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Investigating Durability and Its Independence from Traditional Physiological Markers in Highly-trained Endurance Athletes

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Abstract

In recent years durability has been suggested as a fourth landmark of endurance sports. In contrast to threshold models, the maximal oxygen consumption ($\dot{V}O_{2max}$) and efficiency its focus lies on the physiological changes caused by fatigue. As previous studies have shown the parameters influencing endurance sports shift during prolonged exercise, which leads to a loss in performance. This highlights the importance of durability, which describes one's individual resilience towards fatigue. However, the connection or influence of durability on the established landmarks of endurance sports is still not yet clarified. The purpose of the present study is to investigate the connection between a loss in 5-minute maximal power output (ΔMPO_{5min}) and physiological landmarks such as VT1, VT2, $\dot{V}O_{2max}$ and GE under fresh conditions. Furthermore, a possible connection between ΔMPO_{5min} and the decrease in $\dot{V}O_{2max}$ and GE values has been analysed.

Seven well-trained amateur cyclists performed an incremental Ramp test and a GE base line test in order to identify their physiological profile. Based on their values they completed two 5min time trials, where the average power output was measured. Whilst the first trial was carried out in a non-fatigued-state, the second trial took place after a one-hour fatigue-protocol, which resulted in a preload of roughly 15 kj/kg. Afterwards correlations between the values and repeated measures ANOVA between the GE values were investigated.

The results showed no significant correlations between the drop in MPO_{5min} values and the GE, $\dot{V}O_{2max}$ and VT1 values. Absolute power output at VT2 however showed a significant negative correlation with reduction in MPO_{5min}. Furthermore, the repeated repeated measures ANOVA showed a significant negative effect of the fatigue-protocol on GE values.

In conclusion the findings suggest that disability cannot be predicted by the already well-established landmarks of endurance sports. This strengthens the hypothesis that durability can be seen as a fourth landmark, that needs to be further examined.

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1 Introduction

1.1 Physiological profiling

In recent years physiological profiling has led to a better understanding of athletes' performances. It serves as the foundation of training programming, load monitoring, intensity regulation and prediction of exercise performance (Maunder et al., 2021). The three most commonly assessed variables during physiological profiling are maximal oxygen uptake ($\dot{V}O_{2max}$), sustainable percentage of $\dot{V}O_{2max}$ (%$\dot{V}O_{2max}$) and exercise economy/efficiency, often described by gross efficiency (GE). Already back in 2008 Joyner and Coyle suggested those as the main parameters to predict performances in endurance sports.

In 1991, Joyner used these three parameters to create a model, that would predict the optimal marathon performance. He concluded that a hypothetical athlete with a $\dot{V}O_{2max}$ of 84 mL/min/kg, a lactate threshold of 85%$\dot{V}O_{2max}$ and an exceptional running economy would finish a marathon (42.195 km) in 1:57:58 h. His calculations where way below the record at the time, which was held by Belayneh Densamo, who finished the Rotterdam Marathon in 2:06:50 h (Díaz et al., 2018). Recent developments however saw the gap decline by more than 70%. The current world record stands at 2:00:35 h, whilst an unofficial record below the 2h-mark underlined the human potential under optimized conditions (Fernandes & Maldonado, 2024). Besides the previously mentioned parameters, Joyner hypothesized various other variables to explain the gap between his calculations and the record at the time. Besides the influence of genetics, which must be close to ideal, when thinking about the described hypothetical athlete, he also pointed towards substrate utilization and fatigue.

The importance of fatigue is a rather new approach in physiological profiling and makes up the focus of the present study. At first however the previous variables of physiological profiling should be explained more closely.

1.1.1 $\dot{V}O_{2max}$

$\dot{V}O_{2max}$ is defined as the maximum rate at which an individual can consume and utilize oxygen. It sets the upper limit of endurance exercise as athletes cannot operate at intensities above their $\dot{V}O_{2max}$ for extended periods. Furthermore, $\dot{V}O_{2max}$ is often used as a method of monitoring training effects, as well-trained athletes show higher numbers than untrained individuals (Bassett & Howley, 2000). Professional cyclists for example can achieve $\dot{V}O_{2max}$ vales above 80 mL/min/kg, which is double the amount of healthy not-trained individuals (Valenzuela et al., 2022, Väisänen et al., 2024).

$\dot{V}O_{2max}$ values differ largely between individuals and occur due to biological and methodological factors (Meyler et al., 2021). Whilst endurance training is the most beneficial way of increasing $\dot{V}O_{2max}$, findings by Bouchard et al. (1999) have shown a heterogeneous effect on the magnitude of improvement. The adaptations triggered by endurance training ranged from large to small changes, whilst roughly 20% of the individuals did not show any meaningful increase in $\dot{V}O_{2max}$.

Roughly 50% of the difference in training response is believed to stem from a heritability background, whilst the role of genetics on $\dot{V}O_{2max}$ trainability is not yet fully understood (Bouchard et al. 1999). Although, it is well documented that $\dot{V}O_{2max}$ values decline with age, numerous studies have pointed out little differences in actual trainability (Meyler et al., 2021). A study by McGuire et al. (2001) found a change in the primary drivers of increased $\dot{V}O_{2max}$, which shifts from both maximal cardiac output and arteriovenous oxygen difference to an exclusive increase of the latter. However, these changes that occur due to a reduction in maximum heart rate and maximum cardiac output did not influence the magnitude of change in $\dot{V}O_{2max}$. In contrast a difference in trainability has been found between the sexes with men showing larger adaptations than women (Montero & Diaz-Canestro, 2019). These differing changes of roughly 3% can be explained by different adaptive pathways (Sarzynski et al., 2017, Ansdell et al., 2020). Finally, a higher $\dot{V}O_{2max}$ baseline appears to be an indicator of less room for improvement, indicating a ceiling for this important parameter of endurance sports. In terms of methodological factors, it can be stated that the type, volume and intensity of training, as well the prescription of exercise intensity have shown an influence on $\dot{V}O_{2max}$ values (Meyler et al., 2021).

