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Master thesis

Interval-training at higher percentages of VO_{2max} induces greater training adaptions than lower percentages in cyclists

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Abstract

Purpose: Training at high intensities is well known to improve endurance performance, yet the importance of training intensity measured as percentage of maximal oxygen consumption (VO_{2max}) has never been verified. Thus, the present study aimed to investigate the importance of training intensity measured as the average percentage of VO_{2max} elicited during every interval-sessions ($\%VO_{2max@IS}$) during an entire training intervention on changes in endurance performance and physiological determinants of endurance performance in cyclists.

Methods: Twenty-two cyclists (VO_{2max} = 67.1 ± 6.4 $mL \cdot min^{-1} \cdot kg^{-1}$; males, *n* = 19; females, *n* = 3) performed a nine-weeks training intervention including twenty-one 5x8-minutes interval-sessions at a mean power output (PO) corresponding to the individual cyclists´ 40-minutes PO. Oxygen consumption was measured during all interval-sessions, and the half of the cyclists eliciting the highest $\%VO_{2max@IS}$ were allocated to HIGH ($\%VO_{2max@IS}$ = 86.2 ± 3.8%; *n* = 11) whereas the half eliciting the lowest $\%VO_{2max@IS}$ were allocated to LOW ($\%VO_{2max@IS}$ = 79.9 ± 4.0%; *n* = 11). Physiological tests were performed prior to (pre), two times during, and after the intervention (post).

Results: Across both groups, the pre to post percentage increase in PO at 4 $mmol \cdot L^{-1}$ blood lactate concentration ($PO_{@4mmol}$; *p* = 0.001) and in a performance index (composed of $PO_{@4mmol}$, maximal aerobic PO (W_{max}), and 15-minutes PO ($PO_{@15min}$); *p* = 0.042) were both positively related to $\%VO_{2max@IS}$. Comparing HIGH to LOW, larger percentage increases were observed in VO_{2max} (8.0 ± 4.3% and 2.7 ± 2.7%, *p* = 0.003), W_{max} (8.6 ± 5.6% and 4.2 ± 2.8%, *p* = 0.035), $PO_{@4mmol}$ (7.8 ± 3.6% and 3.0 ± 4.5%, *p* = 0.013), and the performance index (7.7 ± 3.4% and 3.6 ± 2.9%, *p* = 0.007).

Conclusion: Performing twenty-one interval-sessions over nine weeks at a higher $\%VO_{2max@IS}$ induces greater training adaptions than at a lower $\%VO_{2max@IS}$ in cyclists.

Table of contents

1 Theory

1.1 Endurance performance

A fundamental principle in exercise physiology is that work requires energy (Basset & Howley, 2000). Endurance performance is to a great extent determined by the body´s or more specifically the muscle mitochondria´s ability to convert stored energy in fat and carbohydrate to adenosine triphosphate (ATP) that can be utilized by the muscle fibers to create movement (Tønnessen & Rønnestad, 2018). As the mitochondria is an aerobic i.e., oxygen (O_2) dependent energy system, the rate at which O_2 is used is a measure of the total work that can be performed during prolonged submaximal exercise (Basset & Howley, 2000). Endurance performance is hence primarily determined by the maximal oxygen consumption (VO_{2max}), in addition to the fractional utilization of VO_{2max} (%VO_{2max}), and exercise efficiency (Joyner & Coyle, 2008). The theory part of this thesis will address what determents these physiological factors and how they are affected by training at high percentages of VO_{2max}.

1.2 VO_{2max}

VO_{2max} is defined as the highest rate at which O_2 can be consumed and utilized by the body during exercise (Basset & Howley, 2000). Thus, VO_{2max} sets an upper limit for the body´s energy production, and consequently also for endurance performance (Basset & Howley, 2000). In both untrained and well-trained individuals (Lundby & Robach, 2015), regularly endurance training leads to several physiological adaptions potentially influencing VO_{2max} (Hawley et al., 2014; Hellsten & Nyberg, 2015). For most endurance athletes it is assumed that the upper limit for VO_{2max} is within the range of 83-85 $mL \cdot min^{-1} \cdot kg^{-1}$ (Lundby & Robach, 2015), yet the highest value ever published is 96.7 $mL \cdot min^{-1} \cdot kg^{-1}$ (Rønnestad et al., 2019). In theory, VO_{2max} may be limited by any factor influencing the cardiorespiratory system´s ability to deliver O_2 to the exercising muscles (i.e., the lungs´ diffusion capacity, the maximal cardiac output (Q_{max}), and the blood´s O_2-carrying capacity; central factors) and the muscle mitochondria´s ability to consume O_2 (i.e., skeletal muscle characteristics; peripheral factors; Basset & Howley, 2000; Lundby & Montero, 2015).

1.2.1 The diffusion capacity of the lungs

The lungs´ diffusion capacity is the first potential limiting factor in the O_2 delivery and utilization chain (Basset & Howley, 2000). The lungs´ job is to saturate the arterial blood with O_2, and as the arterial O_2 saturation remains almost as high during maximal work as at rest

(~95 and ~98%, respectively; Mortensen et al., 2005), the lungs are normally not considered a limiting factor of VO_{2max} at sea level (Basset & Howley, 2000; Powers et al., 1989). However, as an increased Q_{max} decreases the mean transit time in the pulmonary capillaries (Dempsey et al., 1984), the lungs´ diffusion capacity may present itself as a limiting factor in well-trained athletes (Basset & Howley, 2000; Dempsey et al., 1984; Dempsey et al., 2003; Powers et al., 1989). This was demonstrated by Powers et al. (1989), who observed that highly trained subjects increased their O_2 saturation from 90.6 to 95.9% and VO_{2max} from 70 to 75 $mL \cdot min^{-1} \cdot kg^{-1}$ breathing O_2-enriched air (26% O_2), whereas no changes were observed in normal subjects. Notably, the structural and functional properties of the lungs do not appear to change in response to endurance training (McKenzie, 2012).

1.2.2 Maximal cardiac output & the O_2-carrying capacity of the blood

The O_2 delivery capacity is a product of Q_{max} and the O_2-carrying capacity of the blood, and is considered the main limitation to VO_{2max} during exercise recruiting a large muscle mass at sea level (di Prampero, 2003; di Prampero & Ferretti, 1990; Lundby et al., 2017; Mortensen et al., 2005). That is, as the skeletal muscles´ mitochondrial oxidative capacity is observed to exceed the cardiorespiratory system´s ability to supply the exercising muscles with O_2 (Boushel et al., 2011; Boushel & Saltin, 2013; Mortensen et al., 2005). Consistently, it has been demonstrated that when removing a training-induced gain in blood volume (BV), the concomitantly increased Q_{max} and VO_{2max} are reverted to baseline (Bonne et al., 2014; Montero et al., 2015).

Q_{max} is a product of the heart´s maximal stroke volume (SV_{max}) and maximal heart rate (HR_{max}), and as HR_{max} is close to identical in elite athletes and untrained individuals, the increased Q_{max} of elite athletes must be attributed to an increased SV_{max} (Lundby & Robach, 2015). An increased SV_{max} could be a result of an increased end-diastolic volume due to cardiac eccentric hypertrophy, increased left ventricular compliance, and/or reduced peripheral vascular resistance (Fleg et al., 1994; Klausen et al., 1982; Levine et al., 1991; Spence et al., 2011). Moreover, an expanded BV is argued to be of the utmost importance as it increases the venous return leading to an increased end-diastolic volume, and stroke volume (SV) via the Frank-Starling mechanism (Convertino et al., 1991; Coyle et al., 1986; Hopper et al., 1988; Kanstrup & Ekblom, 1982). As Q_{max} is reverted to pre-training values when the training-induced gain in BV is removed (Bonne et al., 2014; Montero et al., 2015), it has been suggested that an increased Q_{max} in untrained individuals is exclusively attributed to BV expansion (Lundby et al., 2017). Noteworthy, the Q_{max} (Ekblom & Hermansen, 1968) and BV

(Heinicke et al., 2001) of elite athletes are observed to be almost twice that of healthy untrained individuals.

However, it has been argued that a BV-mediated increase in Q_{max} has little impact on O_2 delivery and hence VO_{2max} if only the plasma volume (PV), and not also the red blood cell volume (RBCV) is expanded (Keiser et al., 2015; Lundby et al., 2017; Montero et al., 2015; Warburton et al., 2000). That is because the blood´s O_2-carrying capacity is determined by the number of circulating red blood cells and more specifically, the hemoglobin molecules inside them (Lundby & Montero, 2019; Lundby et al., 2017). As 1 g hemoglobin binds 1.34 mL O_2 (Heinicke et al., 2001), an expanded RBCV and increased total hemoglobin mass (Hb_{mass}) would enhance the O_2-carrying capacity, O_2 delivery, and consequently VO_{2max} (Lundby & Montero, 2019; Lundby et al., 2017). Additionally, an expanded RBCV would lead to an expanded BV, concomitantly contributing to the enhancement of VO_{2max} via the aforementioned Frank-Starling mechanism (Lundby et al., 2017).

The underlying mechanisms of RBCV-expansion in response to endurance training are not fully understood (Lundby & Montero, 2019; Lundby et al., 2017; Montero et al., 2017; Montero & Lundby, 2018). Yet, it is believed that the expanded PV and the resultant decrease in arterial O_2 content following endurance training may facilitate an increase of circulating erythropoietin (EPO), which via a negative feedback loop expands RBCV in an attempt to preserve a normal hematocrit (HCT) and O_2-carrying capacity (Lundby & Montero, 2019; Montero & Lundby, 2018). Other hormones enhanced during and after training which may also contribute to expand RBCV, are catecholamines, cortisol, and growth hormone (Lundby & Montero, 2019). Relative to untrained individuals, elite endurance athletes typically possess up to 40% greater RBCV (Heinicke et al., 2001; Lundby & Robach, 2015) and 35-40% greater total Hb_{mass} (Lundby & Robach, 2015; Schmidt & Prommer, 2008).

1.2.3 Skeletal muscle characteristics

Although O_2 delivery is considered the main limiting factor of VO_{2max} (Montero et al., 2015; Mortensen et al., 2005), it is still debated to what extent the peripheral O_2 extraction capacity contributes to the limitation (Saltin & Calbet, 2006; Skattebo, Calbet, et al., 2020; Wagner, 2006). Skeletal muscle adaptions potentially contributing to an increased O_2 extraction mainly include increases in capillary density and mitochondrial content (Lundby et al., 2017). During maximal work in well-trained individuals, the O_2 content in the arterial and venous blood is

observed to be ~210 and ~30 mL·L^{-1} blood, respectively, indicating that the O_2 extraction reserve is minimal (Mortensen et al., 2005). As there appear to be little "room" for increasing the arterial-venous O_2 difference (a-vO_{2diff}; Lundby et al., 2017), many argue that the O_2 extraction capacity does not limit VO_{2max} (Boushel et al., 2011; Boushel & Saltin, 2013; Lundby et al., 2017; Saltin & Calbet, 2006). Yet, some calculations indicate that central and peripheral factors account for 70–75% and 25-30% of the limitation of VO_{2max}, respectively (di Prampero, 2003; di Prampero & Ferretti, 1990). Furthermore, resent findings suggests that the O_2 extraction capacity increase following endurance training in untrained individuals, and that the muscles become gradually more limited by O_2 delivery with increasing VO_{2max}, interestingly indicating that the factors limiting VO_{2max} may change with training status (Skattebo, Calbet, et al., 2020).

1.3 Fractional utilization of maximal oxygen consumption

$\%VO_{2max}$ is defined as the percentage of VO_{2max} that can be sustained during exercise of a given duration (Joyner & Coyle, 2008). As VO_{2max} sets the upper limit for endurance performance (Basset & Howley, 2000) and exercise intensities corresponding to VO_{2max} only can be sustained for ~6 minutes (Billat & Koralsztein, 1996; Mortensen et al., 2005), $\%VO_{2max}$ is an important determinant of endurance performance exceeding this duration (Davies & Thompson, 1986). $\%VO_{2max}$ is often expressed as percentage of VO_{2max} at lactate threshold i.e., at the highest intensity where the blood lactate concentration ([La$^-$]) remains stable and the homeostasis in the muscle cells is not disturbed (Basset & Howley, 2000; Joyner & Coyle, 2008). Within cycling, lactate threshold is often defined as a [La$^-$] of 4 mmol·L^{-1}, and the corresponding $\%VO_{2max}$ is typically 75-80% in well-trained cyclists (Støren et al., 2013), and 80-85% in elite cyclists (Rønnestad et al., 2017; Rønnestad, Hansen, et al., 2020).

Despite these observed interindividual differences, most studies cannot find an increased $\%VO_{2max}$ after weeks of endurance training in already trained individuals (Rønnestad, Ellefsen, et al., 2014; Rønnestad et al., 2017; Sunde et al., 2010). Yet, investigating this over a four-year period, Rusko (1987) observed that young cross-country skiers increased their $\%VO_{2max}$ from 73 to 78%. This suggests that the enhanced $\%VO_{2max}$ in elite athletes probably is a result of systematic training over several years (Tønnessen & Rønnestad, 2018).

The training induced enhancement of $\%VO_{2max}$ is most likely related to peripheral adaptions in the skeletal muscle (Coyle, 1995; Holloszy & Coyle, 1984; Ivy et al., 1980; Joyner & Coyle, 2008). Increased quantity of mitochondria and aerobic enzymes are observed to be especially important, as this increases the muscles capacity for oxidative ATP generation, consequently making it possible to exercise at a higher percentage of VO_{2max} before the muscle cell homeostasis gets so disturbed that fatigue occurs (Coyle, 1995; Holloszy & Coyle, 1984; Ivy et al., 1980; Joyner & Coyle, 2008). Another important adaption is increased capillary density, as it reduces the diffusion distances between the blood and working muscles (Coyle, 1995), and maintains mean transit time when Q_{max} is increased (Saltin, 1988). Also, a greater proportion of involved muscle mass allows a greater number of mitochondria to share the given VO_2, resulting in a reduced work rate per muscle fiber when exercising at a given workload (Coyle, 1995).

1.4 Exercise efficiency

Whereas VO_{2max} and $\%VO_{2max}$ determines the O_2 consumption and hence the energy turnover that can be sustained during endurance exercise of a given duration, the exercise efficiency determines the power that can be generated at the given O_2 consumption (Joyner & Coyle, 2008). Exercise efficiency is hence considered an important determinant of endurance performance (Joyner & Coyle, 2008), especially when VO_{2max} is relatively similar between individuals (Horowitz et al., 1994). Within cycling, exercise efficiency is often measured as gross efficiency (GE), defined as the percentage of power produced relative to the energy expended (Coyle, 1995). GE is typically 16-18% in untrained individuals (Hopker et al., 2013; Hopker et al., 2007), and 19-23% in well-trained or elite cyclists (Hopker et al., 2007; Horowitz et al., 1994).

It appears that the interindividual variability in exercise efficiency is related to the proportion of type I muscle fibers (Coyle et al., 1992; Horowitz et al., 1994; Mogensen et al., 2006). That is probably because type I fibers are observed to be more energy-efficient than type II fibers (Bottinelli & Reggiani, 2000). Notably, transitions from type II to type I fibers do not appear to occur in humans (Tønnessen & Rønnestad, 2018). Type IIA fibers is suggested to be more energy-efficient than type IIX fibers (Westerblad et al., 2010), and increases in type IIA at the expense of type IIX have been observed in both well-trained (Vikmoen et al., 2016) and elite cyclists (Aagaard et al., 2011) following concurrent strength and endurance training.

Noteworthy, an improved endurance performance was observed in both of the latter studies, but only Vikmoen et al. (2016) observed an improved exercise efficiency. Furthermore, exercise efficiency may be influenced by muscle metabolism, technique, body composition, muscle stiffness, and storage and return of elastic energy (Saunders et al., 2004).

1.5 Training intensity & endurance performance

As put forward by Lundby and Montero (2019), "one deep-rooted axiom in biology asserts that the rate limiting step(s) of a physiological function adapts when stressed." Cellular stress is observed to occur in proportion to training intensity (Egan & Zierath, 2013), and it appears that higher training intensities elicit a greater metabolic signal than moderate intensities (MacInnis & Gibala, 2017). The heart, capillaries, and mitochondria are morphological factors with a central role in the improvement of endurance performance, and the training induced stimuli responsible for their improved function and/or increased quantity appear to be enhanced when training intensity is increased (Tønnessen & Rønnestad, 2018).

These stimuli mainly comprise different cellular signals, and for remodulation of the heart, important stimuli are increased calcium concentration ($[Ca^{2+}]$), increased concentration of the anabolic hormone insulin-like growth factor 1 (IGF-1), and increased activation of proliferator-activated receptor-gamma coactivator 1-alpha (PGC-1α; Bernardo et al., 2010; DeBosch et al., 2006; Ellison et al., 2012; Lehman et al., 2000; McMullen et al., 2003; Patten et al., 2012). For increasing the capillary density, important stimuli are increased pressure against the capillary walls (due to increased blood flow; Hudlická et al., 2000), increased $[Ca^{2+}]$, increased $[La^{-}]$, and reduced O_2 pressure in the capillaries (Fukumura et al., 2001; Hoier & Hellsten, 2014; Hunt et al., 2007; Wahl et al., 2014). Also, PGC-1α appears to be an important regulator of the expression of vascular endothelial growth factor (VEGF), a protein of great importance in the regulation of angiogenesis (Arany et al., 2008; Chinsomboon et al., 2009; Lu et al., 2012; Pogozelski et al., 2009; Tadaishi et al., 2011). For increasing the mitochondrial content, important stimuli are increased $[Ca^{2+}]$, increased concentration of adenosine diphosphate and adenosine monophosphate ([ADP] and [AMP], respectively), and reduced glucose concentration ([glucose]), collectively facilitating an increased expression of PGC-1α, the master regulator of mitochondrial biogenesis (Egan & Zierath, 2013; Rose et al., 2006; Wu et al., 2002).

