TT (Figure 7). Differences in RE within or between groups was not observed. HR at 7 $km \cdot h^{-1}$ was significantly more reduced following HIIT than following TT. No differences between groups was found for $\dot{V}O_2$, velocity, or %$\dot{V}O_{2max}$ at LT. $\dot{V}O_2$ at LT ($L \cdot min^{-1}$ and $mL \cdot kg^{-0.75} \cdot min^{-1}$) increased after TT, while no significant change was seen after HIIT. A difference between groups was observed for LT as percentage of HR_{max}, but neither HIIT nor TT demonstrated a significant change within group for this parameter (Table 3). Velocity at LT improved significantly in both groups by 7 - 9%.

Table 3. Endurance performance and its determinants.

	HIIT (N = 5)		Tabata (N = 7)	
	Pre-training	Post-training	Pre-training	Post-training
$\dot{V}O_{2max}$				
$\dot{V}O_2$ (L · min^{-1})	3.22 ± 0.38	3.47 ± 0.38*	3.51 ± 0.42	3.53 ± 0.45#
$\dot{V}O_2$ (mL · kg^{-1} · min^{-1})	54.1 ± 1.9	58.9 ± 3.3*	53.4 ± 3.3	53.2 ± 4.5##
$\dot{V}O_2$ (mL · kg$^{-0.75}$ · min^{-1})	150.2 ± 8.3	163.2 ± 11.1*	151.9 ± 9.8	151.8 ± 13.3##
$\dot{V}_E$ (L · min$^{-1)}$	97.8 ± 20.5	101.5 ± 21.6*	110.3 ± 15.9	111.5 ± 18.8
RER	1.08 ± 0.04	1.16 ± 0.05*	1.09 ± 0.05	1.13 ± 0.04*#
[La$^-$]$_b$ (mM)	10.2 ± 1.6	11.2 ± 2.0*	12.0 ± 2.1	12.6 ± 3.2
HR$_{max}$ (beats · min^{-1})	192 ± 17	190 ± 16	199 ± 7	198 ± 7
O$_2$ Pulse (mL · beat^{-1})	16.8 ± 2.0	18.3 ± 1.6*	17.4 ± 2.3	17.7 ± 2.4#
v$\dot{V}O_{2max}$ (km · h^{-1})	10.9 ± 0.9	12.2 ± 0.5*	11.2 ± 1.1	11.7 ± 1.4*
Running economy				
$\dot{V}O_2$ (L · min^{-1})	2.18 ± 0.28	2.12 ± 0.17	2.31 ± 0.30	2.27 ± 0.28
$\dot{V}O_2$ (mL · kg^{-1} · min^{-1})	36.7 ± 3.1	35.9 ± 1.7	35.2 ± 1.7	34.2 ± 1.6
$\dot{V}O_2$ (mL · kg$^{-0.75}$ · min^{-1})	101.8 ± 8.9	99.5 ± 5.0	100.1 ± 6.2	97.5 ± 5.8
$\dot{V}O_2$ (mL · kg$^{-0.75}$ · m^{-1})	0.872 ± 0.076	0.852 ± 0.042	0.857 ± 0.054	0.834 ± 0.051
HR (beats · min^{-1})	160 ± 21	148 ± 17*	168 ± 11	165 ± 12#
Lactate Threshold				
$\dot{V}O_2$ (L · min^{-1})	2.53 ± 0.36	2.68 ± 0.27	2.63 ± 0.40	2.77 ± 0.41*
$\dot{V}O_2$ (mL · kg^{-1} · min^{-1})	42.4 ± 3.2	45.4 ± 2.1	40.8 ± 3.8	41.7 ± 4.3
$\dot{V}O_2$ (mL · kg$^{-0.75}$ · min^{-1})	117.9 ± 10.3	125.8 ± 7.3	116.0 ± 11.5	119.0 ± 12.9*
% $\dot{V}O_{2max}$	78.5 ± 4.9	77.3 ± 4.9	76.3 ± 5.7	78.5 ± 7.5
% HR$_{max}$	90.8 ± 2.3	89.7 ± 1.8	89.5 ± 1.7	91.2 ± 2.3#
vLT (km · h^{-1})	8.3 ± 0.5	9.1 ± 0.8*	8.3 ± 0.8	8.9 ± 1.1*
[La$^-$]$_b$ (mM)	3.0 ± 0.5	2.7 ± 0.4*	2.8 ± 0.4	2.9 ± 0.4
300 meter (sec)	57.5 ± 3.1	54.6 ± 2.6*	55.5 ± 5.3	52.6 ± 3.8*
3000 meter (sec)	843 ± 38	794 ± 38*	856 ± 81	821 ± 78*
Body mass (kg)	59.5 ± 5.4	58.8 ± 3.7	65.7 ± 7.3	66.3 ± 6.8