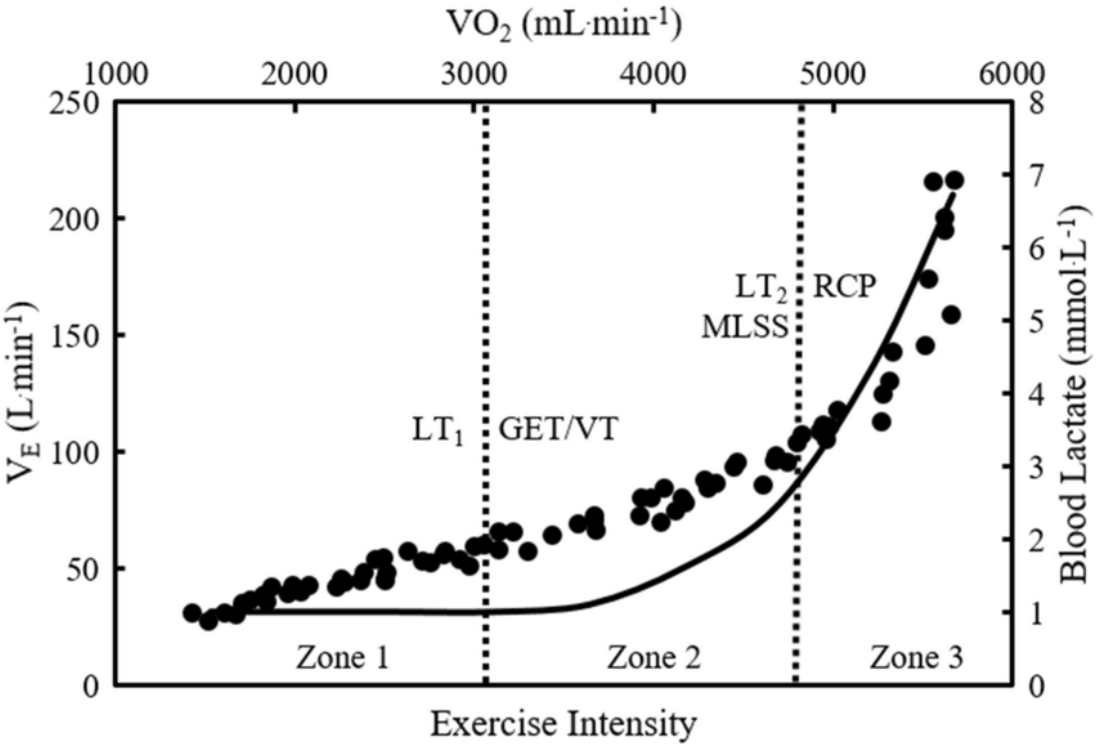
1.1.2 Sustainable percentage of $\dot{V}O_{2max}$

Because intensities of $\dot{V}O_{2max}$ can only be sustained for short periods of time, $\%\dot{V}O_{2max}$ appears to be a much more significant variable, when focusing on longer durations of exercise. As shown by Joyner and Coyle (2008) the spike of blood lactate concentration in trained athletes can be observed at higher values of $\%\dot{V}O_{2max}$. Whilst untrained individuals show strong increases at $60\%\dot{V}O_{2max}$, trained individuals experience the same spike at 75-$90\%\dot{V}O_{2max}$. The difference in intensity at those levels becomes even more notable, when you take the previously stated differences of $\dot{V}O_{2max}$ vales into consideration.

1.1.3 Lactate Threshold

The previously described increase in blood lactate concentration can be associated with rising levels of intensity, as the maximum rate of fat oxidation is no longer able to meet the ATP demands of the muscle contraction. This stimulates glycogenolysis and glycolysis, which is oxidized in the mitochondria as pyruvate. If the rate of pyruvate delivery exceeds the ability of the mitochondria to oxidize pyruvate, an accelerated generation of lactic acid can be measured, which shows in a higher blood lactate concentration (Robergs et al., 2004).

The different stages of energy supply have let to the threshold concept, which plays a key role in physiological profiling. Underneath the lactate threshold (LT1), the blood lactate concentration is essentially equal to baseline, as it remains relatively stable. Above the LT the blood lactate concentrations rise above baseline, whilst toping out at a steady state which depends on the level of intensity. This occurs until the maximum lactate steady state (MLSS) is reached, which is defined as the highest work rate where steady state conditions can be identified. In the past this point has been labelled as the anaerobic threshold (LT2). Intensities at the maximal metabolic steady state (MMSS) can be endured at longer durations, making it a key variable in athletes' performances (Maunder et al., 2021). The black line in Figure 1 shows the rise in blood lactate concentrations.



$\dot{V}_E$ = ventilation, $\dot{V}O_2$ = oxygen uptake, LT = lactate turning point, GET/VT = ventilatory threshold, MLSS = Maximal lactate steady state, RCP = respiratory compensation point

Fig 1: The rise of blood lactate at increasing exercise intensities (Jamnick et al., 2020)

1.1.4 Critical Power

In addition to MMSS, which is a physiological concept, other methods have been brought forward to identify the transition between the heavy and severe exercise intensity domains. Far easier to implement is the critical power model (CP). CP is not directly measured but calculated form a various maximal effort time trials, that stem from different durations.

The groundwork of the CP model was laid in 1925 by Hill. When analysing running and swimming world records, he found a hyperbolic relationship between maximal sustainable speed and time to exhaustion. A similar relationship was reported by Monod and Scherrer (1965), during bouts of repetitive lifting exercises with different isolated muscle groups. CP was then defined as an asymptote of the hyperbolic function, that can be identified as the maximum rate one could sustained for a very long time. As shown by Moritani et al. (1981) CP highly correlates with the ventilatory anaerobic threshold.

Based on the CP model intensities up to the asymptote can theoretically be performed to infinity. In contrast workloads exceeding CP represent a finite work capacity. Exercises at these intensities tap into the sector of W Prime (W'), which can be portrait as a batterie. The higher the workload the faster the energy store is emptied, whilst lower workloads can be sustained for longer durations (Skiba et al. 2012). The CP model with the addition of W' is portrait in Figure 2.