In summary, the heart, capillaries and mitochondria are affected by many of the same training-induced stimuli, and PGC-1α appear to be of particular importance for the enhancement of endurance performance (Tønnessen & Rønnestad, 2018). Importantly, the strength of these stimuli appears to be training intensity dependent (Tønnessen & Rønnestad, 2018; figure 1). In line with this, several authors have argued that training at or near VO_{2max} maximally stress the O_2 delivery and utilization systems, and thus provide the most effective stimulus for enhancing VO_{2max} (Buchheit & Laursen, 2013; Midgley & Mc Naughton, 2006; Midgley et al., 2006).

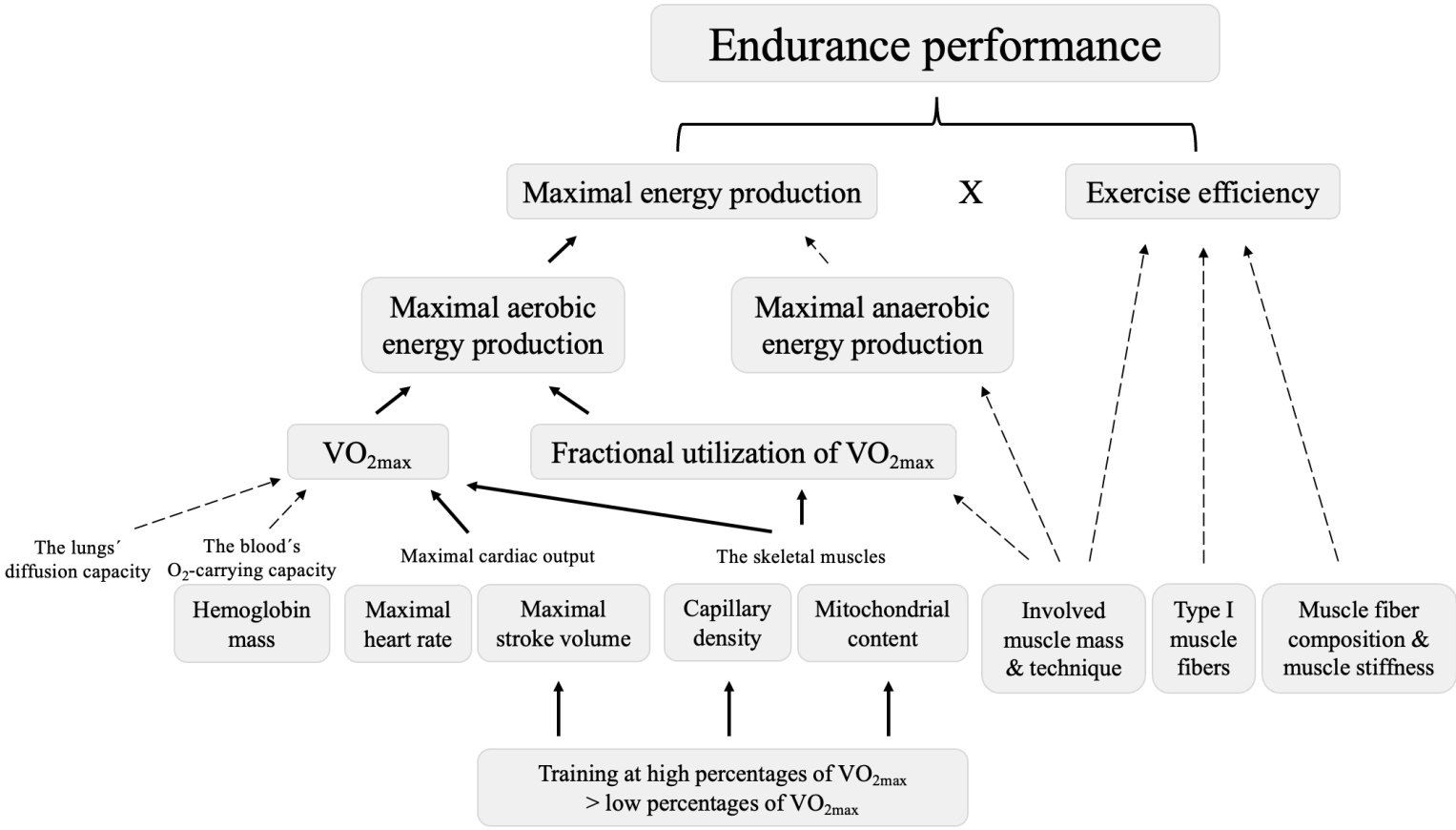

Figure 1: *Overview of the determining factors of endurance performance (bold and dashed arrows) and how they to a greater extent are affected by training at high percentages of VO_{2max} compared to lower percentages of VO_{2max} (bold arrows). The figure is modified from Joyner & Coyle (2008), Basset & Howley (2000), and Tønnessen & Rønnestad (2018). O_2, oxygen; VO_2, oxygen consumption; VO_{2max}, maximal oxygen consumption.*

In their frequently referenced review of fifty-nine training studies, Wenger and Bell (1986) observed a positive relationship between training intensity in the range of 50-100% of VO_{2max} and the degree of enhancement in VO_{2max}. Their conclusion that "the greatest improvements in aerobic power (VO_{2max}) occur when the greatest challenge to aerobic power occurs i.e., when intensity is from 90 to 100% of VO_{2max}" (Wenger & Bell, 1986), is supported by several

studies (Buchheit & Laursen, 2013; Midgley & Mc Naughton, 2006; Midgley et al., 2006; Turnes et al., 2016). Consequently, the percentage of VO_{2max} attained and the time it is sustained ≥ 90% of VO_{2max} have been suggested as a good criterion to judge the effectiveness of the training stimulus (Thevenet et al., 2007).

As continuous work at intensities ≥ 90% of VO_{2max} cannot be sustained for longer periods and thus limits the total time at such intensities during training (Rønnestad & Hansen, 2016), interval-training may be favorable as it extends the time to exhaustion at a given training intensity compared to continuous work (Daniels & Scardina, 1984). To optimize interval-training in respect of time spent ≥ 90% of VO_{2max}, several studies have measured VO_2 acutely and compared different interval designs (Almquist et al., 2020; Bossi et al., 2020; Rønnestad et al., 2022; Rønnestad & Hansen, 2016; Rønnestad, Rømer, et al., 2020).

1.6 Limitations of the current literature

A great limitation to most of the studies supporting the superior effect of training near VO_{2max} (Buchheit & Laursen, 2013; Midgley & Mc Naughton, 2006; Midgley et al., 2006; Thevenet et al., 2007; Wenger & Bell, 1986), is the use of percentage of VO_{2max} as a measure of training intensity when VO_2 during training sessions was not measured (Midgley et al., 2006; Turnes et al., 2016). Instead of VO_2 measures, heart rate measures (Wenger & Bell, 1986) or the minimum velocity or power output known to elicit VO_{2max} (Buchheit & Laursen, 2013) have been used. Consequently, these studies have not measured the VO_2 their participants actually elicited during training (Buchheit & Laursen, 2013; Midgley & Mc Naughton, 2006; Midgley et al., 2006; Wenger & Bell, 1986), and hence nor investigated the importance of training intensity measured as percentage of VO_{2max}. Moreover, most studies have almost exclusively focused on changes in VO_{2max}, and not investigated potential changes in endurance performance and other physiological determinants of endurance performance (Buchheit & Laursen, 2013; Midgley & Mc Naughton, 2006; Midgley et al., 2006; Wenger & Bell, 1986).

To our knowledge, Turnes et al. (2016) is the only study that has measured VO_2 during interval-sessions and investigated the importance of time spent at VO_{2max} on the enhancement of physiological determinants of endurance performance. Their study included twelve high-intensity interval-sessions (HIT-sessions) performed over four weeks, with two groups

performing the HIT-sessions at different relative power outputs. Turnes et al. (2016) observed that the HIT-session eliciting the longest time at VO_{2max} also elicited the greatest improvements in VO_{2max} and power output at lactate threshold in the recreationally trained cyclists. However, a correlation between time spent at VO_{2max} and VO_{2max} improvements was not observed (Turnes et al., 2016). Noteworthy, Turnes et al. (2016) only measured VO_2 during two out of twelve HIT-sessions. Therefore, no study has ever measured VO_2 during every interval-session during an entire training intervention and investigated the effects of a wider range of intensities measured as percentage of VO_{2max} on changes in endurance performance and physiological determinants of endurance performance in endurance athletes. Doing so would be of great importance, as it would cover a great gap in the existing literature.

1.7 Objectives & hypotheses

Main objective: Investigate the importance of average percentage of VO_{2max} during interval-sessions on changes in endurance performance and physiological determinants of endurance performance in cyclists.

Main hypothesis: Improvements in endurance performance and physiological determinants of endurance performance are positively related to average percentage of VO_{2max} during twenty-one interval-sessions performed over nine-weeks in cyclists.

Secondary objective: Investigate whether the half of the cyclists eliciting the highest average percentage of VO_{2max} during interval-sessions elicits greater training adaptions than the half eliciting the lowest percentage of VO_{2max}.

Secondary hypothesis: Greater training adaptions are elicited in the half of the cyclists eliciting the highest average percentage of VO_{2max} during twenty-one interval-sessions performed over nine weeks compared to the half eliciting the lowest percentage of VO_{2max}.

2 Introduction

As endurance performance is primary determined by the maximal oxygen consumption (VO_{2max}), the fractional utilization of VO_{2max} (%VO_{2max}), and exercise efficiency (Joyner & Coyle, 2008), the sport science community has taken a great interest in identifying training protocols that best target and improve these physiological factors (Almquist et al., 2022; Bossi et al., 2020; Buchheit & Laursen, 2013; Laursen & Jenkins, 2002; Laursen et al., 2002; Rønnestad & Hansen, 2016). The heart, capillaries, and mitochondria are morphological factors with a central role in the improvement of endurance performance, and it appears that the strength of the training induced stimuli responsible for their improved function and/or increased quantity are enhanced when training intensity is increased (Tønnessen & Rønnestad, 2018). In line with this, several authors have argued that training at or near VO_{2max} maximally stress the oxygen (O_2) delivery and utilization systems, consequently providing the most effective stimulus for enhancing VO_{2max} (Buchheit & Laursen, 2013; Midgley & Mc Naughton, 2006; Midgley et al., 2006). Therefore, it may come as no surprise that high-intensity training (HIT) is considered one of the most effective forms of training to improve endurance performance in endurance trained athletes (Buchheit & Laursen, 2013; Laursen & Jenkins, 2002).

In their review of fifty-nine training studies, Wenger and Bell (1986) observed a positive relationship between training intensity in the range of 50-100% of VO_{2max} and the degree of enhancement in VO_{2max}. They hence concluded that "the greatest improvements in aerobic power (VO_{2max}) occur when the greatest challenge to aerobic power occurs i.e., when intensity is from 90 to 100% of VO_{2max}" (Wenger & Bell, 1986). The superior effect of training at or near VO_{2max} has been supported by several studies (Buchheit & Laursen, 2013; Midgley & Mc Naughton, 2006; Midgley et al., 2006; Turnes et al., 2016), and the percentage of VO_{2max} attained and the time it is sustained ≥ 90% of VO_{2max} have been suggested as a good criterion to judge the effectiveness of the training stimulus (Thevenet et al., 2007). Particularly in well-trained endurance athletes, training at or near VO_{2max} has been proposed as effective or even necessary to enhance VO_{2max} (Midgley et al., 2006). Yet, adequate support for this premise is surprisingly still unavailable (Midgley et al., 2006; Turnes et al., 2016).

The main limitation of most of the aforementioned studies, is the use of percentage of VO_{2max} as a measure of training intensity when oxygen consumption (VO_2) during training sessions have not been measured (Midgley et al., 2006; Turnes et al., 2016). Instead of VO_2 measures, heart rate (HR) measures (Wenger & Bell, 1986) or the minimum velocity or power output known to elicit VO_{2max} (Buchheit & Laursen, 2013) have been used. As these studies cannot verify the percentage of VO_{2max} their participants actually elicited during training (Buchheit & Laursen, 2013; Midgley & Mc Naughton, 2006; Midgley et al., 2006; Wenger & Bell, 1986), they cannot verify the importance of training at a high percentage of VO_{2max} for training adaptions either. This could make one wonder whether conclusions in a number of studies may have been drawn on insufficient basis. Moreover, most studies have almost exclusively focused on changes in VO_{2max}, and not investigated potential changes in endurance performance and other physiological determinants of endurance performance (Buchheit & Laursen, 2013; Midgley & Mc Naughton, 2006; Midgley et al., 2006; Wenger & Bell, 1986).

To our knowledge, there is only one study that has measured VO_2 during training sessions and investigated the importance of time spent at VO_{2max} on the enhancement of physiological determinants of endurance performance (Turnes et al., 2016). Turnes et al. (2016) verified that two different high-intensity interval-sessions (HIT-sessions) elicited different times at VO_{2max}, and observed that after four weeks and the completion of twelve HIT-sessions, the HIT-session eliciting the longest time at VO_{2max} also elicited the greatest improvements in VO_{2max} and power output at lactate threshold in the recreationally trained cyclists. However, because of the absence of a positive correlation between time spent at VO_{2max} and the improvement in VO_{2max}, the authors argued that their findings did not allow concluding that longer times at VO_{2max} leads to greater VO_{2max} improvements (Turnes et al., 2016). Noteworthy, Turnes et al. (2016) only measured their participants´ VO_2 during the first and last HIT-sessions. Consequently, no study has ever measured VO_2 during every interval-session during an entire training intervention and investigated the effects of a wider range of intensities measured as percentage of VO_{2max} on changes in endurance performance and physiological determinants of endurance performance in endurance athletes. Doing so would be of great importance, as it would cover a great gap in the existing literature.

Therefore, the main purpose of the present study was to investigate the importance of average percentage of VO_{2max} during interval-sessions ($\%VO_{2max@IS}$) on changes in endurance performance and physiological determinants of endurance performance in cyclists. We

performed a nine-weeks training intervention including twenty-one interval-sessions, where we measured the cyclists´ VO_2 during all interval-sessions. We hypothesized that improvements in endurance performance and physiological determinants of endurance performance would be positively related to $\%VO_{2max@IS}$. We also hypothesized that greater training adaptions would be elicited in the half of the cyclists eliciting the highest $\%VO_{2max@IS}$ compared to the half eliciting the lowest $\%VO_{2max@IS}$.

3 Materials and methods

3.1 Participants

Thirty cyclists were initially recruited to participate in the study. Inclusion criteria were an initial VO_{2max} > 60 mL·min^{-1}·kg^{-1} for males and > 55 mL·min^{-1}·kg^{-1} for females. By means of stratified randomization, the cyclists were assigned and counterbalanced to create six homogenous groups based on initial VO_{2max}. Due to sickness (n = 5), injurie (n = 1) and personal reasons unrelated to the training intervention (n = 2), eight cyclists withdrew from the study, leaving a total of twenty-two cyclists (males, n = 19; females, n = 3). Because of measurement error, hematological data are presented from twenty cyclists, whereas all other data are presented from twenty-two cyclists. At the start of the intervention, the cyclists had just finished their competition season and had hence undertaken their usual in-season training (08:49 ± 02:55 hours per week recorded during the four weeks preceding pre-testing; appendix A). The cyclists had a history of 3.7 ± 2.9 years of competitive cycling. Based on initial VO_{2max}, the cyclists were categorized as performance level 3 (n = 5), 4 (n = 9), and 5 (n = 8) according to the classification systems by De Pauw et al. (2013) and Decroix et al. (2016) for male and female cyclists, respectively. Before inclusion, the cyclists were informed of any potential risks and discomfort associated with the study, and they all provided their written informed consent to participate (appendix B). The study was conducted accordingly to the ethical standards established by the Helsinki Declaration of 1975 and was approved by the local ethical committee at Inland Norway University of Applied Sciences and the Data Protection Authority.

3.2 Experimental design

An overview of the study is presented in figure 2. The cyclists completed a nine-weeks training intervention divided into three three-weeks periods including seven interval-sessions each. Physiological tests were performed at two following days (testday 1 and testday 2) before the intervention (pre), after the first period (mid 1), after the second period (mid 2), and after the third period (post). Only the results from pre- and post-tests are presented in this thesis. An overview of the testing procedures at testday 1 and testday 2 are presented in part 3.5 (figure 7).