Data are presented as mean ± SD. LT, RE and $\dot{V}O_{2max}$ was tested running on a treadmill at 5.3% incline. 300m and 3000m was carried out at an indoor 200m track and field. $\dot{V}O_2$, oxygen uptake; $\dot{V}_E$, pulmonary ventilation; RER, respiratory exchange ratio; [La$^-$]$_b$, blood lactate concentration; HR, heart rate; O$_2$ pulse, oxygen pulse; v$\dot{V}O_{2max}$, minimum theoretical velocity at $\dot{V}O_{2max}$; vLT, velocity at lactate threshold. *Significant difference within group (P < 0.05) from pre- to post-training. #Significant different change (P < 0.05) from pre- to post-training compared to HIIT. ##Significant different change (P < 0.01) from pre- to post-training compared to HIIT.
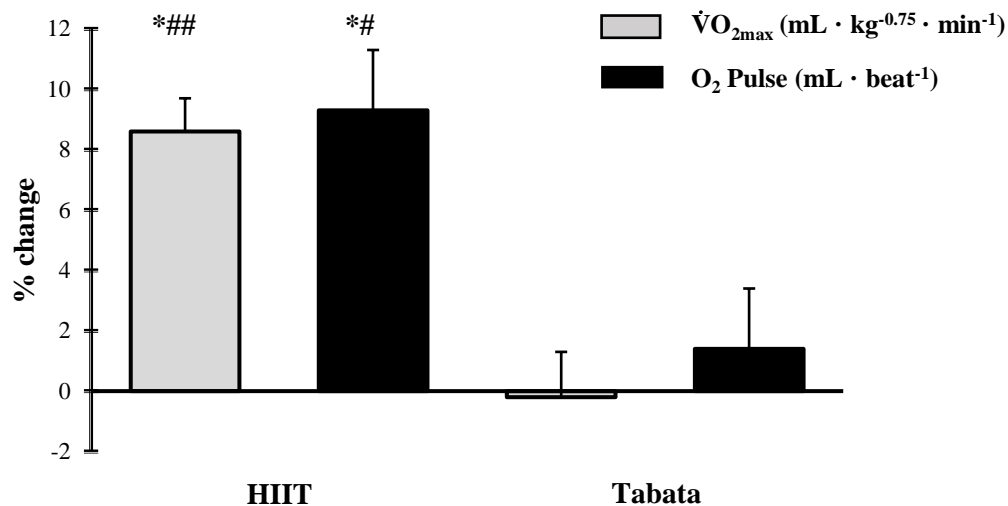
Figure 7. Percentage change in $\dot{V}O_{2max}$ and O_2 pulse from pre- to post-training, presented as mean and standard error of the mean. *Significant difference within group ($P < 0.05$) from pre- to post-training. #Significant difference between groups ($P < 0.05$) from pre- to post-training. ##Significant difference between groups ($P < 0.01$) from pre- to post-training.

3.2 300m and 3000m performance

Both groups significantly reduced their time at both 300m and 3000m, and no differences between groups was found (Table 3). The improvement on 300m was 5.0% in the HIIT group and 4.9% in the TT group. 3000m time was reduced by 5.7% following HIIT and 4.1% following TT.

3.3 Anaerobic capacity – AC / MAOD

No change within groups or differences between groups was observed for AC, measured as MAOD (Table 4).

Table 4. Anaerobic capacity.

	HIIT (N = 5)		Tabata (N = 7)	
	Pre-training	Post-training	Pre-training	Post-training
MAOD				
L	4.07 ± 1.01	4.16 ± 0.89	4.39 ± 0.75	4.46 ± 0.68
mL $\cdot$ kg^{-1}	68.1 ± 13.0	70.3 ± 12.7	65.8 ± 7.0	66.9 ± 4.0
Velocity % v$\dot{V}O_{2max}$	120 ± 6	119 ± 3	119 ± 5	$124 \pm 5*$
Velocity (km $\cdot$ h^{-1})	13.3 ± 0.8	$14.7 \pm 0.7*$	13.6 ± 0.8	$14.7 \pm 1.4*$
Time (sec)	132 ± 28	124 ± 21	147 ± 22	134 ± 19
$\dot{V}O_{2peak}$				
$\dot{V}O_2$ (L $\cdot$ min^{-1})	3.07 ± 0.36	$3.25 \pm 0.28*$	3.38 ± 0.34	3.39 ± 0.45
$\dot{V}O_2$ (mL $\cdot$ kg$^{-1} \cdot$ min^{-1})	51.4 ± 3.0	$55.1 \pm 3.7*$	51.5 ± 2.9	51.2 ± 4.3#
$\dot{V}O_2$ (mL $\cdot$ kg$^{-0.75} \cdot$ min^{-1})	142.9 ± 9.6	$152.5 \pm 10.3*$	146.5 ± 7.4	146.0 ± 13.0#
RER	1.09 ± 0.02	1.14 ± 0.06	1.15 ± 0.02	1.15 ± 0.01
HR$_{peak}$ (beats $\cdot$ min^{-1})	186 ± 14	184 ± 18	191 ± 4	190 ± 4
[La$^-$]$_b$ (mM)	10.8 ± 2.0	11.6 ± 2.2	12.7 ± 2.1	12.9 ± 2.2
% $\dot{V}O_{2max}$ Reached	95.2 ± 4.1	93.7 ± 5.3	96.6 ± 2.9	96.3 ± 5.6
% HR$_{max}$ Reached	97.0 ± 2.2	97.0 ± 3.6	94.6 ± 1.1	95.2 ± 1.1

Data presented as mean $\pm$ SD. MAOD was tested running on a treadmill at 5.3% incline. MAOD, maximal accumulated oxygen deficit; $\dot{V}O_2$, oxygen uptake; RER, respiratory exchange ratio; [La$^-$]$_b$, blood lactate concentration; HR, heart rate. *Significant difference within group ($P < 0.05$) from pre- to post-training. #Significant different change ($P < 0.05$) from pre- to post-training compared to HIIT.