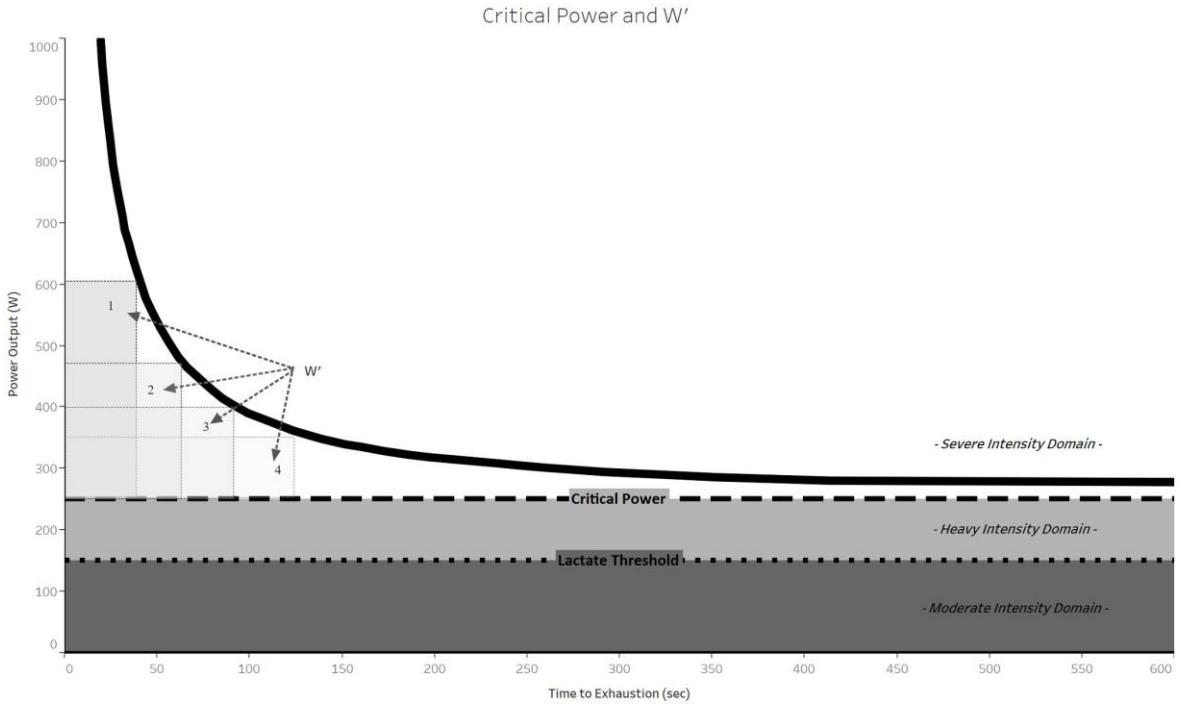
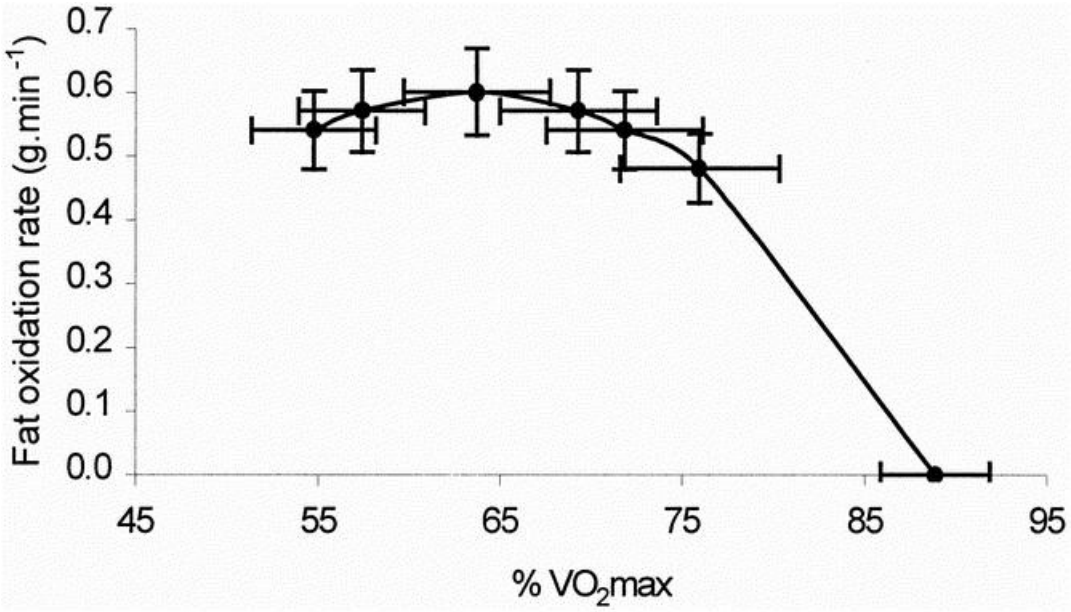


Fig 2: Schematic representation of the CP model (Puchowicz et al., 2018)

1.1.5 Maximal fat oxidation (Fat_{max})

Another frequently used variable in physiological profiling is the rate maximal of fat oxidation (MFO) also known as Fat_{max}. This parameter gives an insight into substrate utilization, which shifts from fat to carbohydrate with increasing intensity (Achten et al., 2002). The basic premise of the importance of MFO relies on the concept of carbohydrate sparing, which would enable an athlete to produce higher workloads in the later stages of competition (Holloszy et al., 1996).

Although MFO is often used in physiological profiling the actual importance of the parameter can be described as controversial. Besides the challenge of reproducibility which stems from different protocols and gas analyzers, multiple studies have found maximal fat oxidation rates between 25 and 85%$\dot{V}O_{2max}$ (Chávez Guevara & Amaro-Gahete, 2025, Romijn et al., 1993). Interestingly, findings by Achten et al. (2002) reinforced the thesis that fat oxidation rates are at a high level trough-out lower intensities before dropping off at workloads above MFO (Figure 3). Therefore, the Fat_{max} zone, which has been defined as the range of exercise intensities with fat oxidation rates within 10% MFO, might be a more practical tool when looking at the larger picture of carbohydrate sparing.



$\%\dot{V}O_2$ = percentage of maximal oxygen uptake

Fig 3: Fat oxidation rates versus exercise intensity expressed as percentage of $\dot{V}O_{2max}$ (Achten et al., 2002)

1.1.6 Mechanical efficiency

Whilst $\dot{V}O_{2max}$ and lactate threshold are used to determine 'Performance $\dot{V}O_2$', efficiency focuses on how much speed or power can be generated at a given level of oxygen consumption. In general, the human body appears to be relatively inefficient, when you compare the vales of energy expenditure and power output (Joyner & Coyle, 2008) examined. In cycling efficiency is commonly described by gross efficiency (GE), which has been shown to differ between 18.5 and 23.5% trained individuals (Coyle et al., 1992).

In 1994 Horowitz et al. suggested that GE could have a large effect on cycling performance in trained athletes. A theoretical model by Olds et al. (1995) showed a 3% improvement in 26-km time trial times. Later, Jeukendrup et al. (2000) calculated that a 1% improvement in GE results in a 48 s improvement in a 40 km time trial time for a 70 kg rider who can sustain an average power of 400 W for 1 h. Similar effects have been reported by Hettinga et al. (2007). Using an energy flow model, the study showed that a change in GE of 0,9% results in a difference of 25.6 s over 20 km.

GE is defined as the ratio between mechanical power output (PO) and the metabolic power input (PI). It can be calculated by a simple formula (Van Ingen et al., 1990). Both PO and PI are displayed in $J \times s^{-1}$. Whilst PO can be converted from different work rate units, PI represents the rate of energy expenditure (EE). Calculations of EE are only valid under steady-state conditions. Furthermore, Bossi et al. (2020) suggest that researchers should share their raw data to allow comparisons between previous and future studies as EE can be calculated by different formulas.