	Period 1			Period 2			Period 3		
	Week 1	Week 2	Week 3	Week 4	Week 5	Week 6	Week 7	Week 8	Week 9
Interval-sessions	↑↑	↑↑↑	↑↑	↑↑	↑↑↑	↑↑	↑↑	↑↑↑	↑↑
Testday 1 ↑			↑			↑			↑
Testday 2 ↑			↑			↑			↑

Figure 2: *Overview and time course of the present study, including twenty-one interval-sessions and physiological testing (testday 1 and testday 2) at four timepoints.*

In total, each cyclists performed 20.6 ± 0.8 interval-sessions consisting of 5x8-minutes work-intervals separated by 3-minutes recovery periods. In all periods the mean power output during the 8-minutes work-intervals corresponded to the individual cyclists´ mean power output during the 40-minutes time trial ($PO_{@40min}$) at testday 2 preceding the period (100% of functional threshold power (FTP)). How the 8-minutes work-intervals were performed, differed between the three periods due to another part of the study: 1) 30-seconds work-intervals (118% of FTP) separated by 15-seconds recovery periods (60% of FTP) performed continuously in each 8-minutes work-interval (30/15), 2) two different 60-seconds work-intervals (110 and 90% of FTP) altering continuously in each 8-minutes work-interval (60/60), and 3) one continuous 8-minutes work-interval (100% of FTP; flat; figure 3). To control for the order the cyclists performed the different interval-designs, the six groups performed the three different interval-designs in six different orders (figure 4).

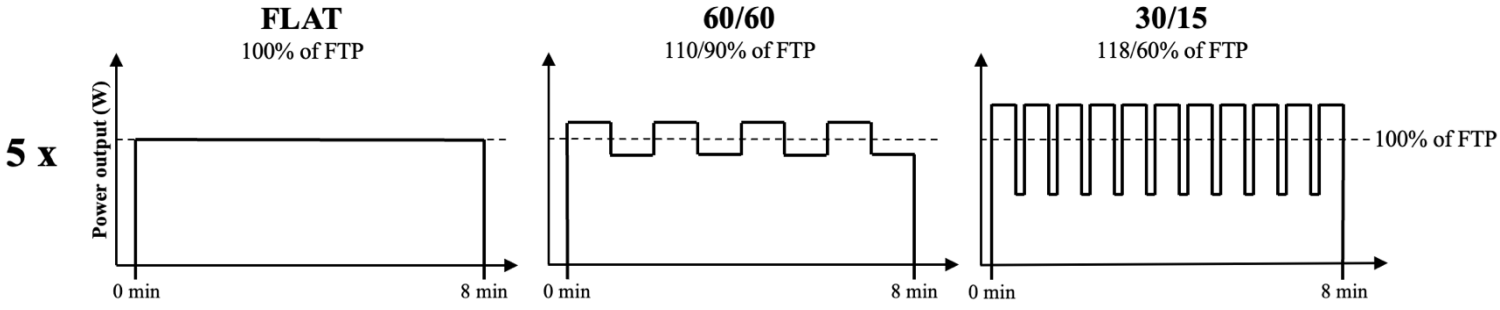

Figure 3: *Overview of the three different interval-designs used in the present study. All interval-sessions consisted of 5x8-minutes work-intervals separated by 3-minutes recovery periods, but the design of the 8-minutes work-intervals differed between periods. Flat, one continuous 8-minutes work-interval; 60/60, two different 60-seconds work-intervals altering continuously in each 8-minutes work-interval; 30/15, 30-seconds work-intervals separated by 15-seconds recovery periods performed continuously in each 8-minutes work-interval; FTP; functional threshold power defined as the mean power output during the 40-minutes time trial at testday 2 preceding the given period.*

	Period 1	Period 2	Period 3
Group 1	30/15	60/60	FLAT
Group 2	60/60	FLAT	30/15
Group 3	FLAT	30/15	60/60
Group 4	FLAT	60/60	30/15
Group 5	30/15	FLAT	60/60
Group 6	60/60	30/15	FLAT

Figure 4: *Overview of the orders the six groups performed the three different interval-designs in. 30/15, 30-seconds work-intervals separated by 15-seconds recovery periods performed continuously in each 8-minutes work-interval; 60/60, two different 60-seconds work-intervals altering continuously in each 8-minutes work-interval; flat, one continuous 8-minutes work-interval.*

To be able to investigate how percentage of VO_{2max} during the interval-sessions effected endurance performance and physiological determinants of endurance performance, VO_2 was measured during all 8-minutes work-intervals at all interval-sessions. After the intervention, the cyclists were allocated to two new groups based on their $\%VO_{2max@IS}$. The half of the cyclists eliciting the highest $\%VO_{2max@IS}$ were allocated to HIGH (n = 11), whereas the half eliciting the lowest $\%VO_{2max@IS}$ were allocated to LOW (n = 11; table 1).

Table 1: *Average percentage of VO_{2max} elicited during interval-sessions and baseline characteristics for the group with the highest (HIGH) and lowest (LOW) average percentage of VO_{2max} elicited during interval-sessions.*

	HIGH	LOW	Group
$\%VO_{2max@IS}$ (%)	86.2 ± 3.8 #	79.9 ± 4	p = 0.001
Age (years)	21.4 ± 6.7	22.6 ± 5.5	p = 0.631
Body mass (kg)	69.2 ± 9.8	74.7 ± 8.2	p = 0.174
Body height (cm)	178.0 ± 8.9	182.0 ± 6.3	p = 0.282
VO_{2max} ($ml \cdot min^{-1} \cdot kg^{-1}$)	65.1 ± 5.3	69.1 ± 7.1	p = 0.149
W_{max} ($W \cdot kg^{-1}$)	5.7 ± 0.6	5.8 ± 0.7	p = 0.745
$PO_{@4mmol}$ ($W \cdot kg^{-1}$)	4.0 ± 0.6	4.0 ± 0.5	p = 0.901
$\%VO_{2max@4mmol}$ (%)	84.1 ± 5.2	80.8 ± 5.2	p = 0.150

$\%VO_{2max@IS}$, average percentage of VO_{2max} elicited during interval-sessions; VO_{2max}, maximal oxygen consumption; W_{max}, maximal aerobic power output; $PO_{@4mmol}$, power output at 4 $mmol \cdot L^{-1}$ blood lactate concentration; $\%VO_{2max@4mmol}$, fractional utilization of VO_{2max} at 4 $mmol \cdot L^{-1}$ blood lactate concentration. Values are mean ± SD. # Significantly different from LOW ($p < 0.050$).

3.3 Self-reported training

In addition to the tests and interval-sessions, the cyclists performed their normal strength and low-intensity endurance training. All training was reported in a personal training diary (appendix C; table 2). Endurance training was reported according to Andrew Coggan´s seven-zone intensity scale, based on percentage of FTP or percentage of HR associated with FTP (HR@FTP; Hunter & Coggan, 2010). FTP and HR@FTP were updated after the 40-minutes time trial at testday 2 at mid 1 and mid 2. Exercise performed as cycling was reported in respect of power output, whereas exercise performed as running or other types of movements were reported in respect of HR. Only the first five zones on the intensity scale were used: zone 1 (< 55% of FTP, < 68% of HR@FTP), zone 2 (56-75% of FTP, 69-83% of HR@FTP), zone 3 (76-90% of FTP, 84-94% of HR@FTP), zone 4 (91-105% of FTP, 95-105% of HR@FTP), and zone 5 (> 106% of FTP, > 106% of HR@FTP). Of the cyclists´ total endurance training, 85.7 ± 8.4% was performed as cycling, 7.3 ± 3.4% as running, and 5.5 ± 3.1% as other types of movement. Eventual duration of heavy (> 80% of 1 repetition maximum (RM)) and general (< 80% of 1 RM) strength training were also reported in the training diary. To evaluate how the intervention affected the cyclists´ perceived well-being in the legs, this was reported after all training sessions using a 9-point scale (Rønnestad, Hansen, et al., 2014): 1 (very, very good), 2 (very good), 3 (good), 4 (somewhat good), 5 (normal), 6 (somewhat bad), 7 (bad), 8 (very bad), and 9 (very, very bad).

Table 2: *Average weekly distribution of training in period 1, 2 and 3 in HIGH and LOW, respectively.*

	HIGH			LOW		
	Period 1	**Period 2**	**Period 3**	**Period 1**	**Period 2**	**Period 3**
Zone 1 (hh:mm)	03:46 ± 01:34 €	02:56 ± 01:47 $	02:54 ± 01:37 $	02:02 ± 00:53	01:43 ± 01:07	01:40 ± 00:59
Zone 2 (hh:mm)	03:04 ± 01:18	03:14 ± 01:25	03:37 ± 02:03	02:58 ± 01:22	02:13 ± 01:26	02:13 ± 01:00
Zone 3 (hh:mm)	01:23 ± 00:31	01:03 ± 00:30	01:02 ± 00:29	01:13 ± 00:34	01:07 ± 00:32	01:08 ± 00:28
Zone 4 (hh:mm)	01:18 ± 00:32	01:17 ± 00:29	01:25 ± 00:21	01:26 ± 00:39	01:30 ± 00:21	01:19 ± 00:15
Zone 5 (hh:mm)	00:21 ± 00:19	00:31 ± 00:26	00:24 ± 00:22	00:25 ± 00:30	00:31 ± 00:25	00:46 ± 00:29
Heavy strength (hh:mm)	00:06 ± 00:18	00:01 ± 00:03	00:00 ± 00:01	00:20 ± 00:33	00:14 ± 00:27	00:18 ± 00:37
General strength (hh:mm)	00:06 ± 00:11	00:22 ± 00:30	00:19 ± 00:24	00:11 ± 00:15	00:21 ± 00:32	00:22 ± 00:25
Total training (hh:mm)	10:04 ± 02:30	09:23 ± 02:37	09:41 ± 02:57	08:10 ± 02:25	07:20 ± 02:54	07:29 ± 02:24
Feeling legs (1-9)	5.1 ± 0.3	5.1 ± 0.4	5.2 ± 0.3	5.2 ± 0.4	5.2 ± 0.4	5.1 ± 0.3

Feeling legs, perceived well-being in the legs. € Tendency to different from LOW within period (p < 0.100 and > 0.050). $ Tendency to different from period 1 within group (p < 0.100 and > 0.050).

3.4 Interval-sessions

The cyclists performed the interval-sessions in a training room at either Kongsvinger or Lillehammer, using their own bikes connected to an individual, stationary trainer device (Tacx Neo 2T Smart T2875 or Tacx Neo 2 Smart T2850, Wassenaar, the Netherlands). All interval-sessions were supervised by a test leader, who controlled the power output at the interval-sessions using an app (Tacx Training-app, version 4.26.2, Garmin Ltd., the Netherlands) connected to the trainer device. Prior to all interval-sessions, the two first steps of the Lamberts and Lambert Submaximal Cycle Test (LSCT) was used as a warm-up protocol (Lamberts et al., 2011). The LSCT consisted of 6 minutes of cycling at a power output eliciting 60% of maximal HR (HR_{max}) measured at testday 1 at pre-test (step 1), followed by 6 more minutes at a power output eliciting 80% of HR_{max} (step 2; Garmin Edge 530, Garmin Ltd., Olathe, Kansas, USA; figure 5). Data from the LSCT are not presented in this thesis. 3 minutes after the completion of the LSCT, the cyclists were asked to report their perceived well-being in the legs (Rønnestad, Hansen, et al., 2014). The interval-sessions started 5 to 10 minutes after the LSCT.

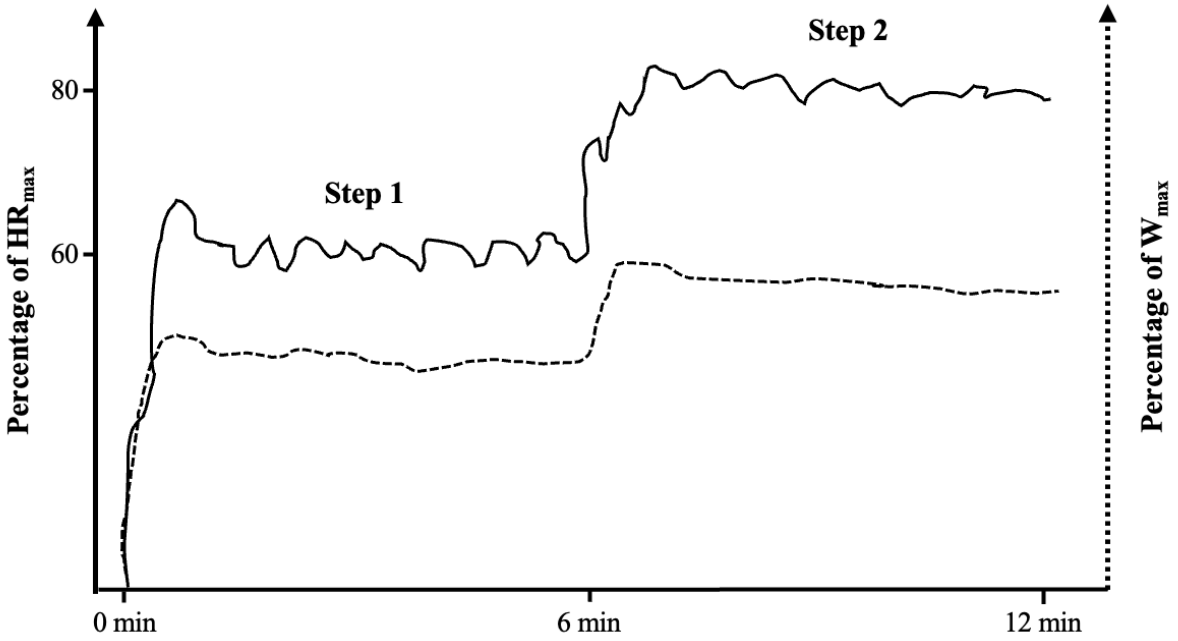

Figure 5: *Overview of the two first steps of the Lamberts and Lambert Submaximal Cycle Test (LSCT) used as a warm-up protocol in the present study. Step 1 and 2 consisted of 6 minutes of cycling at the power output eliciting 60 and 80% of HR_{max}, respectively. The figure is modified from Lamberts, Swart, Noaks & Lambert (2009). HR_{max}, maximal heart rate during the maximal oxygen consumption test; W_{max}, maximal aerobic power output.*

If the cyclists could not complete the 8-minutes work-intervals at 100% of FTP, the power output was lowered to the highest feasible percentage of FTP. If the power output corresponding to 100% of FTP was perceived as far too easy, discretionary assessments to increase the power output were made. VO_2 was measured every 10^{th} second using a metabolic system with mixing chamber (Oxycon Pro, Erich Jaeger, Hoechberg, Germany; figure 6). A standardized calibration of the metabolic system was performed before each interval-session, including calibration of the gas analyzers with certified calibration gasses of known concentrations, and calibration of temperature and ambient conditions. The flow turbine (Triple V, Erich Jaeger) was calibrated with a 3 L, 5530 series, calibration syringe (Hans Rudolph, Kansas City, USA). Due to measurement error, 3.8% of the 10-seconds VO_2-measurements were estimated. The missing measurements were estimated by copying the corresponding VO_2-measurements at the previous work-interval. However, if the power output differed between the work-intervals or the measurement error occurred at the first work-interval of the interval-session, conservative, discretionary assessments of which measurements to copy were made.

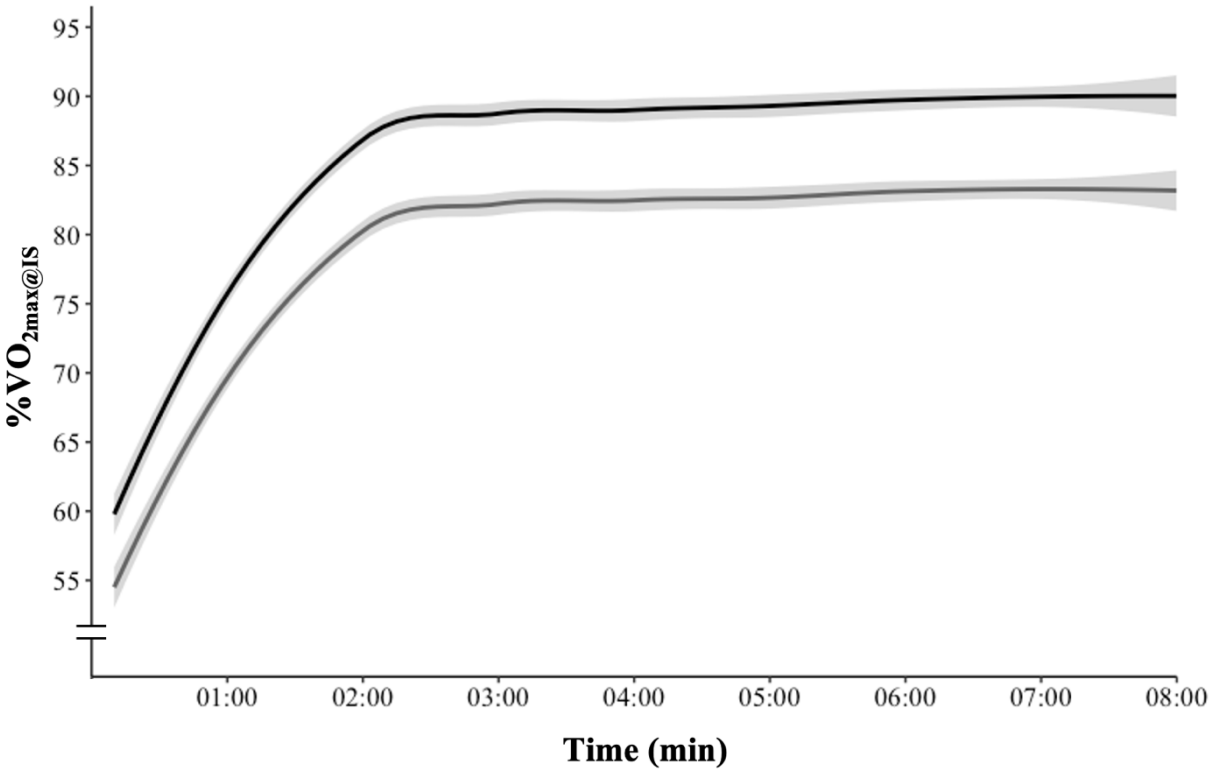

Figure 6: *Average percentage of maximal oxygen consumption elicited during the 8-minutes work-intervals at all interval-sessions for HIGH (black line) and LOW (gray line), respectively. The light gray areas represent 95% confidence intervals. $\%VO_{2max@IS}$, average percentage of maximal oxygen consumption elicited during interval-sessions.*

HIGH and LOW performed the 8-minutes work-intervals at an average of 86.2 ± 3.8% and 79.9 ± 4.0% of VO_{2max}, respectively. All cyclists performed the work-intervals at an average percentage of VO_{2max} in the range of 74.2-90.8%. Cadence was freely chosen, and power output ($\%FTP_{@IS}$) and HR ($\%HR_{max@IS}$) were measured every 10th second during the work-intervals. Rate of perceived exhaustion (RPE) was recorded using Borg's 6-20 scale (Borg, 1982) immediately after each work-interval. At the second, fifth and seventh interval-session in all periods, blood samples from the fingertip were obtained immediately after each work-interval for determination of blood lactate concentration ($[La^-]$; Biosen C-line Lactate Analyzer, EKF Diagnostic GmbH, Barleben, Germany). 10 minutes after each interval-session, the cyclists reported their session rate of perceived exhaustion using a 10-points scale (sRPE; Foster et al., 2001). Data from the interval-sessions are presented in table 3.