3.4 Correlations

Running performance on the 3000m significantly correlated with $\dot{V}O_{2max}$ as L$\cdot$min^{-1} (R = -0.58, P < 0.01), $\dot{V}O_{2max}$ as mL$\cdot$kg$^{-1}\cdot$min^{-1} (R = -0.74, P < 0.001), $\dot{V}O_{2max}$ as mL$\cdot$kg$^{-0.75}\cdot$min^{-1} (R = -0.76, P < 0.001), v$\dot{V}O_{2max}$ (R = -0.73, P < 0.001), O_2 pulse as mL $\cdot$ beat^{-1} (R = -0.69, P < 0.001), LT as L$\cdot$min^{-1} (R = -0.55, P < 0.01), LT as mL$\cdot$kg$^{-1}\cdot$min^{-1} (R = -0.73, P < 0.001), LT as mL$\cdot$kg$^{-0.75}\cdot$min^{-1} (R = -0.77, P < 0.001), vLT (R = -0.88, P < 0.001) and 300m performance (R = 0.59, P < 0.01).

Running performance on the 300m significantly correlated with $\dot{V}O_{2max}$ as L$\cdot$min^{-1} (R = -0.43, P < 0.05), $\dot{V}O_{2max}$ as mL$\cdot$kg$^{-1}\cdot$min^{-1} (R = -0.51, P < 0.05), $\dot{V}O_{2max}$ as mL$\cdot$kg$^{-0.75}\cdot$min^{-1} (R = -0.51, P < 0.05), v$\dot{V}O_{2max}$ (R = -0.52, P < 0.05), O_2 pulse as mL $\cdot$ beat^{-1} (R = -0.49, P < 0.05), vLT (R = -0.42, P < 0.05) and 3000m performance (R = 0.59, P < 0.01).

4. Discussion

The main findings in the present study are that following eight weeks of three weekly sessions, TT did not change $\dot{V}O_{2max}$ while HIIT improved $\dot{V}O_{2max}$ significantly more than TT. RE was unchanged following both HIIT and TT, but HR at a standardized workload was significantly reduced following HIIT but not TT, with a significant difference between groups observed. Velocity at LT increased in both groups with no difference between groups. The changes in $\dot{V}O_{2max}$ following HIIT was not accompanied by changes in $\dot{V}O_2$ at LT, while the TT group increased $\dot{V}O_2$ at LT following training. No changes in AC occurred in neither of the groups, while performance on 300m and 3000m was significantly improved in both groups without any differences between the groups.

4.1 Maximal oxygen uptake - $\dot{V}O_{2max}$

Relative $\dot{V}O_{2max}$ increased by about 9% following the 4 x 4 minutes HIIT protocol, an improvement in line with other studies using the same protocol (Helgerud et al., 2007; Storen et al., 2017; Wang et al., 2014). The increased $\dot{V}O_{2max}$ and unchanged HR_{max} together resulted in a 9% increase in maximal O_2 pulse, which indicates an increased SV_{max} following HIIT. This indication is based on the findings that HIIT commonly improve $\dot{V}O_{2max}$ by increasing SV_{max}, while other possible underlying factors, such as blood volume and Hb concentration, are unchanged (Helgerud et al., 2007; Wang et al., 2014; Wisloff et al., 2007). Furthermore, Slordahl et al. (2004) demonstrated that moderately trained females increased both left ventricular mass and function concomitantly with an increased $\dot{V}O_{2max}$ following HIIT, suggestive of cardiac adaptations. An additional indication of increased SV following HIIT is the reduced HR at 7 km$\cdot$h^{-1} despite unchanged RE. The increased maximal O_2 pulse and reduced submaximal HR are, in light of previous research, therefore a strong indication that both maximal and submaximal SV did increase following HIIT.

Following TT, no change in $\dot{V}O_{2max}$ was observed. The absence of changes in $\dot{V}O_{2max}$, O_2 pulse and submaximal HR in the present study suggests that no adaptations of SV did occur following TT. The unchanged $\dot{V}O_{2max}$ is in conflict with every study identified by the author where the effect of running or cycling TT on $\dot{V}O_{2max}$ has been investigated (Bonafiglia et al., 2017; Bonafiglia et al., 2016; Foster et al., 2015; Laird et al., 2016; Ravier et al., 2009; Schaun et al., 2018; Scribbans, Edgett, et al., 2014; Scribbans, Ma, et al., 2014; Tabata et al., 1996). It should however be mentioned that Bonafiglia et al. (2016) reported improved

$\dot{V}O_{2max}$ following TT only when the male (N = 9) and female (N = 12) samples were pooled, while no change in $\dot{V}O_{2max}$ was found when analyzing the sexes separately.