1.2 Critique on physiological profiling

In 2021 a review by Maunder et al. questioned the accuracy of physiological profiling models used in endurance sports. The critic was based on the negligence towards shifts in performance parameters, which have been shown during prolonged exercises. The four most common models of physiological profiling, namely Critical Power (CP), Maximal lactate steady-state (MLSS), Incremental exercise tests (GXT) and Functional threshold power (FTP), lack the parameter of fatigue as they are usually performed in fresh conditions (Table 1).

The fundamental problem with these models lies in the assumption of an indefinite physiological steady-state or homeostasis, which has been disproved by a number of studies dating back to the 1990s. During prolonged exercise changes such as an increase in core and muscle temperature, depletion of endogenous fuel stores, increases in circulating catecholamines, changes in hydration status and changes in endocrine profiles have been observed. To put things simple, it can be said that even work rates at low intensities put physiological stress on the body, which results in fatigue (Maunder et al., 2021).

Table 1: Critique on frequently used profiling models (Maunder et al., 2021)

Model	Outputs sought	Critique
CP	MMSS, W′/D′	Output differs between heavy and severe exercise-intensity domains at constant-load, but does not account for changes that occur over time within long-duration exercise bouts
MLSS	MMSS	Presents several challenges, such as a clear definition for the duration of a steady-state period and a clear turning-point in blood lactate concentration. It requires multiple visits, while being susceptible to errors of individual measurements. Furthermore, MLSS does not account for changes in profiled variables that occur within a long-time duration exercise bouts
GXT	LT, MMSS, $\dot{V}O_{2max}$, W′/D′, Economy, Substrate utilisation	Lacks a clear definition of MMSS and produces large variation in estimates from the same data. It captures many variables in one visit, but does not account for changes in profiled variables over time within long-duration exercise bouts
FTP	MMSS	Is not based on physiological responses and has a poor relationship with laboratory-derived measures. It does not account for changes in profiled variables over time within long-duration exercise bouts

1.2.1 Fatigue

Fatigue is defined as an internal breakdown of homeostasis, caused by an increase in energy production, which is demanded by an external stimulus (Tornero-Aguilera et al., 2022). It plays an important physiological role during exercise by altering one's sensations to adapt exercising strategies (Ament & Verkerke, 2009). It must be acknowledged that every form of physical exercise, regardless of its intensity, will sooner or later deplete the energy stocks of the human body. This could explain why the "sense of effort" increases during prolonged exercise, despite constant motor output (Enoka & Stuart, 1992). Currently the ethology of fatigue is believed to stem from two different branches summarized in central fatigue and peripheral fatigue (Ament & Verkerke, 2009). The central governor theory by Noakes (2007) combines the two models and states that fatigue is the result of central regulation, which happens before any physical failure in the peripheral system can happen.

1.2.1.1 Central Fatigue

Central fatigue is defined as a decrease in the voluntary activation of muscles caused by a deficient drive of motor cortical output (Ament & Verkerke, 2009). It is directly related to a decrease in the frequency and synchronization of motoneurons, and a reduced drive from the motor cortex (Enoka & Stuart, 1992). Recent studies have shown that central fatigue originates directly from the brain, while fatigue-caused changes have also been observed in the spinal cord. The origin of central fatigue although is mainly explained by the biochemistry alterations in the brain.

Serotonin and Dopamine have been shown to impact central fatigue, whilst more recent studies point towards the ratio between the two neurotransmitters. Furthermore, the serotonergic and dopaminergic systems have been shown to interact with each other, which supports this thesis (Cordeiro et al., 2017). Both neurotransmitters cannot pass the blood-brain barrier and must therefore be synthesized in the central nerval system (CNS). Tryptophan enacts as the precursor of Serotonin and uses the same carrier as branched chain amino acids (BCAA) to pass through the blood-brain barrier. During exercise BCAAs are consumed at a higher rate by the skeletal muscle tissue, which leads to an increase of Tryptophan in the CNS (Ament & Verkerke, 2009). When synthesized in the CNS, serotonin negatively effects the influence of the firing motor neuron during prolonged release. This can be explained by the fact that serotonin binds to 5HT1a receptors when 5HT2 receptors are saturated. In contrast to its normal role serotonin now acts as an inhibitor of the firing motor neuron (Tornero-Aguilera et al., 2022). Other effects of a high serotonin concentration in the CNS are a rise in core body temperature and a decline in cutaneous heat loss as well as a lack of motivation and an increased sense of lethargy (Cordeiro et al., 2017).

Dopamine is synthesized from the amino acid tyrosine, which can cross the blood-brain barrier. The limiting factor of the synthesis is believed to be the conversion by tyrosine hydroxylase, which is stimulated by calcium and transforms tyrosine to L-3,4 dihydroxyphenylalanine. In a second step dopamine is created by dopadecarboxylase. The dopaminergic system, which originates from the SNpc and ventral tegmental area (VTA), is activated by an increase in central levels of calcium. Subsequently the activity of tyrosine hydroxylase increases at the beginning of physical exercise. Over longer durations however Serotonin has an inhibitory effect on the Dopamine production. As a result, Dopamine levels decrease, which results in a decline of mechanical efficiency, motor control and the perception of motivation and reward (Cordeiro et al., 2017).

Besides Serotonin and Dopamine other factors such as Glutamate and Gamma-Aminobutyric Acid (GABA) have been suggested to cause central fatigue. Studies have reported a decrease in glutamate transporter-1 (GLT-1) during exercise, which results in an increase of extracellular glutamate concentration. This leads to a declining velocity of the potential action in the axon and results in a loss of the activation of the muscle fiber. Similarly, the inhibitory neurotransmitter GABA is triggered by rising levels of blood lactate and results in the hyperpolarisation of the neuron membrane, making an action potential less likely (Tornero-Aguilera et al., 2022).

1.2.1.2 Peripheral Fatigue

Peripheral fatigue originates from the outside of the central nerval system (CNS) and can be described by a reduction in neuromuscular junction efficiency and biochemical changes in the muscle. As a result, peripheral fatigue leads to a decrease in the contractile strength of muscle fibers (Tornero-Aguilera et al., 2022). Peripheral fatigue strongly correlates with changes in the internal environment often referred to as homeostasis. Therefore, the onset blood lactate accumulation (OBLA), which in sport science is often described as the second lactate turning point (LTP2) or anaerobic threshold, represents an important cause of fatigue. The homeostasis in these conditions is broken by an accumulation of metabolites.