Table 3: *Interval-session data, presented as averages of all collected measurements during and after interval-sessions in period 1, 2 and 3 in HIGH and LOW, respectively.*

	HIGH			**LOW**		
	Period 1	**Period 2**	**Period 3**	**Period 1**	**Period 2**	**Period 3**
Time≥90%VO_{2max} (mm:ss)	15:32 ± 10:03 #	15:05 ± 10:47 #	14:52 ± 09:43 #	02:14 ± 03:38	03:06 ± 04:18	05:52 ± 07:53 *
$\%VO_{2max@IS}$ (%)	86.3 ± 3.6 #	86.2 ± 4.2 #	85.9 ± 3.8 #	79.0 ± 3.6	79.7 ± 3.6	81.0 ± 4.5 *
$\%HR_{max@IS}$ (%)	87.6 ± 2.0	88.4 ± 2.4	88.9 ± 2.4 *	86.8 ± 2.3	86.9 ± 2.3	87.7 ± 1.7 $ £
$\%FTP_{@IS}$ (%)	97.8 ± 3.2 €	99.6 ± 1.8 *	100.7 ± 2.7 * §	99.8 ± 2.6	100.2 ± 0.9	99.4 ± 2.4
$[La^-]$ ($mmol \cdot L^{-1}$)	5.15 ± 1.94	5.88 ± 1.39	6.19 ± 2.53	5.31 ± 1.62	5.83 ± 1.90	6.45 ± 2.17 *
RPE (6-20)	16.2 ± 1.0	16.4 ± 1.2	16.6 ± 1.1 *	15.8 ± 1.0	15.9 ± 1.1	16.0 ± 1.2
Feeling legs (1-9)	5.2 ± 0.9	5.5 ± 0.9	5.4 ± 0.7	5.4 ± 0.9	5.2 ± 0.7	5.4 ± 0.7
sRPE (0-10)	6.9 ± 1.7	7.0 ± 1.6	7.4 ± 1.4	5.9 ± 1.4	6.1 ± 1.3	6.3 ± 1.4
Number of sessions	6.8 ± 0.4	6.9 ± 0.3	6.9 ± 0.5	7.0 ± 0.0	7.0 ± 0.0	6.8 ± 0.6 * §

*Time≥90%VO_{2max}, time spent ≥ 90% of maximal oxygen consumption during all interval-sessions; $\%VO_{2max@IS}$, average percentage of VO_{2max} during all interval-sessions; $\%HR_{max@IS}$, average percentage of maximal hart rate during all interval-sessions; $\%FTP_{@IS}$, average percentage of functional threshold power during all interval-sessions;$[La^-]$, average blood lactate concentration collected after each work-interval at interval-session 2, 5 and 7; RPE, average rate of perceived exhaustion reported after each 8-minutes work-interval at all interval-sessions; feeling legs, perceived well-being in the legs reported after the warm-up protocol prior to all interval-sessions; sRPE, session rate of perceived exhaustion reported 10 minutes after all interval-sessions. Values are mean ± SD. # Significantly different from LOW within period ($p < 0.050$). € Tendency to different from LOW within period ($p < 0.100$ and > 0.050). * Significantly different from period 1 within group ($p < 0.050$). $ Tendency to different from period 1 within group ($p < 0.100$ and > 0.050). § Significantly different from period 2 within group ($p < 0.050$). £ Tendency to different from period 2 within group ($p < 0.100$ and > 0.050).*

3.5 Testing procedures

To ensure comparable preparations, the cyclists were asked to refrain from training the day before testday 1 at all timepoints. At pre-tests, the cyclists reported their three last meals, fluid intake and eventual caffeine intake prior to the tests, and two to three days before mid 1-, mid 2- and post-tests, the cyclists received a message with instructions to repeat this. The individual amount of water, sports drink and solid foods consumed during pre-tests were also noted and repeated at the following tests. The individual cyclists had the same test leader at the respective tests, and strong verbal encouragement was given during both the strength and endurance tests to ensure maximal effort. All endurance tests were performed with a fan ensuring circulating air around the cyclist. To avoid influence from the circadian rhythm, the respective tests were performed at the same time of the day (± 2 hours) for the individual cyclists. At testday 1, a standardized warm-up protocol on a cycle ergometer was performed before the strength and endurance tests. The warm-up-protocol consisted of 2 minutes at an intensity corresponding to 11 RPE, 2 minutes at 13, 1 minute at 15 and 2 minutes at 12. At testday 2, the same warm-up protocol as used before the interval-sessions was performed. Testday 1 was conducted at the physiological test laboratory at Inland Norway University of Applied Sciences Lillehammer and testday 2 was conducted in a training room at either Kongsvinger or Lillehammer. The test protocols were developed by the professor leading the project to evaluate potential changes in physiological, hematological, and performance-related measures (figure 7).

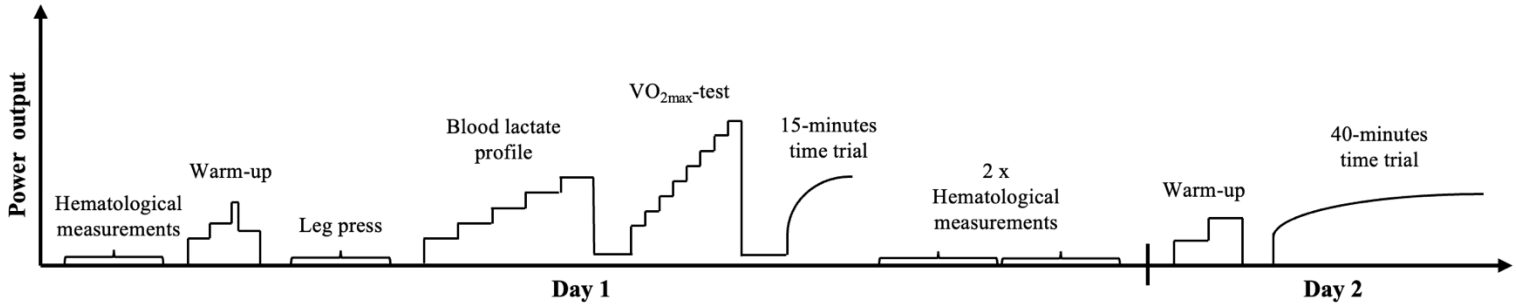

Figure 7: *Overview of the testing procedures at testday 1 and testday 2. Briefly, testday 1 started with the measurement of hematocrit and hemoglobin concentration. Following a standardized warm-up protocol, peak power in leg press was determined. Then, an approximately one-hour long cycling-test was performed, including a blood lactate profile, an incremental test to determine maximal oxygen consumption, and a 15-minutes time trial. Following the cycling test, a carbon monoxide (CO) rebreathing procedure was performed in duplicate. At testday 2, another standardized warm-up protocol was performed prior to a 40-minutes time trial. VO_{2max}-test; test to determine maximal oxygen consumption.*

3.6 Hematological measurements: hematocrit, hemoglobin concentration, hemoglobin mass & intravascular volumes

Before the strength and endurance tests at testday 1, hematocrit (HCT) and hemoglobin concentration ([Hb]) were measured. Starting the procedure, the cyclists drank 300 mL of water before resting in a supine position with legs elevated for 15 minutes. For the measurement of HCT, the microhematocrit method was used (Mondal & Budh, 2021). Briefly, blood from the fingertip was filled in three 75 mm capillary tubes and centrifuged in a microcentrifuge (Heraeus PICO 17 Hematokritrotor, Thermo Electron LED GmbH, Osterode, Germany) at a rate of 13500 rpm for 4 minutes. The samples were then analyzed using a microhematocrit card reader. For the measurement of [Hb], blood from the fingertip was filled in three 40 mm capillary tubes and analyzed using a blood gas analyzer (ABL830 FLEX CO-OX analyzer, Radiometer, Copenhagen, Denmark). The means of the triple HCT and [Hb] measurements were used in following calculations and in the data analyses.

30 minutes after the endurance tests, hemoglobin mass (Hb_{mass}), red blood cell volume (RBCV), plasma volume (PV), and blood volume (BV) was determined using the carbon monoxide (CO) rebreathing technique (Siebenmann et al., 2017). The cyclists rested in a supine position with legs elevated for 5 minutes before blood from the fingertip was filled in three 40 mm capillary tubes and analyzed for percent carboxyhemoglobin (%HbCO; ABL830 FLEX CO-OX analyzer, Radiometer, Copenhagen, Denmark). Then the cyclists breathed through a mouthpiece in a closed system (CO-Applikator, WGT Elektronik CmbH & Co KG, Kolsass, Austria) for 15 seconds before a dose of 1.5 $mL \cdot kg^{-1}$ body mass of 99.997% chemically pure CO (Carbon monoxide 100%, AGA, Oslo, Norway) was administrated through the system. The cyclists rebreathed the CO/O_2 mixture for 6 minutes, and before the mouthpiece was removed, they performed a total exhalation of the lungs for determination of the remaining CO volume in the rebreathing circuit (Dräger Pac 5500, Dräger INC., Houston, USA). After breathing normally for 4 more minutes, three new blood samples were collected and analyzed for %HbCO. Total Hb_{mass} was calculated form the absorbed CO dose and the change in %HbCO pre to post CO rebreathing, using the means of the triple pre and post %HbCO measurements. RBCV, PV, and BV were derived from total Hb_{mass}, [Hb] and HCT. Following 10 minutes of rest in the same supine position with legs elevated, the CO rebreathing procedure was performed one more time. The means of the repeated measurements were used in the data analyses.

3.7 Strength test: peak power in leg press

As a change in the legs power generation could influence endurance performance (Rønnestad et al., 2015), a test for determination of peak power (P_{peak}) in leg press was performed to control for this. The test was performed in a Keiser AIR300 horizontal leg-press dynamometer (Keiser Corp., Fresno, CA, USA), using a 10-repetition incremental protocol (table 4). For male cyclists, the resistance at the 10th repetition was set to 250 kg if the cyclist´s body weight was < 75 kg, and 280 kg if it was > 75 kg. For female cyclists, the resistance at the 10th repetition was set to 200 kg if the cyclist´s body weight was < 75 kg. The resistance at the 10th repetition determined the increase in resistance between each repetition (resistance increase = (resistance at the 10th repetition – 18.14) · 10^{-1}), and the starting resistance was determined by the resistance increase (starting resistance = resistance increase + 18.14). The cyclists sat with knees and hip flexed at ~ 90° and ~ 45°, respectively, with the same individual seating position at all timepoints. Instructions to exert force "as fast as possible" at each lift was given, and the attempts continued until failure. The criteria for approved lift were full knee extension with the back to the back of the seat. P_{peak} was defined as the highest product of velocity and force achieved during the test and was calculated by the manufacturer's software.

Table 4: *Overview of the 10-repetition incremental protocol for determination of peak power in leg press.*

Repetition number	**Warm up**	**Warm up**	**1st**	**2nd**	**3rd**	**4th**	**5th**	**6th**	**7th**	**8th**	**9th**	**10th**	**n + 1**
Males													
Resistance (kg) if < 75 kg	41	41	41	64	87	110	133	157	180	203	226	250	+
Resistance (kg) if > 75 kg	44	44	44	71	97	123	149	175	201	228	254	280	++
Females													
Resistance (kg) if < 75 kg	36	36	36	55	73	91	109	127	146	164	182	200	#
Recovery period (s)	3.0	3.0	3.0	4.2	5.8	8.1	11.4	15.8	22.1	30.8	43.0	60.0	60.0

+ Previous repetition + 23.2; ++ previous repetition + 26.2; # previous repetition + 18.2.

3.8 Endurance tests

3.8.1 Blood lactate profile

All endurance tests at testday 1 were performed on the same electromagnetic braked cycle ergometer (Lode Excalibur Sport, Groningen, The Netherlands). The cycle ergometer was adjusted according to each cyclist's preference for seat height, handlebar position, and horizontal distance between tip of seat and bottom bracket. The tests were performed seated with the same individual seating positions at all timepoints. The blood lactate profile started with 5 minutes of cycling at 125 W (175 W if lactate threshold > 325 W) and continued with 50 W increases every 5th minute. With 30 seconds left at each 5-minutes bout, a blood sample from the fingertip was collected for determination of [La^-] (Biosen C-line Lactate Analyzer, EKF Diagnostic GmbH, Barleben, Germany). When a [La^-] of $\geq$ 2 mmol·L^{-1} was measured, the power output continued to increase with 25 W. The test was ended when a [La^-] of $\geq$ 4 mmol·L^{-1} was reached. VO_2 and respiratory exchange ratio (RER) were measured every 30th second from the 2.5th to 4.5th minute at each bout, using the same computerized metabolic system as at the interval-sessions. The metabolic system was calibrated prior to all lactate profiles, following the same protocol as used prior to all interval-sessions. The individual cyclists started the blood lactate profile at the same power output at all timepoints, but the power output increased independently of the increments at previous tests. When the power output at bouts were equal to what the individual cyclists had previously performed, individual instructions to cycle at the same cadence as at previous tests were given. The blood lactate profile was used to determine the power output, HR, and $\%VO_{2max}$ at 4 mmol·L^{-1} [La^-] ($PO_{@4mmol}$, $HR_{@4mmol}$, and $\%VO_{2max@4mmol}$, respectively). Steady-state VO_2 and RER were used to calculate exercise efficiency measured as gross efficiency at 175 and 225 W ($GE_{@175W}$ and $GE_{@225W}$, respectively), where GE was defined as the ratio between mechanical power output (PO) and metabolic power input (PI): $GE = PI \cdot PO^{-1} \cdot 100$. The O_2 equivalent (Péronnet & Massicotte, 1991), and VO_2 and RER at the given power output (Noordhof et al., 2013) were used to calculate PI: $PI = VO_2\ L \cdot s^{-1} \cdot (4840\ J \cdot L^{-1} \cdot RER + 16{,}890\ J \cdot L^{-1})$.

3.8.2 Maximal oxygen consumption & maximal aerobic power output

Following 5 minutes of active recovery cycling at 70 to 100 W, an incremental test to determine VO_{2max} and maximal aerobic power output defined as the mean power output during the last minute of the incremental test (W_{max}) was initiated. The test started at 200 W (250 W if lactate threshold > 320 W) and increased by 25 W every minute until exhaustion

defined as cadence < 60 rpm. For female cyclists, the test started at 160 W and increased by 20 W every minute. VO_2 was measured every 30th second, and VO_{2max} was defined as the mean of the two highest consecutive 30-seconds measurements. HR_{max} was measured during the test and RPE was recorded immediately after (RPE_{max}). A blood sample from the fingertip was collected 1 minute after the test had ended for determination of [La^-] ($[La^-]_{max}$). Cadence was freely chosen.

3.8.3 15-minutes time trial

The cyclists then got 10 minutes of active recovery at 70 to 100 W, including a gradual increase in power output to a power output corresponding to 15 RPE from the 6th to 7th minute during the recovery period. At the 15-minutes time trial, the cyclists were instructed to aim for the highest possible mean power output during the 15 minutes. The cyclists regulated the power output themselves using an external control unit placed next to the handlebar of the ergometer setup. Cadence was freely chosen, and VO_2 and HR were measured every 30th second for determination of $\%VO_{2max}$ ($\%VO_{2max@15min}$), average HR ($HR_{mean@15min}$; the cyclists´ personal HR monitors), and HR at the end of the test ($HR_{end@15min}$). RPE was recorded immediately after the test had ended ($RPE_{@15min}$), and a blood sample from the fingertip was collected 1 minute after for determination of [La^-] ($[La^-]_{end@15min}$). Endurance performance was measured as the mean power output during the 15 minutes ($PO_{@15min}$).

Based on the main performance indicators from testday 1 (W_{max}, $PO_{@4mmol}$, and $PO_{@15min}$), a performance index was calculated as the average of the given indicators after normalization ($x_i \cdot \max(x)^{-1}$ where x_i is a single observation from one performance indicator). The performance index was calculated to increase the statistical power for performance-related measures for which small differences may be very difficult to detect but still of relevance for elite endurance performance.