Several studies have previously demonstrated that TT are equally effective as MCT in improving $\dot{V}O_{2max}$ (Bonafiglia et al., 2016; Foster et al., 2015; Schaun et al., 2018; Scribbans, Edgett, et al., 2014). MCT do commonly not improve $\dot{V}O_{2max}$ in subjects with baseline values above average (Esfarjani & Laursen, 2007; Helgerud et al., 2007), and the subjects in the present study had a baseline $\dot{V}O_{2max}$ of approximately 25% above the average Norwegian healthy woman between 20 and 29 years of age (Loe et al., 2013). Based on previous findings, it is therefore not surprising that TT was an ineffective modality for improving $\dot{V}O_{2max}$ in the well-trained subjects of the present study. The previous indirect findings, e.g. in a recent meta-analysis (Williams et al., 2019), indicative of a superior effect on $\dot{V}O_{2max}$ exerted by HIIT compared to TT, are thus supported by the direct comparison in the present study.

Of all the previous TT studies, only the subjects in Ravier et al. (2009) can be characterized as above averagely trained. The male karate athletes improved their $\dot{V}O_{2max}$ significantly from 58.7 mL·kg^{-1}·min^{-1} to 61.4 mL·kg^{-1}·min^{-1} following seven weeks of two weekly TT sessions in addition to continuing their normal activities (Ravier et al., 2009). Since training intensity, volume and frequency in total was greater in the present study, the logical explanation for the discrepancy with Ravier et al. (2009) are their relatively poorer baseline $\dot{V}O_{2max}$, which corresponds to 8% above the average for young and healthy males (Aspenes et al., 2011; Loe et al., 2013).

In contrast to previous investigations of TT, several previous HIIT studies have included male samples with a mean baseline $\dot{V}O_{2max}$ above average (54 mL·kg^{-1}·min^{-1}) (Esfarjani & Laursen, 2007; Helgerud et al., 2001; 2007; 2011; Laursen et al., 2002, 2005). However, no study including exclusively female subjects with a mean baseline $\dot{V}O_{2max}$ above average (43 mL·kg^{-1}·min^{-1}) have, to the author's awareness, been published. The studies by Bishop et al. (2008), Slordahl et al. (2004) and Edge et al. (2006) all included female samples which increased $\dot{V}O_{2max}$ with 10% - 18% following HIIT, from a baseline of ~43 mL·kg^{-1}·min^{-1}. These three studies (Bishop et al., 2008; Edge et al., 2006; Slordahl et al., 2004) represents, to the author's awareness, the most well-trained female samples investigated following a HIIT intervention. Thus, the female sample in the present study are unique, indicating that well-trained women respond to HIIT in the same manner as well-trained men.

In averagely and well-trained subjects, HIIT commonly increase $\dot{V}O_{2max}$ via an increased SV_{max} (Helgerud et al., 2007; 2009; Wang et al., 2014). It is recommended that to improve SV_{max}, especially in well-trained subjects, the heart should be highly stimulated, reaching a high $SV/\dot{Q}$ during exercise (Helgerud et al., 2007; Hoff & Helgerud, 2004; Wang et al., 2012). In well-trained individuals, both HR and SV increase with increasing intensity up to $\dot{V}O_{2max}$, meaning that SV does not plateau at submaximal intensities as previously thought (Wang et al., 2012; Zhou et al., 2001). Therefore, training at intensities close to maximal is required to reach a high $\dot{Q}$ and sufficiently overload and stimulate the heart in well-trained subjects.

In the present investigation, the mean number of intervals completed during TT was 7.8 (Table 2), meaning that subjects often were exhausted during the eighth interval. $\dot{V}O_{2max}$ are commonly reached in a TT session where the subjects are exhausted during bout number 6 - 8 (Tabata et al., 1997; Viana, Naves, et al., 2018). Furthermore, according to Viana, Naves, et al. (2018), 53 ± 49 seconds are spent above 90% of $\dot{V}O_{2max}$ during a single TT session. These data are collected during cycling TT, but may also be representative for running TT. Thus, even though the TT protocol is at a supramaximal workload for about 160 seconds (8 x 20sec, excluding breaks), the oxygen transporting system are not highly taxed for more than about one minute. Therefore, based on the findings in the present investigation, one may suggest that the relatively short time (volume) at high-intensity during TT is insufficient to induce cardiac adaptations in a well-trained sample. However, when including subjects with relatively low $\dot{V}O_{2max}$, it is not surprising that previous studies have demonstrated effects of TT, since the relative improvement in $\dot{V}O_{2max}$ are highly influenced by a subject's baseline $\dot{V}O_{2max}$ (Storen et al., 2017).

During the 4-minute intervals in the present study, mean HR after 3 minutes was 94% of HR_{max}, which may roughly translate to 85 - 90% of $\dot{V}O_{2max}$ (Swain, Abernathy, Smith, Lee, & Bunn, 1994). The HIIT intervals are not of maximal intensity, and the subjects are not pushing themselves to exhaustion during a 4 x 4 HIIT session (Storen et al., 2017). Therefore, in contrast with TT, $\dot{V}O_{2max}$ is not reached during HIIT. However, the time spent around 90% of $\dot{V}O_{2max}$ are much greater during HIIT compared to TT, as indicated by the mean HR in the present study. In light of the results presented, the slightly higher percentage of $\dot{V}O_{2max}$ reached during TT compared to HIIT are not able to compensate for the higher volume at about 90% of $\dot{V}O_{2max}$ during HIIT.