In skeletal muscles the actin-myosin cross-bridge is a result of calcium ion release from the sarcoplasmic reticulum. This event is called excitation-contraction coupling and can be measured by electromyography (EMG). During exercise conduction velocity of action potentials decreases throughout the sarcolemma, which is a cell membrane that surrounds skeletal muscle fibre. The decrease is believed to be a result of exercise-associated biochemical changes in and around the muscle fibres. However, the changes in the EMG show no immediate effect on muscular force. Inside the sarcolemma lies the sarcoplasm and the myofibrils, which are surrounded by the sarcoplasmic reticulum. During exercise

the levels of inorganic phosphate (P_i), hydrogen ions (H^+ ions) and magnesium ions (Mg^{2+} ions) rise in the sarcoplasm. P_i, H^+ ions and Mg^{2+} ions reduce calcium (Ca^{2+}) re-uptake and release from the sarcoplasmic reticulum. This results in a decrease in contractile force due to an inhibition of the cross-bridge interactions, which is triggered by calcium (Ca^{2+}) release from the sarcoplasmic reticulum. In addition, studies have shown an increase of potassium ions (K^+ ions) in the lumen of the t-tubuli, which could block the tubular action potential and decrease excitation-contraction coupling on a second pathway (Ament & Verkerke, 2009).

1.2.1.3 Intensity and Fatigue

Central and peripheral fatigue result in general fatigue also known as neuromuscular fatigue. Together they lead to a decrease in performance, whilst proportion of the two pathways differs depending on the exercise duration and intensity. Studies by Burnley and Jones (2018) and Iannetta et al. (2022) have analysed fatigue at four different intensity domains called moderate, heavy, severe and extreme. Alongside other studies they concluded that fatigue at lower intensities can be associated with central nervous system, whilst higher intensities occur because of peripheral failure. Therefore, there appears to be a shift from central to peripheral fatigue as exercise intensity increases (Ament & Verkerke, 2009). However, findings by Iannetta et al. (2022) suggest, that central fatigue in contrast to peripheral fatigue might not be intensity depended. Still, it is widely acknowledged, that critical power (CP) marks an important turning point in fatigue composition. CP represents the highest point that one can exercise at, without breaking steady-state conditions. It therefore marks the onset of anaerobic energy production, which leads to an accumulation of metabolites in the system (Poole et al., 2016)

Moderate and heavy exercise represent intensity-zones below the CP. The aerobic threshold marks the upper limit of the moderate intensity domain, whilst heavy exercise takes place between the aerobic and anaerobic threshold. Fatigue at moderate intensities remains a largely unexplored territory, as exercise below the aerobic threshold can be sustained for long periods of time before they lead to task failure (Burnley & Jones, 2018). Studies by Davies and Thompson (1986) and Lepers et al. (2002) found an increase of the pulmonary $\dot{V}O_2$ response in the later part of a prolonged running and cycling, which results in an increase of energy expenditure (EE). Together with a 25% drop in maximal voluntary contraction (MVC) these findings suggest peripheral changes as the origin of fatigue. Davies and Thompson (1986) explained this effect by the shift from carbohydrate to fatty acid utilisation during prolonged exercise, which results in a decreased phosphate/oxygen ratio (P/O ratio). However, only 13% of the increase in EE cloud be explained by the change in the respiratory exchange ratio (RER), which records the shift in substrate utilisation. In combination with the fact that the loss of MVC did not result in a change in tetanic force

output Burnley and Jones (2018) argued, that fatigue at moderate intensities might primarily have a central origin.

In heavy exercise domains the $\dot{V}O_2$ response developed at the same magnitude as in moderate workrates, but advanced more rapid. MVC decreased by 25% (Sahlin & Seger., 1995). Burnley and Jones (2018) found a progressive increase in peripheral fatigue, which occurred slower than at higher intensities. The main reason appears to be production of reactive oxygen species, K^+ accumulation and glycogen depletion (Allen et al., 2008). The connection of glycogen depletion and peripheral fatigue has been extensively observed at the beginning of the century, before a more detailed explanation was given by Ørtenblad et al. in 2013. According to their findings the intra-myofibrillar ATP store is the first to deplete during prolonged exercise, leading to excitation–contraction coupling failure. Although central fatigue also develops during heavy exercise the serotonin pathway has been questioned, as an increase in the concentration of the neurotransmitter needs around two hours to occur (Nybo & Secher, 2004). Furthermore, the carrier-hypothesis that links serotonin and BCAA has been questioned, as BCAA-supplementation has not prohibited the effects of central fatigue (Cheuvront et al., 2004).

The severe and extreme intensity domain lies above the CP. In these zones a steady-state cannot be obtained. Severe intensities lead to a $\dot{V}O_2$ response, which results in an attainment of $\dot{V}O_{2max}$. In this case the higher the workload surpasses the CP, the faster the $\dot{V}O_2$ response, which shows that the kinetics (trajectory) of the slow response serves as a better representation of fatigue than the amplitude. As studies have shown peripheral fatigue might even have a direct or indirect relationship with the power-duration-curve, as it develops progressively above the CP. With the loss of contractile function above CP comes the progressive requirement to recruitment additional motor units to maintain the power demand. Eventually the $\dot{V}O_{2max}$ is reached, which marks the point where no further motor units can be added. This inevitably leads to task failure (Burnley et al., 2012). In addition to the peripheral effects, the accumulation of metabolites leads to increasing levels of inhibitory feedback from the central nervous system. Central fatigue is therefore also believed to play an important role as muscle activation is also influenced by the fatigue-induced changes in the shape of the motor unit action potentials (Dideriksen et al., 2011). At extreme intensities however, Iannetta et al. (2022) did not find any signs of central fatigue. Furthermore, there appears to be a difference in the responsible mechanism that leads to peripheral fatigue when compared to server intensities.

1.3 Durability

Based on the effects of fatigue, Maunder et al. (2021) argued, that in addition to the three previously described parameters, a fourth dimension was needed to classify endurance performance. Their hypothesis was supported by finding that showed a decrease in mean maximal power (MMP) efforts after prolonged duration of physical exercise (Mateo-March et al., 2022). Furthermore, Maunder et al. (2021) pointed at past studies that found a decrease in exercise economy during endurance performances and argued that these changes would alter the work rate associated with the MMSS. Therefore, all models that describe this critical point of endurance exercise would lose their accuracy in a fatigued state.

As a solution to this problem, that has so far not been taken into account of physiological profiling, Maunder et al. (2021) presented a fourth parameter called “durability”, which would focus on increasing levels of fatigue and act as a predictor of endurance performances after prolonged physical exercise.