3.8.4 40-minutes time trial

At testday 2, the cyclists performed a 40-minutes time trial using their own bikes connected to the same individual, stationary trainer device used at interval-sessions. The cyclists were instructed to aim for the highest possible mean power output during the 40 minutes and regulated the power output themselves using the app connected to the trainer device. Cadence was freely chosen, and HR was measured every 10th second for determination of average HR

($HR_{mean@40min}$) and HR at the end of the test ($HR_{end@40min}$; Garmin Edge 530 Cycle Computer, Garmin Ltd., Olathe, Kansas, USA). At the 5th, 10th, 18th, 26th, and 34th minute of the test as well as 1 minute after, a blood sample from the fingertip was collected for determination of average [La^-] during the test ($[La^-]_{mean@40min}$) and [La^-] at the end of the test ($[La^-]_{end@40min}$), respectively. RPE was recorded immediately after the test ($RPE_{@40min}$), and endurance performance was measured as $PO_{@40min}$.

3.9 Statistics

All descriptive data in text, tables and figures are presented as mean and standard deviation (mean ± SD). VO_{2max}, power outputs, and hematological measures are presented per kg body mass. To detect potential differences between groups at baseline, the data was fitted as the dependent variable in a one-way analysis of variance (ANOVA) with group as the explanatory variable. A mixed model was used to evaluate potential within group differences between periods and within period differences between groups in interval-session measures and total training load during the intervention. In the mixed model the data was fitted as the dependent variable, period as the explanatory variable, and period:group + (1|id) as covariates to compare groups within each period and include all cyclists in all periods. To test for between groups differences in pre to post changes, an analysis of covariance (ANCOVA) was used with the percent change as the dependent variable, group as the explanatory variable, and pre-test values as a covariate to adjust for potential differences at baseline. Pre- and post-intervention changes in each group were compared using two-tailed paired Students t-tests. To investigate the relationship between $\%VO_{2max@IS}$ and pre to post changes, the groups were pooled, and a linear regression model was fitted with percentage change as the dependent variable, $\%VO_{2max@IS}$ as the explanatory variable, and pre-test values as a covariate. To investigate whether the pre to post change in one variable could explain the pre to post change in another variable, the groups were pooled, and a linear regression model was fitted with percentage change in one variable as the dependent variable, percentage change in another variable as the explanatory variable, and pre-test values of the dependent variable as a covariate. To investigate the relationship between $\%VO_{2max@4mmol}$ at baseline and $\%VO_{2max@IS}$ and time spent ≥ 90% of VO_{2max} during interval-sessions ($time{\geq}90\%VO_{2max@IS}$), the groups were pooled, and a linear correlation model was fitted with $\%VO_{2max@IS}$ or $time{\geq}90\%VO_{2max@IS}$ as the dependent variable and $\%VO_{2max@4mmol}$ at baseline as the explanatory variable. $R^2_{adjusted}$ was calculated to interpret the accuracy of the linear models.

The results from all analyses were considered statistically significant if $p < 0.050$ and described as tendencies if $p < 0.100$ and > 0.050. Because of the relatively small sample size and the expectation of relatively modest changes in the already trained cyclists, Cohen´s d effect size (ES) was calculated to interpret the practical significance of the differences between groups. ES was calculated using the mean pre to post change in HIGH minus the mean pre to post change in LOW, divided by the pooled pre-test standard deviation (Morris, 2008). The scale proposed by Rhea (2004) for highly trained subjects was used to interpret the magnitude of the treatment effect: 0.0-0.24 trivial, 0.25-0.49 small, 0.5-1.0 moderate, and > 1.0 large. The data was collected in Microsoft Office Excel (Microsoft, Redmond, USA) and analyzed in R (R Core Team, 2018).

4 Results

4.1 Baseline & body mass

At baseline, there was a difference between HIGH and LOW in $GE_{@225W}$ (20.0 ± 0.6% and 19.4 ± 0.6%, respectively, $p = 0.026$) and a tendency to difference in $GE_{@175W}$ (19.2 ± 0.7% and 18.6 ± 0.9%, respectively, $p = 0.072$). No other hematological, physiological, or performance-related measures differed between the two groups prior to the intervention. Body mass did not change within or between the groups during the intervention (table 5).

4.2 Endurance performance variables

Following the nine-weeks training intervention, there tended to be a positive relationship between percentage change in W_{max} and $\%VO_{2max@IS}$ ($p = 0.098$; $R^2_{adjusted} = 0.048$; figure 8B). W_{max} increased with 8.6 ± 5.6% in HIGH ($p = 0.001$) and 4.2 ± 2.8% in LOW ($p = 0.001$), and the percentage increase was larger in HIGH than in LOW ($p = 0.035$; figure 9B). The ES analysis revealed a small practical effect of HIGH (ES = 0.45). There was a positive relationship between the percentage change in $PO_{@4mmol}$ and $\%VO_{2max@IS}$ ($p = 0.001$; $R^2_{adjusted} = 0.235$; figure 8C). $PO_{@4mmol}$ increased with 7.8 ± 3.6% in HIGH ($p = 0.001$) and 3.0 ± 4.5% in LOW ($p = 0.049$), and the percentage increase was larger in HIGH than in LOW ($p = 0.013$; figure 9C). The magnitude of improvement in HIGH was small compared to LOW (ES = 0.36). There was no relationship between percentage change in $PO_{@15min}$ and $\%VO_{2max@IS}$ ($p = 0.419$; $R^2_{adjusted} = -0.066$; figure 8D). $PO_{mean@15min}$ increased with 6.9 ± 4.5% in HIGH ($p = 0.001$) and 3.6 ± 5.1% in LOW ($p = 0.038$). When comparing the groups, no difference in percentage increase was observed ($p = 0.130$; figure 9D), and the ES analysis revealed a small practical effect of HIGH (ES = 0.28). There was a positive relationship between percentage change in the performance index and $\%VO_{2max@IS}$ ($p = 0.042$; $R^2_{adjusted} = 0.129$; figure 8E), and a positive relationship between percentage change in the performance index and time≥90%$VO_{2max@IS}$ ($p = 0.035$, $R^2_{adjusted} = 0.144$). The performance index increased with 7.7 ± 3.4% in HIGH ($p = 0.001$) and 3.6 ± 2.9% in LOW ($p = 0.002$). The percentage increase was larger in HIGH than in LOW ($p = 0.007$; figure 9E), and the ES analysis revealed a small practical effect of HIGH (ES = 0.37). There was no relationship between percentage change in $PO_{@40min}$ and $\%VO_{2max@IS}$ ($p = 0.272$; $R^2_{adjusted} = -0.031$; figure 8F). $PO_{@40min}$ remained unchanged in HIGH (3.9 ± 8.2%, $p = 0.109$) and increased with 5.9 ± 3.8% in LOW ($p = 0.001$), yet the percentage change did not differ between the groups ($p = 0.491$; figure 9F). The practical effect was trivial for LOW (ES = 0.15).

4.3 Maximal oxygen consumption

There tended to be a positive relationship between percentage change in VO_{2max} and $\%VO_{2max@IS}$ ($p = 0.089$; $R^2_{adjusted} = 0.138$; figure 8A). VO_{2max} increased with 8.0 ± 4.3% in HIGH ($p = 0.001$) and 2.7 ± 2.7% in LOW ($p = 0.010$). The percentage increase was larger in HIGH than in LOW ($p = 0.003$; figure 9A), and the magnitude of improvement in HIGH was moderate compared to LOW (ES = 0.55).

4.5 Fractional utilization of maximal oxygen consumption

$\%VO_{2max@4mmol}$ remained unchanged in both HIGH (-1.1 ± 4.3%-points, $p = 0.403$) and LOW (-1.3 ± 4.4%-points, $p = 0.351$), and no difference in percentage point change was observed between the groups ($p = 0.914$; table 5). The ES analysis revealed a trivial practical effect of HIGH (ES = 0.03). $\%VO_{2max@15min}$ tended to decrease in HIGH (-1.9 ± 3.3%-points, $p = 0.086$) and decreased with -2.7 ± 2.4%-points in LOW ($p = 0.004$). There was no difference in percent point change between HIGH and LOW ($p = 0.507$; table 5), and the practical effect of HIGH was trivial (ES = 0.19).

4.6 Gross efficiency

$GE_{@175W}$ remained unchanged in HIGH (0.1 ± 0.5%-points, $p = 0.692$) and increased with 0.7 ± 1.0%-points in LOW ($p = 0.038$). There was a tendency to difference between HIGH and LOW in percent point change ($p = 0.065$; table 5), with a moderate practical effect of LOW (ES = 0.82). $GE_{@225W}$ remained unchanged in HIGH (0.3 ± 0.7%-points, $p = 0.160$) and increased with 0.6 ± 0.7%-points in LOW ($p = 0.013$). There was no difference in percent point change between the groups ($p = 0.369$; table 5), and the ES analysis revealed a small practical effect of LOW (ES = 0.43).

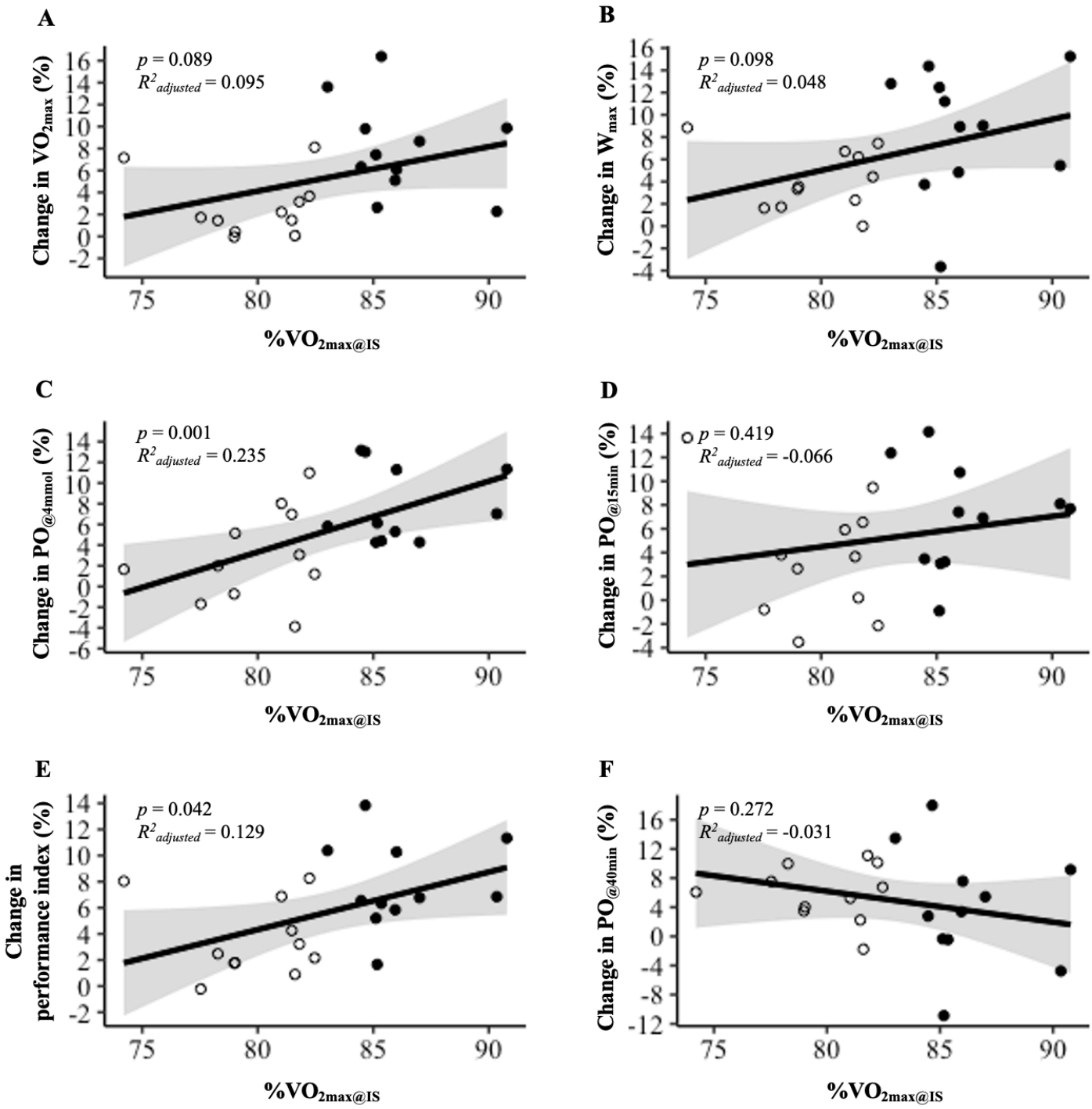

Figure 8: *Linear regressions for average percentage of maximal oxygen consumption elicited during interval-sessions (%$VO_{2max@IS}$) and percentage change in A) maximal oxygen consumption (VO_{2max}), B) maximal aerobic power output (W_{max}), C) power output at 4 mmol·L^{-1} lactate concentration ($PO_{@4mmol}$), D) 15-minutes mean power output ($PO_{@15min}$), E) performance index, and F) 40-minutes mean power output ($PO_{@40min}$), when controlling for baseline values. Individual datapoints for HIGH (black dots) and LOW (white dots), and pooled regressions slopes (solid lines) with 95% confidence limits (light gray areas) are shown.*

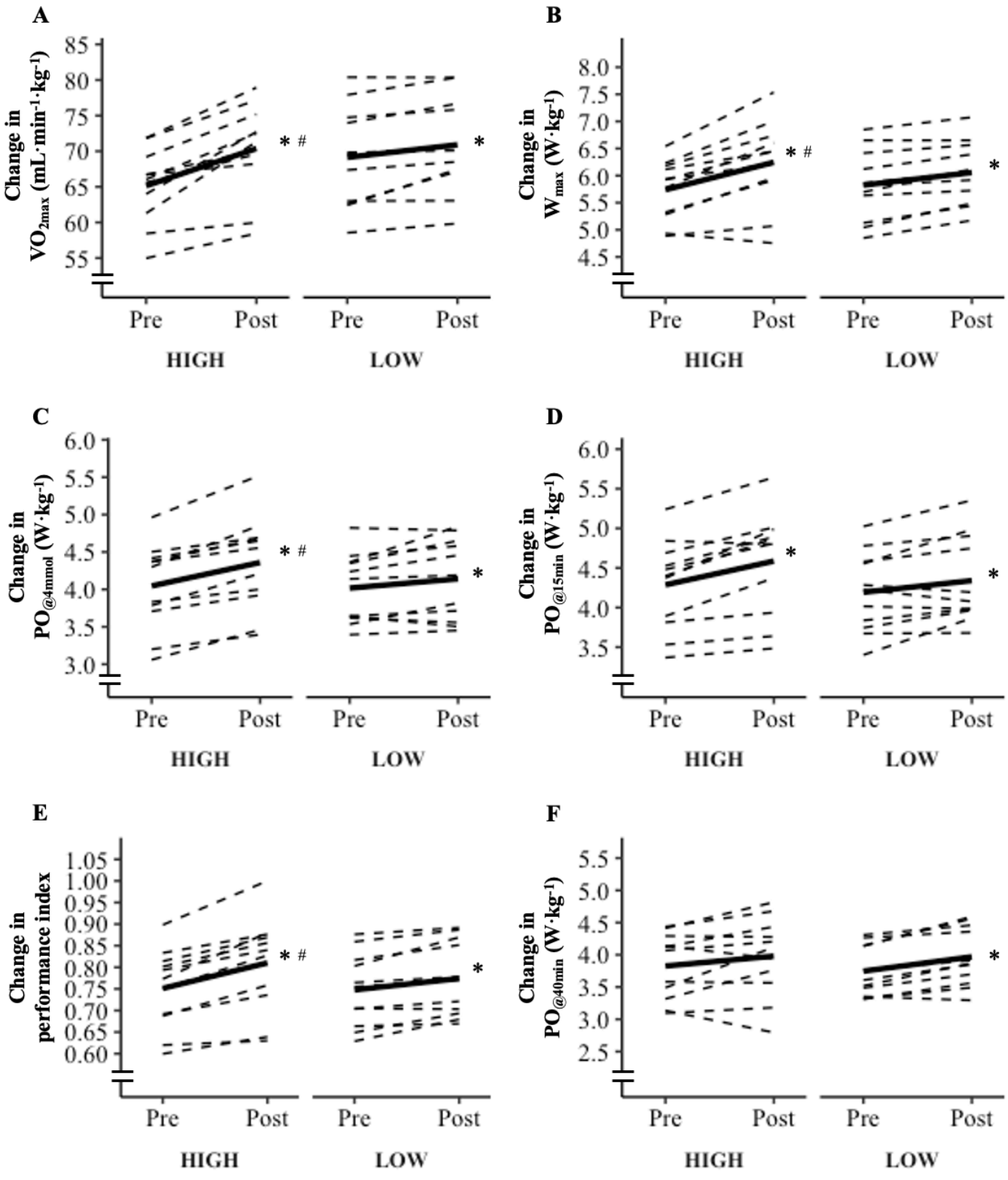

Figure 9: *Individual data points (dotted lines) and mean values (solid lines) for A) maximal oxygen consumption (VO_{2max}), B) maximal aerobic power output (W_{max}), C) power output at 4 mmol·L^{-1} lactate concentration ($PO_{@4mmol}$), D) 15-minutes mean power output ($PO_{@15min}$), E) performance index, and F) 40-minutes mean power output ($PO_{@40min}$) before (pre) and after (post) the intervention in HIGH and LOW, respectively. * Significantly different from pre ($p < 0.050$). # Significant difference in percentage change from pre to post compared to LOW ($p < 0.050$).*