Since the purpose of the present investigation was to compare the original TT protocol to the 4 x 4 minutes HIIT protocol, it is impossible to make the two interventions isocaloric. If one

were to make the protocols isocaloric, one or both protocols would have to be compromised compared to the original intention. This would lead to decreased intensity during TT and/or decreased volume of HIIT.

Verification of $\dot{V}O_{2max}$ using the MAOD protocol

During the MAOD test both pre- and post-training, average peak oxygen uptake ($\dot{V}O_{2peak}$) was significantly lower ($P \leq 0.01$, N = 12) compared to average $\dot{V}O_{2max}$ measured during the incremental test to exhaustion (Table 4). In this case, $\dot{V}O_{2peak}$ refers to the highest 10 seconds average $\dot{V}O_2$ measured. Thus, the supramaximal MAOD protocol cannot be regarded as a suitable strategy for verification of $\dot{V}O_{2max}$. In the present study, all subjects demonstrated a plateau (or slight decrease) of $\dot{V}O_2$ at the end of the incremental $\dot{V}O_{2max}$-test. In order to verify an individual's $\dot{V}O_{2max}$, Poole and Jones (2017) emphasized the need of a verification phase following an incremental test to exhaustion. However, the inclusion of a verification phase as a gold standard has later been questioned (Murias, Pogliaghi, & Paterson, 2018). Furthermore, even though a verification phase may be necessary in some populations, this is probably not the case in a young, healthy and well-trained sample (Poole & Jones, 2017).

4.2 Running economy - RE

No changes in RE was observed following training in the present study. Although improvements in RE is normal following HIIT (Baekkerud et al., 2016; Helgerud et al., 2007), unchanged RE are also quite common in well-trained samples (Denadai et al., 2006; Impellizzeri et al., 2006). The lack of significant change in RE in the present study may be explained by the high training status of the subjects combined with the low-volume interventions, but this cannot be concluded.

Only one study has previously investigated RE following TT, reporting a worsened RE following both TT and MCT (Schaun et al., 2018). Improved or unchanged RE are normally reported after a MCT intervention (Baekkerud et al., 2016; Helgerud et al., 2007; Macpherson, Hazell, Olver, Paterson, & Lemon, 2011; Patton & Vogel, 1977), and it may therefore be questioned whether the reports of worsened RE in Schaun et al. (2018) are valid. In the article by Schaun et al. (2018), the authors state that the reason for the worsened RE are unclear.

Since the present study measured RE at 5.3% inclination, comparison with most other studies are difficult. Some other studies have reported RE as allometrically scaled $\dot{V}O_2$ at 5.3% - 5.5% inclination (Helgerud et al., 2001; 2007; 2011), but comparison of values across studies should be interpreted with caution since methodological differences likely exists (e.g. actual velocity, actual inclination, and softness of the treadmill belt). The RE of the male subjects in Helgerud et al. (2001; 2007; 2011) ranges from 0.75 to 0.85 $mL \cdot kg^{-0.75} \cdot m^{-1}$ at baseline and 0.70 to 0.82 $mL \cdot kg^{-0.75} \cdot m^{-1}$ post training, values that are generally lower (i.e. better) than what is seen in the present investigation. However, studies comparing allometrically scaled RE directly between men and women report superior RE in women (Helgerud, 1994; Helgerud et al., 2010). Furthermore, data from our research group demonstrate that well-trained males with a baseline $\dot{V}O_{2max}$ of 62 $mL \cdot kg^{-1} \cdot min^{-1}$ exhibited a RE of about 0.90 $mL \cdot kg^{-0.75} \cdot m^{-1}$, undergoing the same test-procedures in the same laboratory as the present investigation (Hemmingsen, 2018; Lim, 2018; Trane, 2018). In comparison, baseline RE in the present study was 0.86 $mL \cdot kg^{-0.75} \cdot m^{-1}$ (N=12). These previous data from our research group together with the present investigation may thus support the previous reports of superior RE in women compared to men, when expressed with body mass raised to the power of 0.75.

4.3 Lactate threshold - LT

There was no significant difference between the effect of HIIT and TT on LT given as %$\dot{V}O_{2max}$, $\dot{V}O_2$ or velocity. The finding that LT as %$\dot{V}O_{2max}$ was unchanged following training in an above averagely trained sample are in line with many other investigations (Helgerud et al., 2001; 2007; Impellizzeri et al., 2006; Sjodin et al., 1982). To the author's knowledge, the uncontrolled study by Acevedo and Goldfarb (1989) is the only investigation reporting a significantly increased LT as %$\dot{V}O_{2max}$ in relatively well-trained subjects (N = 7 men, baseline $\dot{V}O_{2max}$ = 65 $mL \cdot kg^{-1} \cdot min^{-1}$).

On the other hand, samples of untrained subjects sometimes demonstrate large increases in LT as %$\dot{V}O_{2max}$, like the improvement of ~20% in Burke et al. (1994), ~10% (at 2.5 mM) in Hurley et al. (1984) and ~24% in Weltman et al. (1992). This indicates that LT as %$\dot{V}O_{2max}$ may increase substantially in untrained subjects, but not in well-trained subjects. Therefore, the present investigation are, together with existing research, in line with the suggestion by Sjodin and Svedenhag (1985) that an increased LT as a percentage of $\dot{V}O_{2max}$ primarily occurs as an early training response, but not in already well-trained subjects.