1.3.1 Observation of durability

Ever since Maunder et al. released their review in 2021 there has been a growing interest in the newly presented performance landmark. Smyth et al. (2022) analysed the data of 82,303 runners to investigate the effects of durability on marathon performances and found that both magnitude and onset can be associated with finishing times. Furthermore, they showed a considerable inter-individual variability of durability, which means that the parameter is highly individual and varies between subjects (Figure 4). The study was carried out by decoupling internal and external workload during a marathon. Internal workload was portrayed by the percentage of the maximal heart rate, whilst external workload was described by the running speed relative to critical speed (CS). Decoupling occurred either with an increase of heartrate, a decrease in speed or a mixture of both variants. Subjects who experienced a higher rate of decoupling completed the marathon at a lower percentage of their CS, which resulted in slower finishing times. Smyth et al. (2022) concluded that higher decoupling and therefore lower durability leads to physiological changes caused by fatigue, which leads to a decrease in CS. As a result, the external work rate declines, as speeds above CS cannot be maintained for longer time periods.

Similar findings were presented by Stevenson et al. (2022) who found a decrease of the first ventilatory threshold (VT_1) after prolonged cycling at moderate intensities. Whilst CS describes the anaerobic threshold, VT_1 equates to the aerobic threshold, which underlines the influenceability of both thresholds under fatigued and semi-fatigued conditions. More importantly both studies found the amount of decline differed between the subjects.

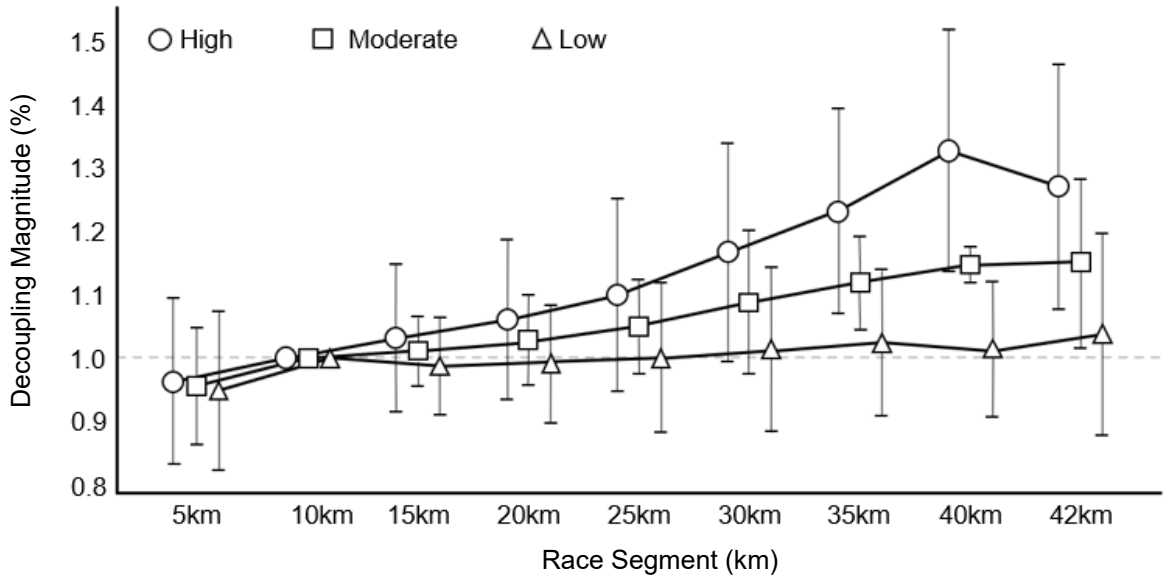


Fig 4: Decoupling of internal-to external workload in different groups (Smyth et al., 2022)

1.3.2 Importance of Durability

With durability appearing to vary between individuals the question was raised, whether it alters performances in elite sport competitions. A study by Muriel et al. (2022) analysed power-data from the Vuelta a España, which is a three-week-long cycling race in Spain. In a fresh state no difference in MMP outputs were found between first division (WorldTour) riders and second division (ProTour) riders, whilst the differences enlarged with increasing amounts of fatigue and race duration. Although race tactics might have influenced the outcome, this study underlines the status as an important performance landmark in endurance sports.

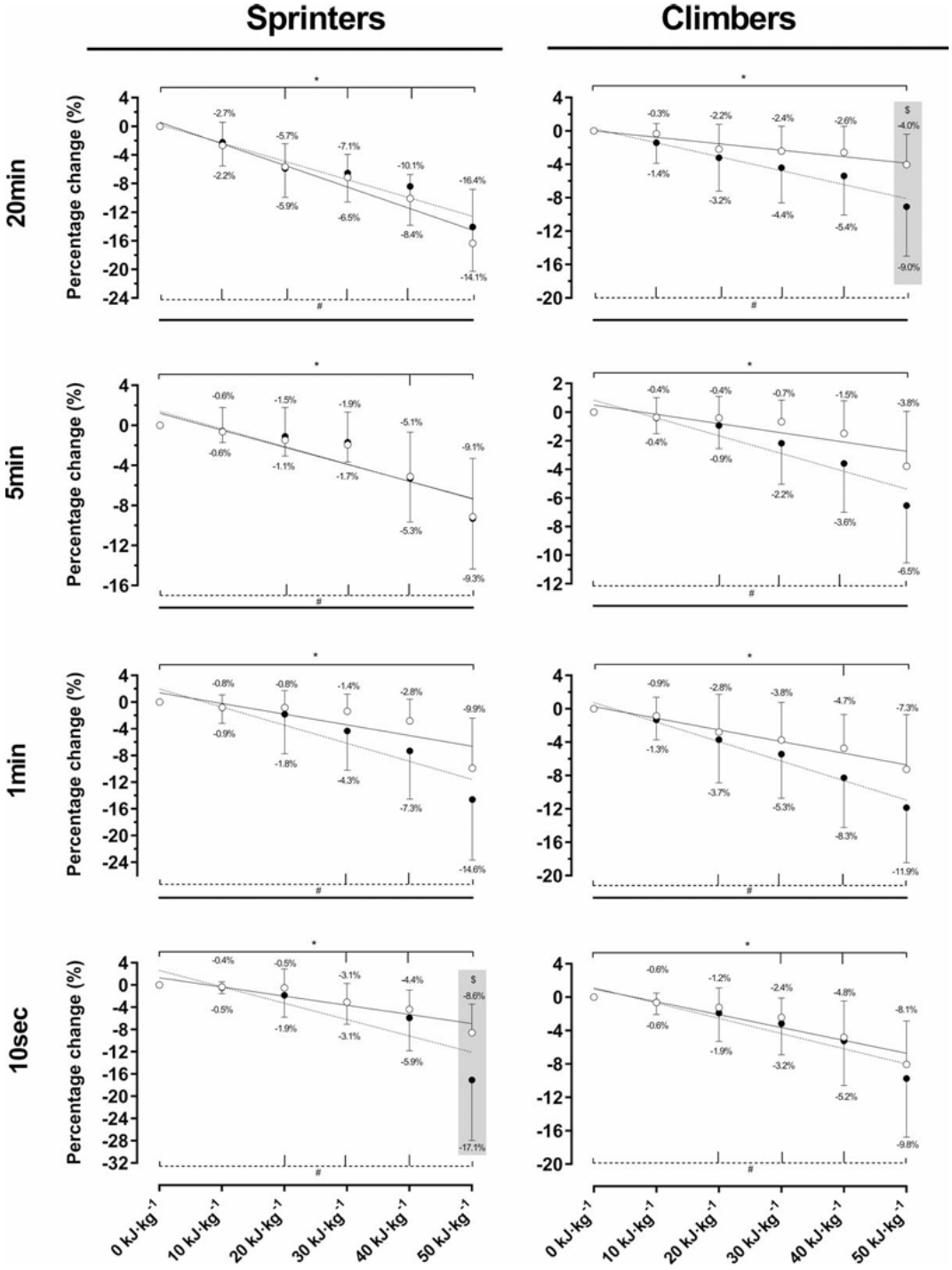
The findings were supported by Van Erp et al., who had already shown in 2021 that more successful climbers and sprinters in professional cycling showed a higher resistance toward fatigue in their respective disciplines (Table 2, Figure 5). Their tests were carried out in a laboratory, which eliminates the limitations of out-door race analyses, although the rate of fatigue was significantly smaller. The importance of durability becomes even more apparent, when considering, that decisive events in cycling usually take place in the latter part of the races.