Table 5: *Physiological and performance-related measures before (pre) and after (post) the training intervention in HIGH and LOW, respectively.*

	HIGH		LOW	
	Pre	**Post**	**Pre**	**Post**
Body mass (kg)	69.2 ± 9.8	68.8 ± 9.2	74.7 ± 8.2	74.9 ± 8.3
P_{peak} ($W \cdot kg^{-1}$)	17.5 ± 3.1	16.5 ± 2.9 *	18.5 ± 2.2	17.8 ± 2.6 $
VO_{2max}-test				
HR_{max} (bpm)	197 ± 8	197 ± 6	188 ± 5	188 ± 7
$[La^-]_{max}$ ($mmol \cdot L^{-1}$)	11.14 ± 1.29	12.02 ± 1.90	11.99 ± 2.50	12.4 ± 2.38
RPE_{max} (6-20)	19.2 ± 1.0	19.5 ± 0.7	19.3 ± 0.5	19.2 ± 0.8
Blood lactate profile				
$\%VO_{2max@4mmol}$ (%)	84.1 ± 5.2	82.9 ± 4.5	80.8 ± 5.2	79.5 ± 5.1
$GE_{@175W}$ (%)	19.2 ± 0.7	19.4 ± 0.5 €	18.6 ± 0.9	19.3 ± 1.1 *
$GE_{@225W}$ (%)	20.0 ± 0.6	20.4 ± 0.5	19.4 ± 0.6	20.0 ± 0.8 *
15-minutes time trial				
$\%VO_{2max@15min}$ (%)	88.2 ± 3.3	86.3 ± 3.9 $	85.3 ± 4.6	82.6 ± 3.9 *
$HR_{mean@15min}$ (bpm)	183 ± 8	182 ± 6	173 ± 4	172 ± 6
$HR_{end@15min}$ (bpm)	190 ± 9	190 ± 7	180 ± 4	180 ± 8
$[La^-]_{end@15min}$ ($mmol \cdot L^{-1}$)	9.22 ± 2.59	9.68 ± 2.16	9.28 ± 3.56	9.90 ± 4.14
$RPE_{@15min}$ (6-20)	18.5 ± 1.2	19.1 ± 1.2	18.8 ± 1.0	18.9 ± 0.9
40-minutes time trial				
$HR_{mean@40min}$ (bpm)	179 ± 7	180 ± 6	171 ± 5	170 ± 5
$HR_{end@40min}$ (bpm)	195 ± 8	193 ± 6 $	183 ± 4	181 ± 6 $
$[La^-]_{mean@40min}$ ($mmol \cdot L^{-1}$)	5.54 ± 1.75	6.08 ± 2.38 #	4.93 ± 1.23	6.59 ± 1.78 *
$[La^-]_{end@40min}$ ($mmol \cdot L^{-1}$)	10.22 ± 2.41	10.99 ± 2.69 #	8.47 ± 2.54	11.48 ± 2.11 *
$RPE_{@40min}$ (6-20)	19.5 ± 0.8	19.7 ± 0.5	18.7 ± 1.0	19.3 ± 0.9 *

*P_{peak}, peak power in leg press; VO_{2max}, maximal oxygen consumption; HR_{max}, maximal heart rate during the VO_{2max}-test; $[La^-]_{max}$, blood lactate concentration 1 minute after the VO_{2max}-test; RPE_{max}, rate of perceived exhaustion immediately after the VO_{2max}-test; $\%VO_{2max@4mmol}$, fractional utilization of VO_{2max} at 4 $mmol \cdot L^{-1}$ blood lactate concentration; $GE_{@175W}$, gross efficiency at 175 W; $GE_{@225W}$, gross efficiency at 225 W; $\%VO_{2max@15min}$, fractional utilization of VO_{2max} during the 15-minutes time trial; $HR_{mean@15min}$, average heart rate during the 15-minutes time trial; $HR_{end@15min}$, heart rate at the end of the 15-minutes time trial; $[La^-]_{end@15min}$, blood lactate concentration 1 minute after the 15-minutes time trial; $RPE_{@15min}$, rate of perceived exhaustion immediately after the 15-minutes time trial; $HR_{mean@40min}$, average heart rate during the 40-minutes time trial; $HR_{end@40min}$, heart rate at the end of the 40-minutes time trial; $[La^-]_{mean@40min}$, average blood lactate concentration during the 40-minutes time trial; $[La^-]_{end@40min}$, blood lactate concentration 1 minute after the 40-minutes time trial; $RPE_{@40min}$, rate of perceived exhaustion immediately after the 40-minutes time trial. Values are mean ± SD. * Significantly different from pre ($p < 0.050$). $ Tendency to different from pre ($p < 0.100$ and > 0.050). # Significant difference in percentage change from pre to post compared to LOW ($p < 0.050$). € Tendency to difference in percentage change from pre to post compared to LOW ($p < 0.100$ and > 0.050).*

4.7 Hematological measures

BV increased with 3.6 ± 4.0% in HIGH (p = 0.013) and remained unchanged in LOW (-1.9 ± 3.3%, p = 0.111). The percentage change in HIGH was larger than in LOW (p = 0.005; table 6), and the magnitude of improvement in HIGH was moderate compared to LOW (ES = 0.69). PV increased with 5.2 ± 6.8% in HIGH (p = 0.033) and remained unchanged in LOW (-1.9 ± 6.3%, p = 0.361). The percentage change in HIGH was larger than in LOW (p = 0.031; table 6), and the ES analysis revealed a moderate practical effect of HIGH (ES = 0.72). RBCV remained unchanged in both HIGH (1.7 ± 3.4%, p = 0.113) and LOW (-2.0 ± 3.4%, p = 0.157). Yet, there was a difference in percentage change between the groups (p = 0.031; table 6), with a small practical effect of HIGH (ES = 0.39). [Hb] tended to decrease in HIGH (-2.2 ± 3.5% p = 0.051) and remained unchanged in LOW (1.4 ± 4.1%, p = 0.317). There was a difference in percentage change between the groups (p = 0.050; table 6), and the ES analysis revealed a moderate practical effect of LOW (ES = 0.55). Hb_{mass} and HCT did not change within groups, and there was no difference in change between the groups (table 6).

Table 6: *Hematological measures before (pre) and after (post) the intervention in HIGH and LOW, respectively.*

	HIGH		LOW	
	Pre	**Post**	**Pre**	**Post**
Hb_{mass} ($g \cdot kg^{-1}$)	12.8 ± 1.5	12.9 ± 1.5	13.1 ± 1.1	13.0 ± 1.3
[Hb] ($g \cdot dl^{-1}$)	15.4 ± 1.1	15.0 ± 1.1 $ #	15.1 ± 1.0	15.3 ± 1.2
HCT (%)	44.4 ± 2.5	43.7 ± 2.8	44.3 ± 2.3	44.3 ± 3.7
RBCV ($ml \cdot kg^{-1}$)	36.9 ± 3.8	37.5 ± 4.1 #	38.4 ± 2.9	37.6 ± 3.8
PV ($ml \cdot kg^{-1}$)	46.0 ± 3.0	48.3 ± 3.9 * #	48.5 ± 6.0	47.5 ± 5.7
BV ($ml \cdot kg^{-1}$)	82.9 ± 5.7	85.8 ± 6.3 * #	86.9 ± 8.0	85.2 ± 7.1

*Hb_{mass}, hemoglobin mass; [Hb], hemoglobin concentration; HCT, hematocrit; RBCV, red blood cell volume; PV, plasma volume; BV, blood volume. Values are mean ± SD. * Significantly different from pre ($p < 0.050$). $ Tendency to different from pre ($p < 0.100$ and > 0.050). # Significant difference in percentage change from pre to post compared to LOW ($p < 0.050$).*

4.8 Peak power in leg press

P_{peak} decreased with -5.4 ± 5.2% in HIGH (p = 0.008) and tended to decrease in LOW (-3.9 ± 6.1%, p = 0.059). No difference in percentage change was observed between the two groups (p = 0.551; table 5) and the ES analysis revealed a trivial practical effect of LOW (ES = 0.09).

4.9 Other physiological & performance-related measures

During the intervention, HR_{max}, $[La^-]_{max}$, RPE_{max}, $HR_{mean@15min}$, $HR_{end@15min}$, $[La^-]_{end@15min}$, $RPE_{end@15min}$, and $HR_{mean@40min}$ did not change within groups, and there was no difference in percentage change between the groups (table 5). $HR_{end@40min}$ tended to decrease in both HIGH (-1.0 ± 1.8%, $p = 0.091$) and LOW (-1.0 ± 1.7%, $p = 0.082$), with no difference in percentage change between the groups ($p = 0.987$; table 5). $La_{mean@40min}$ remained unchanged in HIGH (8.9 ± 25.5%, $p = 0.297$) and increased with 39.7 ± 39.8% in LOW ($p = 0.009$). When comparing the groups, the percentage change was larger in LOW than in HIGH ($p = 0.042$; table 5). $La_{end@40min}$ remained unchanged in HIGH (10.8 ± 27.9%, $p = 0.351$) and increased with 42.5 ± 33.1% in LOW ($p = 0.001$). The percentage change was larger in LOW than in HIGH ($p = 0.004$; table 5). $RPE_{end@40min}$ remained unchanged in HIGH (1.1 ± 4.7%, $p = 0.506$) and increased with 3.0 ± 3.9% in LOW ($p = 0.025$), yet there was no difference in percentage change between the groups ($p = 0.184$; table 5).

4.10 Explaining regression analyses

4.10.1 Maximal oxygen consumption & 15- & 40-minutes mean power output

There tended to be a positive relationship between absolute change in $PO_{@15min}$ and absolute change in VO_{2max} ($p = 0.084$, $R^2_{adjusted} = 0.090$). There was no relationship between absolute change in $PO_{@40min}$ and absolute change in VO_{2max} ($p = 0.180$, $R^2_{adjusted} = 0.017$).

4.10.2 Fractional utilization of maximal oxygen consumption at 4 mmol·L^{-1} blood lactate concentration & 15- & 40-minutes mean power output

There was no relationship between absolute change in $PO_{@15min}$ and absolute change in $\%VO_{2max@4mmol}$ ($p = 0.442$, $R^2_{adjusted} = -0.036$). There was no relationship between absolute change in $PO_{@40min}$ and absolute change in $\%VO_{2max@4mmol}$ either ($p = 0.576$, $R^2_{adjusted} = -0.065$).

4.10.3 Gross efficiency at 225 W & 15- & 40-minutes mean power output

No relationship between absolute change in $PO_{@15min}$ and absolute change in $GE_{@225W}$ was observed ($p = 0.815$, $R^2_{adjusted} = -0.099$). There was no relationship between absolute change in $PO_{@40min}$ and absolute change in $GE_{@225W}$ either ($p = 0.087$, $R^2_{adjusted} = -0.106$).

4.10.4 BV & maximal oxygen consumption

There was a positive relationship between absolute change in VO_{2max} and absolute change in BV ($p = 0.001$, $R^2_{adjusted} = 0.419$; figure 10).

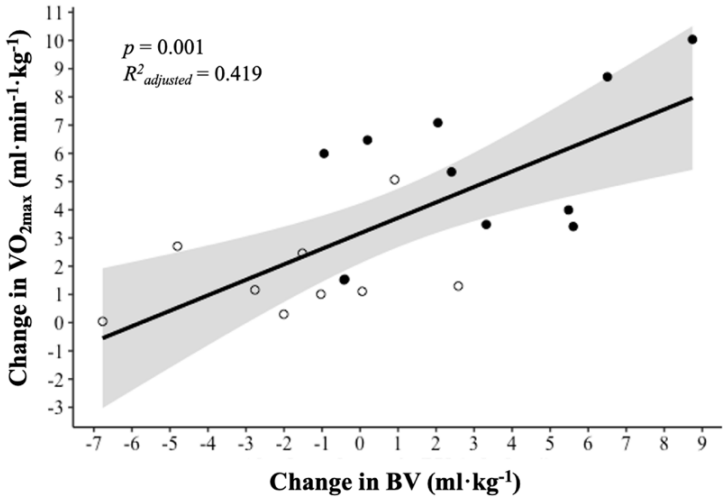

Figure 10: *Linear regression for change in maximal oxygen consumption (VO_{2max}) and change in blood volume (BV) when controlling for VO_{2max} at baseline. Individual datapoints for HIGH (black dots) and LOW (white dots) and a pooled regression slope (solid line) with 95% confidence limits (light gray area) are shown.*

4.10.5 Fractional utilization of maximal oxygen consumption at 4 mmol·L^{-1} blood lactate concentration & training intensity during interval-sessions

There was a positive correlation between $\%VO_{2max@4mmol}$ at baseline and $\%VO_{2max@IS}$ ($p = 0.014$; $R^2_{adjusted} = 0.231$; figure 11A), and between $\%VO_{2max@4mmol}$ at baseline and time≥$90\%VO_{2max@IS}$ ($p = 0.031$, $R^2_{adjusted} = 0.172$; figure 11B).

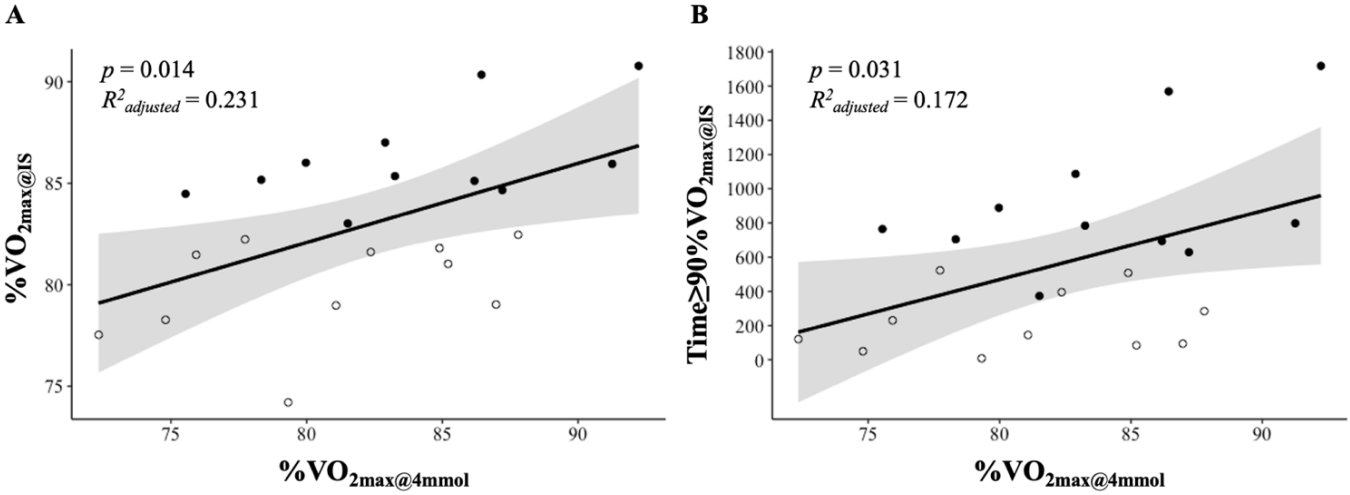

Figure 11: *Linear correlations for fractional utilization of maximal oxygen consumption at 4 mmol·L^{-1} blood lactate concentration ($\%VO_{2max@4mmol}$) at baseline and A) average percentage of maximal oxygen consumption elicited during interval-sessions ($\%VO_{2max@IS}$), and B) time spent ≥ 90% of maximal oxygen consumption during interval-sessions (time≥ $90\%VO_{2max@IS}$). Individual datapoints for HIGH (black dots) and LOW (white dots), and pooled correlation slopes (solid line) with 95% confidence limits (light gray areas) are shown.*

5 Discussion

The main finding of the present study is that the percentage increase in $PO_{@4mmol}$ and the performance index both were positively related to $\%VO_{2max@IS}$. Also, there tended to be a positive relationship between $\%VO_{2max@IS}$ and percentage increase in VO_{2max} and W_{max}. Comparing the groups, the percent increase in VO_{2max}, W_{max}, $PO_{@4mmol}$, and the performance index were larger in HIGH than in LOW.

5.1 Endurance performance variables

To our knowledge, the present study is the first observing a positive relationship between $\%VO_{2max@IS}$ and improvement in $PO_{@4mmol}$ and a performance index. These findings suggest that training at higher percentages of VO_{2max} could be advantageous to improve endurance performance. However, as the W_{max} improvement only tended to be related to $\%VO_{2max@IS}$, and the changes in $PO_{@15min}$ and $PO_{@40min}$ were not, our findings are somewhat inconclusive.

As a significant correlation previously has been reported between W_{max} attained during an incremental test and a 20 km cycle trial (Hawley & Noakes, 1992), W_{max} is considered a good predictor of cycling performance (Faria et al., 2005). W_{max} improved in both groups, but to a greater extent in HIGH compared to LOW. Consistently, an improved W_{max} has been observed following a twelve-weeks HIT-intervention (Skovereng et al., 2018). Also, compared to constant-intensity intervals (CI-intervals), varied-intensity intervals (VI-intervals) have been observed to elicit a longer time≥$90\%VO_{2max}$ acutely (Almquist et al., 2020; Bossi et al., 2020; Rønnestad & Hansen, 2016), and induce superior W_{max} improvements over time (Rønnestad et al., 2014; Rønnestad, Hansen, et al., 2020). As an increased W_{max} previously has been reported after a period of heavy strength training (Rønnestad et al., 2015; Rønnestad et al., 2017), this could potentially have affected W_{max} and our other performance-related measures. However, as heavy strength training did not differ between the groups and P_{peak} in leg press did not improve, this has most likely not influenced our results. More likely, the greater VO_{2max} and $PO_{@4mmol}$ improvements in HIGH compared to LOW may have contributed.