Following TT, vLT and $\dot{V}O_2$ at LT expressed as both L·min^{-1} and mL·kg$^{-0.75}$·min^{-1} increased significantly, while $\dot{V}O_2$ at LT expressed as mL·kg^{-1}·min^{-1} showed only a tendency to increase (P = 0.06). The mean increase in $\dot{V}O_2$ and velocity at LT following TT equals ~3% and ~7%, respectively. Since neither $\dot{V}O_{2max}$, LT as %$\dot{V}O_{2max}$ nor RE changed significantly following TT, the underlying mechanisms for the improved $\dot{V}O_2$ and velocity at LT are unclear. However, one possible explanation could be that insignificant changes in RE (~3%) and LT as %$\dot{V}O_{2max}$ (~3%) together contributed to the improved LT expressed as $\dot{V}O_2$ and velocity, but these speculations cannot be confirmed.

The present finding that $\dot{V}O_2$ at LT was not statistically improved following HIIT, despite a large increase in $\dot{V}O_{2max}$, are in contrast with other studies (Helgerud et al., 2001; Impellizzeri et al., 2006), but in line with some (Helgerud et al., 2007). In the present investigation, one may speculate that an outlier is responsible for the lack of significant change of $\dot{V}O_2$ at LT following HIIT. One subject in the HIIT group experienced a reduction of 0.5 mL·kg^{-1}·min^{-1} at LT, while all other subjects experienced an increase of 1.7 mL·kg^{-1}·min^{-1} (4%) or more. Another possible reason for the lack of significant change in $\dot{V}O_2$ at LT are the finding that [La$^-$]$_b$ at LT was significantly reduced from 3.0 mM to 2.7 mM following training (Table 3). If a fixed [La$^-$]$_b$ of for example 2.8 mM were to be used as LT in the HIIT group, both absolute and relative $\dot{V}O_2$ at LT would be significantly increased following training by 8 - 10% (P < 0.05). Therefore, the lack of significant improvement in $\dot{V}O_2$ at LT following HIIT could possibly be explained by the reduction in [La$^-$]$_b$ after warm-up and consequently at LT (see 2.2 - $\dot{V}O_{2max}$, RE and LT). Interestingly, by using a fixed [La$^-$]$_b$ as LT, $\dot{V}O_2$ at LT would be significantly increased following HIIT and not TT. This contrasts with the present findings (Table 3) and illustrates that the method of choice may affect the interpretation of how LT change following training interventions.

4.4 Anaerobic capacity – AC
AC, measured as MAOD, did not change from pre- to post-training following HIIT or TT. The present study is, to the author's awareness, the first investigation of how MAOD may adapt following TT in females. Furthermore, the present investigation is also the first to examine how HIIT, as defined in the introduction, may affect AC.

The finding that AC was unaltered by TT are in conflict with the results from Tabata et al. (1996) and Ravier et al. (2009), which both reported large increases in AC (28% and 10%,

respectively) in male subjects. The reason why TT did not improve AC in the present study are not clear, and possible underlying mechanisms were not investigated.

Some of the discrepancy between the present study and Tabata et al. (1996) may be explained by the methodological differences, since Tabata et al. (1996) did not reassess the relationship between $\dot{V}O_2$ and power post-training. Many investigators do assume that the relationship between $\dot{V}O_2$ and velocity/power are unchanged following a training intervention, often based on the finding that cycling or running economy are unchanged (Medbo & Burgers, 1990; Ramsbottom et al., 2001; Tabata et al., 1996; Weber & Schneider, 2002). Even though RE post-training are not significantly different from pre-training, small individual changes in RE may nonetheless result in altered regression lines and thus a miscalculation of MAOD. In the present study, the TT group would have demonstrated an 11% ($P < 0.05$) increased MAOD following training if unchanged $\dot{V}O_2$ at submaximal workloads were assumed at post-training. This example underlines the necessity to reestablish the relationship between $\dot{V}O_2$ and velocity/power post-training, and raise doubt about the findings of studies failing to do so, including the study by Tabata et al. (1996).

The mean MAOD of ~67 mL·kg^{-1} in the present investigation is greater than what is normally reported in females during running, and more comparable to normal values for male subjects (Naughton et al., 1997; Ramsbottom et al., 1997; Ravier et al., 2009; Scott et al., 1991). Demonstrating higher values than normal, Hill and Vingren (2014) reported mean running MAOD of 60 mL·kg^{-1} and 80 mL·kg^{-1} in 119 women and 104 men, respectively. However, comparisons of MAOD between studies should be interpreted with caution since several methodological dissimilarities between studies affects the results. It is for example known that MAOD increases with treadmill inclination (Olesen, 1992) and that the curvilinear relationship between $\dot{V}O_2$ and velocity assumed in Hill and Vingren (2014) overestimate MAOD by about 30% compared to the more commonly used linear relationship (Hill & Vingren, 2011). Due to methodological differences between studies, robust MAOD reference data are lacking and it is therefore difficult to conclude how well-developed AC the subjects in the present study possess. There are however no reports, to the author's awareness, of female samples with a mean MAOD above 67 mL·kg^{-1}, indicating that the subjects in the present study have a quite high AC. Therefore, the relatively high AC at baseline in the present investigation may explain the conflicting adaptations of AC following TT in our study compared to Ravier et al. (2009) and Tabata et al. (1996).