Table 2: Changes in MMP in CAT.1 and CAT.2 riders (Van Erp et al., 2021)

	Climbers			**Sprinters**		
	CAT.1	CAT.2	CAT.1 vs CAT.2	CAT.1	CAT.2	CAT.1 vs CAT.2
	PO (W/kg)	PO (W/kg)	P	PO (W/kg)	PO (W/kg)	P
10s						
0 kJ/kg	15.93 ± 0.87	15.67 ± 1.46	0.861	18.25 ± 0.99	17.70 ± 1.01	0.336
10 kJ/kg	15.83 ± 0.83	15.58 ± 1.51	0.872	18.19 ± 0.98	17.62 ± 0.96	0.289
20 kJ/kg	15.74 ± 0.93	15.37 ± 1.50	0.722	18.15 ± 0.97	17.37 ± 0.87*	0.064
30 kJ/kg	15.54 ± 0.97*	15.16 ± 1.37*	0.696	17.71 ± 1.00*	17.14 ± 0.96*	0.280
40 kJ/kg	15.17 ±1.14*	14.87 ± 1.21*	0.765	17.48 ± 1.09*	16.63 ± 1.09*	0.097
50 kJ/kg	14.63 ± 0.89*	14.10 ± 1.31*	0.404	16.75 ± 1.20*	14.62 ± 1.65*	<0.001
1min						
0 kJ/kg	10.17 ± 0.64	9.59 ± 0.66	0.068	9.71 ± 0.66	9.51 ± 0.76	>0.99
10 kJ/kg	10.08 ± 0.64	9.47 ± 0.72	0.048	9.63 ± 0.63	9.42 ± 0.71	>0.99
20 kJ/kg	9.87 ± 0.60	9.23 ± 0.71*	0.023	9.62 ± 0.63	9.32 ± 0.68	>0.99
30 kJ/kg	9.77 ± 0.59	9.06 ± 0.64*	0.006	9.57 ± 0.59	9.08 ± 0.68*	0.129
40 kJ/kg	9.67 ± 0.44*	8.78 ± 0.59*	<0.001	9.43 ± 0.69*	8.79 ± 0.72*	0.059
50 kJ/kg	9.40 ± 0.43*	8.44 ± 0.65*	<0.001	8.73 ± 0.79*	8.08 ± 0.71*	0.084
5min						
0 kJ/kg	7.16 ± 0.29	6.86 ± 0.28	0.014	6.16 ± 0.23	6.15 ± 0.35	>0.99
10 kJ/kg	7.13 ± 0.26	6.83 ± 0.29	0.010	6.13 ± 0.26	6.11 ± 0.38	0.999
20 kJ/kg	7.13 ± 0.27	6.80 ± 0.30*	0.005	6.06 ± 0.28	6.08 ± 0.36*	>0.99
30 kJ/kg	7.11 ± 0.28	6.71 ± 0.29*	<0.001	6.03 ± 0.27	6.04 ± 0.37*	>0.99
40 kJ/kg	7.05 ± 0.32	6.61 ± 0.34*	<0.001	5.85 ± 0.37*	5.83 ± 0.46*	0.987
50 kJ/kg	6.88 ± 0.34*	6.41 ± 0.34*	<0.001	5.62 ± 0.45*	5.58 ± 0.53*	0.995
20min						
0 kJ/kg	6.28 ± 0.12	5.99 ± 0.12	<0.001	5.31 ± 0.32	5.28 ± 0.26	0.983
10 kJ/kg	6.26 ± 0.13	5.90 ± 0.17*	<0.001	5.17 ± 0.35*	5.17 ± 0.29*	>0.99
20 kJ/kg	6.14 ± 0.18	5.79 ± 0.27*	<0.001	5.01 ± 0.27*	5.01 ± 0.37*	0.985
30 kJ/kg	6.13 ± 0.17	5.72 ± 0.28*	<0.001	4.93 ± 0.29*	4.94 ± 0.30*	>0.99
40 kJ/kg	6.12 ± 0.18	5.66 ± 0.31*	<0.001	4.78 ± 0.37*	4.84 ± 0.43*	0.968
50 kJ/kg	6.03 ± 0.25*	5.44 ± 0.39*	<0.001	4.46 ± 0.57*	4.54 ± 0.45*	0.960

* Significantly different from MMP measured at 0 kJ/kg (“fresh”)

CAT.1 = more successful, CAT.2 = less successful (climbers and sprinters)



The MMP changes are expressed as a percentage comparison between fresh and fatigued. The Data is graphically expressed as mean and SD, with the mean percentage change also being displayed

*Significantly different from fresh in CAT.1.

#Significantly different from fresh in CAT 2.

$Significantly different between CAT.1 and CAT.2.