Consistent with our finding of a greater $PO_{@4mmol}$ improvement in HIGH compared to LOW, Turnes et al. (2016) observed that the group spending the longest time at VO_{2max} also improved power output at lactate threshold to the greatest extent. A greater (Rønnestad,

Hansen, et al., 2020) and tendency to greater (Rønnestad et al., 2014) $PO_{@4mmol}$ improvement have also been observed following VI-intervals known to elicit a longer time≥90%VO_{2max} compared to CI-intervals. Moreover, as improvements in lactate threshold has been associated with an increased skeletal muscle mitochondrial density and oxidative enzyme concentration (Midgley et al., 2007), the greater $PO_{@4mmol}$ improvement in HIGH supports the suggestion that spending longer times at higher intensities is important in the promotion of peripheral adaptions (Denadai et al., 2006).

As for $PO_{@15min}$, the almost twice as large improvement in HIGH was not significantly different from LOW. However, supported by the small practical effect of HIGH, this difference could still be of importance for endurance athletes. Comparing our results to others, Rønnestad, Hansen, et al. (2020) observed that VI-intervals known to elicit a longer time≥90%VO_{2max} induced superior improvements in mean power output during a 20-minutes time trial compared to CI-intervals. As for the performance index, the improvement in HIGH was greater than in LOW. Considering that the performance index is composed of three important endurance performance measures, this result should be emphasized when evaluating the overall importance of $\%VO_{2max@IS}$ on changes in endurance performance.

$PO_{@40min}$ remained unchanged in HIGH and increased in LOW, and there was no difference in change between the groups. These findings were somewhat unexpected considering previous observations of an improved $PO_{@40min}$ following HIT-interventions (Rønnestad et al., 2014; Skovereng et al., 2018) and a tendency to greater $PO_{@40min}$ improvement following VI-intervals known to elicit a longer time≥90%VO_{2max} compared to CI-intervals (Rønnestad et al., 2014). Moreover, they were unexpected considering the greater VO_{2max}, W_{max}, and $PO_{@4mmol}$ improvements in HIGH compared to LOW. As an outstanding exercise efficiency have been suggested to explain the high performance-level of elite athletes possessing relatively modest VO_{2max} values (Foster & Lucia, 2007; Lucia et al., 2003; Lucia et al., 2008), the increased $GE_{@175W}$ and $GE_{@225W}$ in LOW could potentially have compensated for the smaller VO_{2max} improvement, influencing the $PO_{@40min}$ and $PO_{@15min}$ results. However, such a compensatory effect should also have affected $PO_{@4mmol}$ and W_{max}, which it appears that it did not. More likely to have affected the $PO_{@40min}$ results, the increased $RPE_{@40min}$, $[La^-]_{mean@40min}$, and $[La^-]_{end@40min}$ in LOW suggest that LOW did not perform the 40-minutes time trial at pre-test until exhaustion. Furthermore, the group differences in $[La^-]_{mean@40min}$ and

$[La^-]_{end@40min}$ indicate that LOW increased their effort from pre- to post-test more than HIGH. Yet speculative, this may be a result of potentially limited experience with time trial pacing in our young participants.

Endurance performance is mainly determined by VO_{2max}, $\%VO_{2max}$, and exercise efficiency (Joyner & Coyle, 2008). Yet, the only relationship we observed between absolute changes in our time trial-variables and absolute changes in VO_{2max}, $\%VO_{2max@4mmol,}$ and $GE_{@225W}$, was a tendency to a positive relationship between $PO_{@15min}$ and VO_{2max}. Accordingly, Skovereng et al. (2018) did not observe a correlation between absolute changes in GE and $PO_{@40min}$ following their twelve-weeks HIT-intervention in well-trained cyclists. Opposed to our findings, they did observe a moderate positive correlation between absolute changes in VO_{2peak} and $PO_{@40min}$ (Skovereng et al., 2018).

5.2 Maximal oxygen consumption

The tendency to a positive relationship between $\%VO_{2max@IS}$ and change in VO_{2max} partially support the suggested superior effect of training near VO_{2max} to improve VO_{2max} (Buchheit & Laursen, 2013; Midgley & Mc Naughton, 2006; Midgley et al., 2006; Thevenet et al., 2007; Turnes et al., 2016; Wenger & Bell, 1986). Notably, Turnes et al. (2016) did not observe a positive correlation between time spent at VO_{2max} and VO_{2max} improvements. As for why we observed a tendency and they did not, it is possible that $\%VO_{2max@IS}$ measured during all twenty-one interval-sessions provided a more accurate measure of the VO_2 actually elicited during interval-sessions than time spent at VO_{2max} measured during two out of twelve HIT-sessions (Turnes et al., 2016). Moreover, considering the relatively small differences in training intensity within both studies, the longer duration of the present study may have increased the likelihood of detecting a relationship between training intensity and training adaptions in the already trained cyclists.

As for group comparisons, HIGH performed the interval-sessions at a higher $\%VO_{2max@IS}$ than LOW, spent a longer time≥$90\%VO_{2max}$, and enhanced VO_{2max} to a greater extent. Consistently, Turnes et al. (2016) observed that the group spending the longest time at VO_{2max} also got the greatest VO_{2max} improvements. Moreover, VI-intervals known to elicit a longer time≥$90\%VO_{2max}$ have been observed to induce superior VO_{2max} improvements compared to CI-intervals (Rønnestad et al., 2014; Rønnestad, Hansen, et al., 2020). Considering that

training at or near VO_{2max} is thought to maximally stress the O_2 delivery and utilization systems (Buchheit & Laursen, 2013; Midgley & Mc Naughton, 2006; Midgley et al., 2006), it is likely that the higher $\%VO_{2max@IS}$ in HIGH facilitated a more effective stimulus for enhancing VO_{2max} than the lower $\%VO_{2max@IS}$ in LOW.

Breaking it down, the O_2 delivery capacity, being a product of the maximal cardiac output (Q_{max}) and the blood´s O_2-carrying capacity, is considered the main limitation to VO_{2max} (Lundby et al., 2017). In HIGH, the unchanged Hb_{mass} and RBCV indicates that the O_2-carrying capacity remained unaltered, even though [Hb] tended to decrease. Considering that enhancements of Hb_{mass} and RBCV appears to be very difficult to induce in already endurance trained individuals (Wehrlin et al., 2016), these findings were not unexpected. As for Q_{max}, it is possible that the expanded PV and BV in HIGH facilitated an increased venous return, leading to an enhanced end-diastolic volume, stroke volume (SV), and Q_{max} via the Frank-Starling mechanism (Convertino et al., 1991; Coyle et al., 1986; Hopper et al., 1988; Kanstrup & Ekblom, 1982). Noteworthy, a BV-mediated increase in Q_{max} is argued to have little impact on O_2 delivery and consequently VO_{2max} if only PV and not also RBCV is expanded (Keiser et al., 2015; Lundby et al., 2017; Montero et al., 2015; Warburton et al., 2000). Still, we observed an increased VO_{2max} in HIGH, and LOW increased VO_{2max} despite that all hematological measures remained unchanged. Collectively, these findings contribute to highlight the multifactorial mechanisms of VO_{2max} improvement (di Prampero, 2003; di Prampero & Ferretti, 1990; Skattebo, Bjerring, et al., 2020). Yet speculative, cardiac remodeling, angiogenesis, and mitochondrial biogenesis could have contributed to the increased VO_{2max} in both groups, by elevating Q_{max} and widening the arterial-venous O_2 difference (a-vO_2diff; Skattebo, Bjerring, et al., 2020). However, as the O_2 extraction reserve is observed to be minimal in well-trained individuals (Mortensen et al., 2005), there was most likely little “room” for increasing the a-vO_{2diff} in our cyclists (Lundby et al., 2017).

Comparing the groups, the difference in RBCV was probably a result of the non-significant increase and decrease in HIGH and LOW, respectively. As Hb_{mass} did not differ between the groups, and [Hb] was reduced in HIGH compared to LOW, a superior O_2-carrying capacity was presumably not what induced the greater VO_{2max} improvement in HIGH. More likely, the greater PV and BV expansions in HIGH compared to LOW may have contributed. Possibly explaining these differences, training intensity appears to be the major stimulus for a training-

induced BV expansion (Convertino, 1991). Notably, a positive relationship between absolute changes in BV and VO_{2max} was observed.

5.3 Fractional utilization of maximal oxygen consumption

In accordance with our findings, most studies cannot find an increased $\%VO_{2max}$ after eight to twelve weeks of endurance training in already trained cyclists (Rønnestad, Ellefsen, et al., 2014; Rønnestad et al., 2017; Sunde et al., 2010). However, over a four-year period, young Finnish cross-country skiers increased their $\%VO_{2max}$ from 73 to 78% (Rusko, 1987), and a world junior champion cyclist increased his $\%VO_{2max@4mmol}$ from 82 to 86% (Rønnestad et al., 2019). This suggests that $\%VO_{2max}$ can be improved in trained individuals, but that systematic training over several years may be required (Tønnessen & Rønnestad, 2018). As $\%VO_{2max}$ is mainly determined by peripheral factors (Coyle, 1995; Holloszy & Coyle, 1984; Ivy et al., 1980; Joyner & Coyle, 2008) and the improved $PO_{@4mmol}$ suggests that peripheral adaptions occurred in our cyclists (Midgley et al., 2007), it could be argued that $\%VO_{2max@4mmol}$ and $\%VO_{2max@15min}$ should have been improved as well. Yet speculative, the potential effects of peripheral adaptions may have been diminished by the increased VO_{2max}.

$\%VO_{2max}$ is argued to be well associated with endurance performance (Coyle et al., 1991). However, when the relationship between $\%VO_{2max}$ and power output at lactate threshold recently was investigated in 108 regional, national, and elite cyclists, only a moderate correlation was observed (Støren et al., 2013). The authors hence argued that $\%VO_{2max}$ at lactate threshold is a poor determinant of power output at lactate threshold, and also likely a poor determinant of endurance performance for competitive cyclists (Støren et al., 2013). Consistently, positive relationships between absolute change in $\%VO_{2max@4mmol}$ and absolute changes in $PO_{@15min}$ and $PO_{@40min}$ was not observed in the present study. Moreover, both groups increased $PO_{@15min}$ despite that $\%VO_{2max@15min}$ tended to decrease in HIGH and decreased in LOW.

5.4 Gross efficiency

To improve the exercise efficiency of endurance athletes, it is suggested that a high volume of low-intensity training over time is required (Scrimgeour et al., 1986). In fact, an improved exercise efficiency is reported after months to years of systematic endurance training (Conley et al., 1984; Jones, 2006), but not after weeks of HIT (Rønnestad, Hansen, et al., 2014;

Rønnestad, Hansen, et al., 2020; Skovereng et al., 2018). The present nine-weeks training intervention was hence not expected to induce any GE improvements. Yet, $GE_{@175W}$ and $GE_{@225W}$ increased in LOW, and the $GE_{@175W}$ improvement tended to be different from HIGH. Noteworthy, $GE_{@225W}$ was lower and $GE_{@175W}$ tended to be lower in LOW compared to HIGH at baseline, and a moderate negative correlation has been observed between initial GE and percentage change in GE (Skovereng et al., 2018). As an increased exercise efficiency previously has been observed after eleven weeks of concurrent strength and endurance training in well-trained cyclists (Vikmoen et al., 2016), this could potentially have affected our results. However, as heavy strength training and changes in P_{peak} in leg press did not differ between the groups, $GE_{@175W}$ and $GE_{@225W}$ were most likely not influenced by this.

5.5 Interval-session measures

Despite $\%FTP_{@IS}$ being the same in both groups, $\%VO_{2max@IS}$ and time≥$90\%VO_{2max@IS}$ differed between the groups in all periods. $\%HR_{max@IS}$, RPE, $[La^-]$, perceived well-being in the legs, and sRPE however, did not. As percentage of HR_{max} and RPE both are convenient and commonly used measures of intensity, this is noteworthy. Nevertheless, it suggests that it was the differences in VO_2 during interval-sessions that induced the different training adaptions in HIGH and LOW. It appears that the training induced stimuli responsible for the improved function and/or increased quantity of the heart, capillaries, and mitochondria are enhanced when training intensity is increased (Tønnessen & Rønnestad, 2018). Consequently, the higher $\%VO_{2max@IS}$ in HIGH may have promoted a greater combination of central and peripheral adaptions, leading to the superior improvements in endurance performance and physiological determinants of endurance performance (Daussin et al., 2007; Laursen & Jenkins, 2002; Turnes et al., 2016). How HIGH were able to obtain and sustain a greater $\%VO_{2max@IS}$ and a longer time≥$90\%VO_{2max}$ compared to LOW, may be explained by the non-significant, yet ~3% higher $\%VO_{2max@4mmol}$ and $\%VO_{2max@15min}$ at baseline. This is further supported by the positive correlations between $\%VO_{2max@4mmol}$ at baseline and $\%VO_{2max@IS}$ and time≥$90\%VO_{2max@IS}$. As for sRPE and perceived well-being in the legs, there was no change within groups, and no differences between groups in any of the periods. This indicates that the intervention was not perceived as gradually more exhausting, nor as more exhausting for HIGH than for LOW.

5.6 Practical implications

Collectively, our findings allow us to recommend cyclists to perform their interval-sessions at power outputs eliciting a VO_2 close to their VO_{2max} to enhance VO_{2max} and other physiological determinants of endurance performance. As for the practical application of our findings, measuring VO_2 during every interval-session to verify the $\%VO_{2max@IS}$ is hardly feasible. As $\%HR_{max@IS}$ was not associated with $\%VO_{2max@IS}$ in the present study, we cannot recommend $\%HR_{max@IS}$ as an alternative intensity-measure. Instead, one could measure VO_2 during one interval-session, find the power output eliciting the wanted $\%VO_{2max@IS}$, and use this at the following interval-sessions. Still, relatively frequent VO_{2max}-tests and VO_2-measurements at interval-sessions would be required to account for potential improvements in training status. Noteworthy, we observed that $\%VO_{2max@4mmol}$ at baseline was positively related to $\%VO_{2max@IS}$ and time≥$90\%VO_{2max@IS}$, and that both $\%VO_{2max@IS}$ and time≥$90\%VO_{2max@IS}$ were positively related to the performance index improvements. This indicates that performing 5x8-minutes intervals at a power output corresponding to $PO_{@40min}$ may be effective to enhance endurance performance in cyclists with a high $\%VO_{2max@4mmol}$. Furthermore, it could be speculated whether cyclists with a lower $\%VO_{2max@4mmol}$ may benefit from performing shorter work-intervals at higher power outputs to increase $\%VO_{2max@IS}$.

5.7 Conclusion

In conclusion, the present study demonstrates that performing twenty-one interval-sessions over nine weeks at a high $\%VO_{2max@IS}$ induces greater improvements in endurance performance and physiological determinants of endurance performance compared to performing interval-sessions at a lower $\%VO_{2max@IS}$ in cyclists.

5.8 Perspectives

As we only investigated changes in relation to $\%VO_{2max@IS}$ in the range of 74.2-90.8%, we cannot conclude whether our observed relationships exist outside this range. Considering that VI-intervals previously have been reported to induce superior training adaptions compared to CI-intervals (Rønnestad et al., 2014; Rønnestad, Hansen, et al., 2020), the order the cyclists performed the three different interval-designs in may have influenced our results, despite that the order was randomized. Furthermore, performing interval-sessions with shorter total durations and higher power outputs would potentially allowed more direct comparison with other studies. Additionally, a larger sample size would have been advantageous considering

the power of our analyses. As for our measurement of VO_{2max} and $PO_{@40min}$ every third week, this is a great strength of the present study as $\%VO_{2max@IS}$ and $\%FTP_{@IS}$ thus was "calibrated" for potential improvements in training status twice during the intervention. As the onset of endurance training is observed to induce considerable physiological adaptions in untrained individuals (Laursen & Jenkins, 2002), and the effects of small differences in $\%VO_{2max@IS}$ may be difficult to detect, it is crucial that future studies only include well-trained individuals. To get further insights in how training at different percentages of VO_{2max} effects central adaptions, additional measurements of SV, plasma erythropoietin concentration, and PV-regulating proteins could be included. As could muscle biopsies to get a greater understanding of potential peripheral adaptions.

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7 Appendix

Appendix A: Training history

Kartlegging av treningshistorikk	
FP nr:	
Navn:	
Antall år du har "satset" på sykling:	5
Antall nasjonale ritt i 2021:	14
Antall internasjonale ritt 2021:	4
Beste resulat 2021 sesongen:	

Hvordan føre treningshistorikk

Du skal føre dine gjennomsnittlige minutter med trening per uke de siste 2, 4 og 12 ukene, samt totalt antall minutter trening det siste året i tabellen under.

Treningstid skal føres som antall minutter (2 timer og 43 minutter = (2*60) + 43 = 163 minutter).

Det skal *kun* føres inn **tall** i tabellen (feks. ikke skriv "min" bak).