4.5 3000m performance

There was no significant difference between the effect of HIIT and TT on 3000m performance. Mean 3000m performance improved by 49 seconds (6%) and 35 seconds (4%) following HIIT and TT, respectively. To the author's awareness, this is the first study to investigate how TT affects performance in a running time trial. The percentage change in 3000m time following HIIT was comparable to, but slightly lower than, the 7% in Esfarjani and Laursen (2007) and 8 - 9% in Cicioni-Kolsky et al. (2013).

The finding that $\dot{V}O_{2max}$ increased significantly more following HIIT than TT was not reflected by the 3000m results, despite a significant correlation of R = -0.76 (P < 0.001) between $\dot{V}O_{2max}$ (mL·kg$^{-0.75}$·min^{-1}) and 3000m time. The reason why 3000m performance improved similarly following HIIT and TT, despite large group differences in $\dot{V}O_{2max}$ alterations, are unclear.

Pooled 3000m data gives a mean ± SD of 830 ± 66 seconds, a SD corresponding to 8% of the mean. The SEE of $\dot{V}O_{2max}$ (mL·kg$^{-0.75}$·min^{-1}) as a predictor of 3000m performance was 45 seconds, corresponding to 5% of the average time to complete the 3000m. It is suggested that during a 3000m / ~800 seconds long race, aerobic sources contribute with about 90% of the total energy (Busso & Chatagnon, 2006; Duffield, Dawson, & Goodman, 2005b). The estimates of ~90% aerobic contribution together with the present findings demonstrate that even though 3000m performance is strongly dependent on $\dot{V}O_{2max}$, this parameter is not the sole determinant of endurance performance.

vLT was the parameter investigated which correlated most strongly with 3000m performance (R = -0.88, P < 0.001), an expected finding since vLT are dependent on both $\dot{V}O_{2max}$, RE and LT as %$\dot{V}O_{2max}$ (Bassett & Howley, 2000; Joyner, 1991). Additionally, vLT was also the factor which most accurate could predict 3000m running performance, with a SEE of 32 seconds (4%). Combining $\dot{V}O_{2max}$ (L·min^{-1}), LT (%$\dot{V}O_{2max}$) and RE (L·min^{-1}) gives a R^2 of 0.87 and a SEE of 25sec / 3%, which clearly demonstrates the strong relationship between these determinants and 3000m performance in the present investigation. Importantly, when both $\dot{V}O_{2max}$ and RE are independent variables describing a performance (time/velocity), the results are indifferent to the method of scaling. This is exemplified by that $\dot{V}O_{2max}$ (mL·kg$^{-0.75}$·min^{-1}), LT (%$\dot{V}O_{2max}$) and RE (mL·kg$^{-0.75}$·m^{-1}) also gives a R^2 of 0.87 and a SEE of 25sec / 3%.

Performance on 3000m showed a similar correlation with allometrically scaled $\dot{V}O_{2max}$ ($mL \cdot kg^{-0.75} \cdot min^{-1}$) (R = -0.76) as with ratio scaled $\dot{V}O_{2max}$ ($mL \cdot kg^{-1} \cdot min^{-1}$) (R = -0.74), and the SEE equaled 5-6% of the average performance on 3000m. Even though there is no doubt $\dot{V}O_{2max}$ should be allometrically scaled to best eliminate the effect of body mass, there is limited data regarding which scaling method that best correlate with and predicts distance running performance. One previous investigation suggests that when expressed as $mL \cdot kg^{-1} \cdot min^{-1}$, $\dot{V}O_{2max}$ is better correlated with endurance performance (avg. velocity during a 5k time-trial) than when it is expressed as $mL \cdot kg^{-0.67} \cdot min^{-1}$ (Nevill, Ramsbottom, & Williams, 1992). However, in contrast to Nevill et al. (1992), Tartaruga, Mota, Peyre-Tartaruga, and Brisswalter (2014) demonstrated that $\dot{V}O_{2max}$ expressed as $mL \cdot kg^{-0.75} \cdot min^{-1}$ and $mL \cdot kg^{-1} \cdot min^{-1}$ correlated equally well with running performance (3000m – 10.000m). To the author's knowledge, no other studies have investigated the relationship between distance running performance and these different scaling methods.

4.6 300m performance

There was no significant difference between the effect of HIIT and TT on 300m time trial in the present study. Performance on 300m improved significantly by 2.9 seconds and ~5% following both HIIT and TT. The author is not aware of other studies investigating 300m performance following HIIT or TT.

In the present study, 300m performance correlated significantly with $\dot{V}O_{2max}$ ($mL \cdot kg^{-0.75} \cdot min^{-1}$) (R = -0.51, P < 0.05). The SEE of $\dot{V}O_{2max}$ ($mL \cdot kg^{-0.75} \cdot min^{-1}$) as a predictor of 300m performance was 3.8 seconds, corresponding to 7% of the average 300m performance. A significant relationship between $\dot{V}O_{2max}$ and 300m performance could be expected since aerobic metabolism contributes to about 32 – 45% of the total energy used during a 50 - 60sec running event (Duffield, Dawson, & Goodman, 2005a; Hill, 1999; Nummela & Rusko, 1995; Spencer & Gastin, 2001). However, pooling the 300m data gives a mean ± SD of 54.9 ± 4.2 seconds, and it is worth noting that the SD equals 8% of the mean. This indicates that $\dot{V}O_{2max}$ ($mL \cdot kg^{-0.75} \cdot min^{-1}$) is a quite poor predictor of 300m performance in the present study, since the SEE (3.8sec) is almost as large as the SD (4.2sec).