Gjennomsnittlige minutter trening per uke siste	Sone 1	Sone 2	Sone 3	Sone 4	Sone 5	Maks styrke	Stab styrke	Totalt
2 ukene før første test (**gjennomsnitt per uke**):	173	261	67	36	4			540
4 ukene før første test (**gjennomsnitt per uke**):	147	248	79	61	57			592
12 ukene før første test (**gjennomsnitt per uke**)	258	375	66	58	54			812
Siste år **(totalt antall minutter)**:	15000	20000	5000	400	350			40750

Appendix B: Written informed consent

Høgskolen i Innlandet

[22.06.2021]

VIL DU DELTA I FORSKNINGSPROSJEKTET:

«OPTIMALISERING AV INTERVALLØKTER»

FORMÅLET MED PROSJEKTET OG HVORFOR DU BLIR SPURT

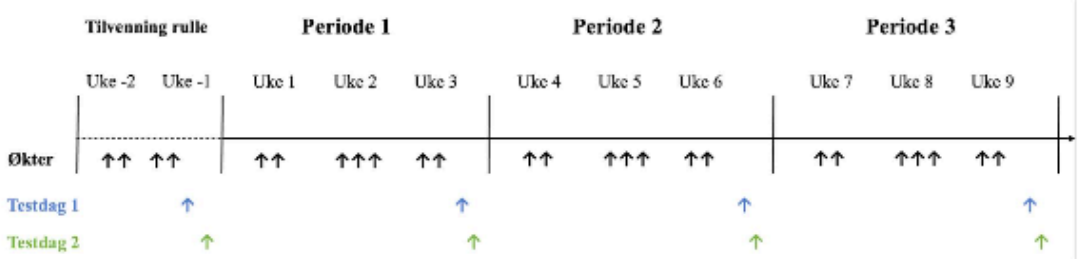
Dette er et spørsmål til deg om å delta i et forskningsprosjekt hvor formålet er å skaffe ny kunnskap om hvordan variasjon i arbeidsintensitet under dragene i en intervalløkt påvirker det maksimale oksygenopptaket (VO_{2maks}) og sykkelprestasjon. I dette skrivet finner du informasjon om målene med prosjektet og hva deltakelse vil innebære for deg.

Bakgrunn. Treningstid ≥ 90 % av VO_{2maks} har blitt foreslått å være et godt kriterium for å bedømme hvor effektiv treningen er med tanke på å forbedre VO_{2maks}. Underveis i en intervalløkt har man sett at det å variere arbeidsintensiteten under dragene i en intervalløkt gir mer tid ≥ 90 % av VO_{2maks} enn å gjennomføre dragene med konstant arbeidsbelastning. Da utholdenhetsprestasjon i stor grad avhenger av VO_{2maks}, vil det være svært gunstig for toppidrettsutøvere å vite hvordan de skal organisere dragene på intervalløktene sine for å så stor effekt som mulig på VO_{2maks} og prestasjon på hver enkelt økt og følgelig over tid.

Formål. I denne studien ønsker vi derfor undersøke hvordan tre ulike måter å organisere arbeidsintensiteten under dragene i en intervalløkt påvirker VO_{2maks} og sykkelprestasjon. Studien skal gjennomføres som et randomisert kryssforsøk, og består av en 9 uker lang treningsintervensjon delt inn i tre perioder på 3 uker. Hver periode inneholder 7 intervalløkter bestående av 5x8 min med 3 min pause mellom hvert drag, der den gjennomsnittlige watten på dragene er den samme, men måten dragene organiseres på varierer mellom hver 3 ukersperiode. Det vil bli gjennomført fysiologiske tester på to påfølgende dager (testdag 1 og testdag 2) både før intervensjonsstart, etter periode 1, etter periode 2 og etter periode 3 (Figur 1).

På prosjektet skal det skrives to masteroppgaver.

Vi ønsker totalt 36 godt trente landeveis- og terrengsyklister (over 7 timer utholdenhetstrening per uke siste 6 mnd før prosjektstart) i alderen 17-30 år til å delta i prosjektet. Forespørselen sendes til aktuelle trenere og utøvere i Innlandet og Oslo regionen.



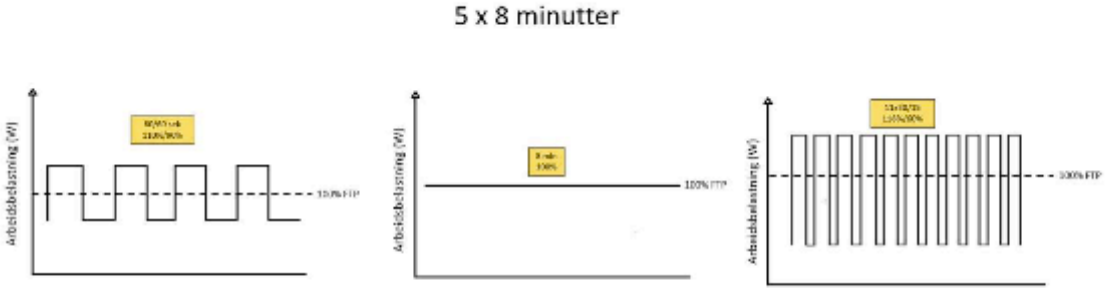
Figur 1: Oversikt over intervalløkter, testdag 1 og testdag 2 under den 9 uker lange treningsintervensjonen.

HVA INNEBÆRER PROSJEKTET FOR DEG?

For oversikt over studien, se Figur 1.

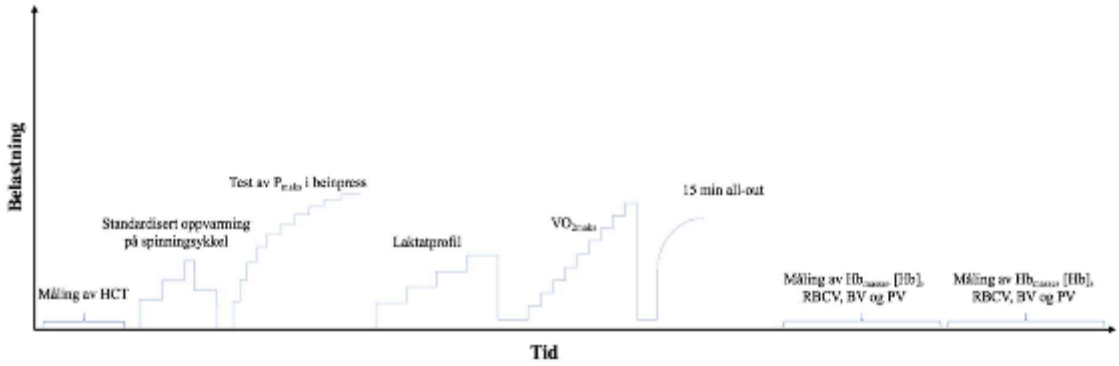
Forberedelser. Som deltaker i prosjektet skal du gjennomføre 2 ukentlige økter inne på rulle de siste 2 ukene før prosjektet starter. For at du skal ha den samme inngangen til testdagene, vil du bli bedt om å ta treningsfri dagen før testdag 1 ved alle testtidspunkter.

Gjennomføring. Som deltaker skal du gjennomføre en 9 uker lang treningsintervensjon bestående av tre perioder på 3 uker, der du i hver periode gjennomfører 7 intervalløkter bestående av 5 x 8 minutters drag med 3 minutter pause. Den gjennomsnittlige effekten på dragene tilsvarer den gjennomsnittlige effekten du klarer å holde på 40 minutter all-out (FTP) før hver periode, men måten dragene organiseres på vil variere mellom de tre periodene: i) 8 min på 100 % av FTP (FLAT), ii) 8 min med vekselsvis 60 sek på 110 % av FTP og 60 sek på 90 % av FTP (60/60) og iii) 8 min med vekselsvis 30 sek på 118 % av FTP og 15 sekunder på 60 % av FTP (30/30) (Figur 2). Det vil bli målt oksygenopptak under hvert drag på alle intervalløktene. Alle intervalløktene må gjennomføres enten på NTG Kongsvinger sitt rullerom eller på NTG Lillehammer sitt rullerom. Utenom intervalløktene står du fritt til å gjennomføre din vanlige lavintensive trening, samt styrketrening.

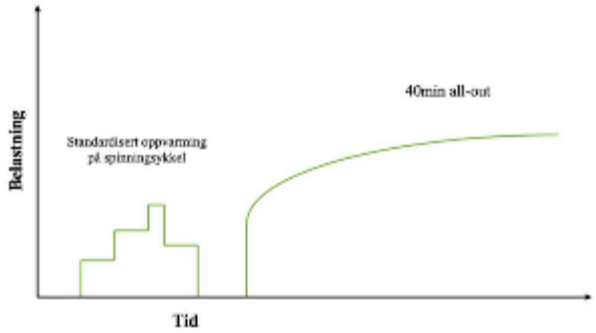


Figur 2: Oversikt over de tre forskjellige måtene å organisere arbeidsdraga i intervalløktene på.

Som deltaker skal du også gjennomføre fysiologiske tester på to påfølgende dager (testdag 1 og testdag 2) ved fire forskjellige testtidspunkt: før intervensjonsstart, etter periode 1, etter periode 2 og etter periode 3 (Figur 1). Testdag 1 vil bli gjennomført ved Høgskolen i Innlandet, Lillehammer sitt idrettsfysiologiske testlaboratorium, og består av blodprøver for bestemmelse av hematokrit, test av maksimal kraft i beinpress, en laktatprofil, test av VO_{2maks}, 15 minutter all-out og til slutt CO-rebreathing gjennomført i duplikat for bestemmelse av hemoglobinmasse, volum av røde blodceller, blodvolum og plasmavolum (Figur 3). Testdag 2 vil bli gjennomført på NTG Kongsvinger og NTG Lillehammer sine rullerom, og består av 40 minutter all-out sykling (Figur 4). Testene vil bli gjennomført til samme tid på døgnet ± 1 time. All trening skal loggføres i en utlevert treningsdagbok, og alle treningsdata og testresultater vil bli registrert elektronisk.



Figur 3: Oversikt over testdag 1.



Figur 4: Oversikt over testdag 2.

MULIGE FORDELER OG ULEMPER

Du som deltaker vil ha stor nytteverdi av å være med i denne studien. Du vil få tett oppfølging på 21 intervalløkter fordelt på 9 uker, samt testing av din fysiske form før, to ganger underveis og etter treningsintervensjonen. Du vil få god erfaring med hvordan de tre ulike intervalløktene skal gjennomføres på best mulig måte, og ikke minst få vite hvilken økt som gir *deg* den største effekten på VO_{2maks} og sykkelprestasjon. Erfaringene og resultatene du får med deg fra prosjektet er noe du kan dra stor nytte av i din videre trening.

Ulemper med å være med i denne studien er at du skal gjennomføre mye hardt fysisk arbeid. Intervalløktene og de fysiske testene vil oppleves som svært fysisk anstrengende, og målingene av VO_2 underveis kan oppleves som ubehagelig. CO-rebreathingen som benyttes for å måle blodvariablene kan også oppleves ubehagelig for noen. Under disse målingen skal du puste inn en liten mengde karbonmonoksid, men denne mengden er så liten at den ikke anses som helseskadelig. Studien vil også kunne oppleves relativt belastende da den den vil være tidskrevende og styre mye av treningen til din.

FRIVILLIG DELTAKELSE OG MULIGHET FOR Å TREKKE DITT SAMTYKKE

Det er frivillig å delta i prosjektet. Dersom du ønsker å delta, undertegner du samtykkeerklæringen på siste side. Du kan når som helst og uten å oppgi noen grunn trekke ditt samtykke. Det vil ikke ha noen negative konsekvenser for deg hvis du ikke vil delta eller senere velger å trekke deg. Du kan også kreve dataene dine slettet så lenge de er identifiserbare i datamaterialet. Dersom du ønsker å trekke deg eller har spørsmål til prosjektet, kan du kontakte prosjektleder (se kontaktinformasjon på siste side).

HVA SKJER MED OPPLYSNINGENE OM DEG?

Opplysningene som registreres om deg skal kun brukes slik som beskrevet under formålet med prosjektet. Prosjektgruppa skal arbeide videre med de innsamlede dataene etter at masteroppgavene er levert slik at prosjektet i sin helhet avsluttes i 2026.

Du har rett til innsyn i hvilke opplysninger som er registrert om deg og rett til å få korrigert eventuelle feil i de opplysningene som er registrert. Du har også rett til å få innsyn i sikkerhetstiltakene ved behandling av opplysningene. Alle data skal oppbevares på sikker server, Tjenester for sensitive data (TSD), ved Universitetet i Oslo som Høgskolen i Innlandet har databehandleravtale med. Du kan klage på behandlingen av dine opplysninger til Datatilsynet og institusjonen sitt personvernombud.

Vi behandler opplysningene konfidensielt og i samsvar med personvernregelverket. Det er bare medlemmer i prosjektgruppa som får tilgang på disse dataene. Navnet og kontaktopplysningene dine vil erstattes med en kode som lagres på egen navneliste adskilt fra øvrige data. Det er kun anonyme testresultater som publiseres, slik at du vil ikke kunne gjenkjennes i publikasjon.

FORSIKRING

Som deltaker i studien er du forsikret gjennom Høgskolen i Innlandets forsikring hos Gjensidige.

GODKJENNINGER

Etter ny personopplysningslov har behandlingsansvarlig Høgskolen i Innlandet og prosjektleder Bent Rønnestad et selvstendig ansvar for å sikre at behandlingen av dine opplysninger har et lovlig grunnlag. Dette prosjektet har rettslige grunnlag i EUs personvernforordning artikkel 6 nr. 1a og artikkel 9 nr. 2a og ditt samtykke. Du har rett til å klage på behandlingen av dine opplysninger til Datatilsynet.

Vi behandler opplysningene basert på ditt samtykke.

KONTAKTOPPLYSNINGER

Dersom du har spørsmål til prosjektet eller ønsker å trekke deg fra deltakelse, kan du kontakte:

Prosjektleder: professor Bent Rønnestad (tlf 95169656, epost: bent.ronnestad@inn.no)

Dersom du har spørsmål om personvernet i prosjektet, kan du kontakte personvernombudet ved institusjonen:

Høgskolen i Innlandets personvernombud:
https://www.inn.no/omhoegskolen/personvern/personvernombud

NSD – Norsk senter for forskningsdata AS: personvernombudet@nsd.no, telefon: 555 82 117.

JEG SAMTYKKER TIL Å DELTA I PROSJEKTET OG TIL AT MINE PERSONOPPLYSNINGER OG MINE DATA BRUKES SLIK DET ER BESKREVET

Sted og dato

Deltakers signatur

Deltakers navn med trykte bokstaver

Appendix C: Training diary

FP	Periode	Dato	Aktivitet	Sone 1	Sone 2	Sone 3	Sone 4	Sone 5	Maksstyrke	Basisstyrke	Øktscore	Følelse bein	Total trening
	1	07.10.2021	Bike	12	24	9	17	22			9	4	84
	1	08.10.2021	Bike	14	9	3	11	33			10	6	70
	1	10.10.2021	Bike	62	107	9	1	1			3	4	180
	1	11.10.2021	Bike	99	20	3	1	1			2	5	124
	1	12.10.2021	Bike	22	1	15	30	0			9	3	68
	1	13.10.2021	Bike	66	49	5	1	0			2	5	121
	1	14.10.2021	Bike	28	10	20	25	0			8	5	83
	1	15.10.2021	Bike	150	63	5	1	1			2	4	220
	1	16.10.2021	Bike	97	133	8	1	0			3	3	239
	1	17.10.2021	Bike	7	108	8	0	0			2	2	123
	1	18.10.2021	Bike	33	7	15	31	0			7	3	86
	1	19.10.2021	Bike	33	1	11	34	1			9	6	80
	1	20.10.2021	Bike	47	7	7	36	2			7	5	99
	1	21.10.2021	Bike	20	102	5	1	0			2	5	128
	1	22.10.2021	Run	54	37	12	3	0			5	3	106
	1	23.10.2021	Bike	190	56	54	0	0			2	6	300
	1	24.10.2021	Bike	7	134	10	1	0			2	2	152
	1	25.10.2021	Bike	3	87	5	0	0			2	7	95
	1	26.10.2021	Bike	57	16	51	37	1			8	5	162
	1	27.10.2021	Run	37	43	10	2	0			3	6	92
	1	28.10.2021	Bike	23	11	4	37	5			9	6	80
	1												0
	1												0
	1												0
	1												0
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FP	Periode			Sone 1	Sone 2	Sone 3	Sone 4	Sone 5	Maksstyrke	Basisstyrke	Øktscore	Følelse bein	Total trening
0	1			1061	1025	269	270	67	0	0	5,0	4,5	2692

Øktscore	
0	Hvile
1	Veldig, veldig lett
2	Lett
3	Moderat
4	Noe hardt
5	Hardt
6	
7	Veldig hardt
8	
9	
10	Ekstremt hardt

Opplevd følelse i beina	
9	Veldig, veldig dårlig
8	Veldig dårlig
7	Dårlig
6	Litt dårlig
5	Normal
4	Litt bra
3	Bra
2	Veldig bra
1	Veldig, veldig bra

Hvordan føre treningsdagbok

Utholdenhetstrening
Før antall minutter trening i de forskjellige sonene (utholdenhetstrening).
Velg riktig bevegelsesform i rullgardina.

Styrketrening
Før antall minutter med maksimal/generell styrketrening.

Øktscore og opplevd følelse i beina
Velg riktig verdi i rullgardina.