The 300m running performance test used in the present study may be regarded as an equivalent to the frequently used Wingate test, since mean power during a Wingate test has been shown to correlate with 300m time (R = -0.64, P ≤ 0.05) (Scott et al., 1991). Following

TT, several studies have used the 30sec Wingate test (or a modified version) as a measure of anaerobic adaptations (Foster et al., 2015; Laird et al., 2016; Scribbans, Edgett, et al., 2014). Scribbans, Edgett, et al. (2014) reported that TT was superior to MCT in eliciting anaerobic improvements. However, Foster et al. (2015) reported significant anaerobic adaptations following both TT and MCT, with no difference between groups. Laird et al. (2016) reported increased mean and peak power following both TT and concurrent TT and strength training. Thus, the decreased time to complete a 300m time trial in the present study are in line with previous TT studies that demonstrates improved mean power during Wingate tests. It should be noted that there are obvious and major differences between a 300m running time trial and a Wingate test, for example the modality (running vs cycling) and the time used (~55sec vs 30sec). These differences, together with the relatively modest correlation of R = -0.64, underlines that comparison between the tests should be interpreted with caution.

In the present study, MAOD did not correlate with 300m time trial. MAOD have previously been shown to correlate with running performance on 100m (Ramsbottom, Nevill, Nevill, Newport, & Williams, 1994) and 300m (Scott et al., 1991), while reports of whether MAOD correlates with 400m performance are conflicting (Ramsbottom et al., 1994; Scott et al., 1991). Additionally, data from our research group are equivocal regarding whether 300m and MAOD are significantly correlated in well-trained men (Hemmingsen, 2018; Lim, 2018; Trane, 2018). Taken together, a significant relationship between MAOD and running performance on events lasting ≤ 60 seconds are not a universal finding. Additionally, it should be noted that there is conflicting evidence whether significant correlations between MAOD and power during Wingate tests exists (Minahan, Chia, & Inbar, 2007; Scott et al., 1991).

Since MAOD relatively often does not display a significant relationship with performance at 30-60 seconds running or cycling, its use as a predictor of performance in such events may be questioned. This does not necessarily indicate that MAOD is an invalid test of AC, but rather that performance during short events (≤ 60sec) depends on several parameters concomitantly, such as $\dot{V}O_{2max}$, maximal sprinting velocity, sprinting economy and counter movement jump as an indicator of muscular power (Dal Pupo et al., 2013; Nummela, Mero, Stray-Gundersen, & Rusko, 1996). Importantly, the lack of a significant correlation in some studies (e.g. the present study), should not be regarded as proof of no relationship between two parameters. For example, it would be fallacious to suggest that RE does not play a part in 3000m performance because a lack of correlation was evident in the present study.

4.7 Study Limitations

The small sample sizes, especially in the HIIT group, are a limitation to the present investigation. Therefore, one should be careful to generalize the findings. It would have strengthened the study if one or several mechanisms associated with the determinants of endurance performance were investigated, e.g. blood volume, Hb concentration, MCT1 and MCT4 density or aerobic and anaerobic enzyme activity. Additionally, the calculation of O_2 pulse are not as valid as more direct tests for measuring/estimating SV. Furthermore, most subjects were unfamiliar with the performance tests despite their well-developed fitness level. Even though time trials in general are more reliable than time to exhaustion tests (Laursen, Francis, Abbiss, Newton, & Nosaka, 2007), a single familiarization session would probably improve the reliability of the performance tests. Finally, by including comparable groups of male subjects in HIIT and TT, a more thorough analysis of sex differences would have been possible.

5. Future research

Future research should aim to investigate whether AC are equally trainable in women and men, and whether HIIT may improve AC in groups with a lower baseline MAOD than in the present study. The relationship between MAOD and performance on both 300m and 3000m are still unclear, and this should be examined in larger and more heterogenous samples. Furthermore, potential underlying mechanisms responsible for sex differences in AC and RE should be investigated with large sample sizes. Finally, the present findings need verification by a larger study including more subjects, since the statistical foundation of the present work are relatively scarce.

6. Conclusion

It is concluded that 4 x 4 minutes HIIT at 90 - 95% of HR_{max} is more effective than TT in improving $\dot{V}O_{2max}$ in a sample of well-trained females. Furthermore, TT seems to be an ineffective strategy for improving $\dot{V}O_{2max}$ in well-trained women, as demonstrated by no change following 8 weeks of training in the present investigation. Both groups improved performance on both 300m and 3000m, with no significant difference between groups. LT as a percentage of $\dot{V}O_{2max}$, RE and AC did not change in neither group. This is the first investigation of TT on well-trained females, and the lack of improvement in $\dot{V}O_{2max}$ and AC

contrast with other studies which are conducted mainly on poor to averagely trained males. Therefore, further research on TT including well-trained subjects, both males and females, are needed.

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