Fig 5: The relative decline in MMP with work done in CAT.1and CAT.2 riders (Van Erp et al., 2021)

1.3.2 Durability and training

With the importance of durability outlined in a number of studies the attention of researchers turned to the origin of the newly presented variable. In 2023 Almquist et al. published a study that analysed the development of cycling performance variables in the national norwegian cycling team. They found the biggest difference in the physiological profiling between juniors, U23 and elite athletes in the ability to maintain performance in a fatigued state. Although U23 and elite athletes had spent more energy before a 5 min time trial, they showed a smaller drop of MMP, which indicates a better durability. The study also found a higher GE in elite athletes but concluded that durability could not be explained by the alterations of the rider's efficiency. In contrast, they ascribed improved durability to increased and consistent training loads over several years. In addition, a study by Matomäki et al. (2023), did not find a connection between different training intensities and improvements in durability. These findings are supported by Spragg et al. (2023), who suggested that improved general fitness in itself appears to be the main attributor.

The fact that durability can be altered by training interventions has been shown by Unhjem (2024), who compared the changes in running economy and $\dot{V}O_{2max}$ values between trained runners and active adults after a fatigue protocol. He found a higher decrease in both parameters in active adults and concluded, that these changes might occur due to durability.

1.3.3. Quantifying durability

All the studies that were presented in the previous sections describe physiological changes that occur in fatigued conditions. Still there remains the underlying issue, to present durability in a quantitative value, such as it has been done with the other endurance landmarks.

Usually, durability is portrayed by a decrease of MMP, a shift in threshold and intensity levels or decoupling between external and internal workload. However, these values alone lack the relation to the amount of fatigue, that is being induced by prolonged exercise. Therefore, to compare individual and inter-individual observations of durability a parameter of fatigue is needed, that displays the previously done work. This presents a problem, as fatigue in itself is hardly understood in its entirety. This claim is supported by findings from Smyth et al. (2022) and Gallo et al. (2024), who found fatigue induced loss of performance not to be linear and described a point of onset, that appears to vary between individuals.

Nevertheless, energy expenditure (EE) has been brought forward as a variable to better quantify the observations durability. EE is described by the SI base unit of Joule (J), where

1 J amounts the work of 1 watt being done for 1 second. Due to the high amounts of workload Kilojoule per body weight (kJ/kg) is commonly used in endurance sports to describe the work done by an individual. EE allows to quantify previous work done and represents accumulation levels of fatigue. Mateo-March et al. (2022) showed a drop in MMP values already after 15 kJ/kg which represents only a small margin of EE which exceeds 5000 kJ in longer cycling races (Van Erp et al., 2021).

The connection of durability and EE was further investigated by Van Erp et al. (2021), where they compared the drop in MMP values with increasing amounts in EE. When comparing the higher successful group of CAT 1 climbers the less successful group of CAT 2 climbers, they found increasing differences in MMP decline at higher rates of EE. The difference in 20-minute MMP tests, for example, rose from 1.1% at 10 kJ/kg to 5% after 50 kJ/kg. These findings support the hypothesis, that EE could be a meaningful parameter when quantifying durability.

In contrast Spragg et al. (2024) argued that the actual intensity at which the previous work is done plays a more important role, than just the quantity portrayed by EE. They measured a higher decline in MMP values after shorter-high-intensity-protocol compared to longer-low-intensity-protocol, despite the later ones higher EE (Figure 6). Therefore, EE can be seen as a flawed parameter of durability when comparing two different races, which usually present a huge variety of time in different intensity-zones.

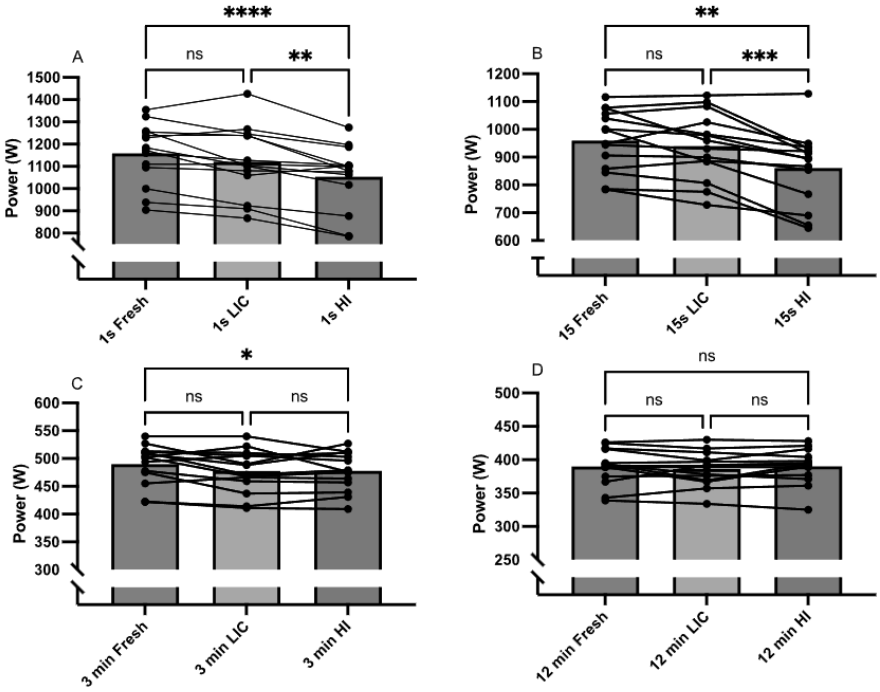


Fig 6: MMP values in fresh state and following low-intensity continuous (LIC) and high-intensity (HI) fatiguing protocols (Spragg et al., 2024)

1.3.4 Durability and laboratory parameters

In addition to quantifying durability in a meaningful manner, the problem of understanding the fundamental physical processes has led to various studies trying to predict the amount of performance loss by using laboratory parameters such as $\dot{V}O_{2max}$ and GE. However, to this date all but one study has failed to demonstrate any association (Ørtenblad et al., 2024).

In 2023 Spragg et al. reported that the decline in CP could be predicted by relative $\dot{V}O_{2max}$, GE and carbohydrate oxidation (CarbOx) or relative $\dot{V}O_{2max}$, GE and fat oxidation (FatOx). Furthermore, they argued that GE in the heavy exercise intensity domain was associated with better durability and found a decrease in GE over time. This led to their hypothesis, that a higher GE baseline may result in a smaller decline over time. As previously mentioned, these findings have not been replicated by Klaris et al. (2024), Ørtenblad et al. (2024) and Gallo et al. (2024).

1.3.5 Durability and substrate utilization

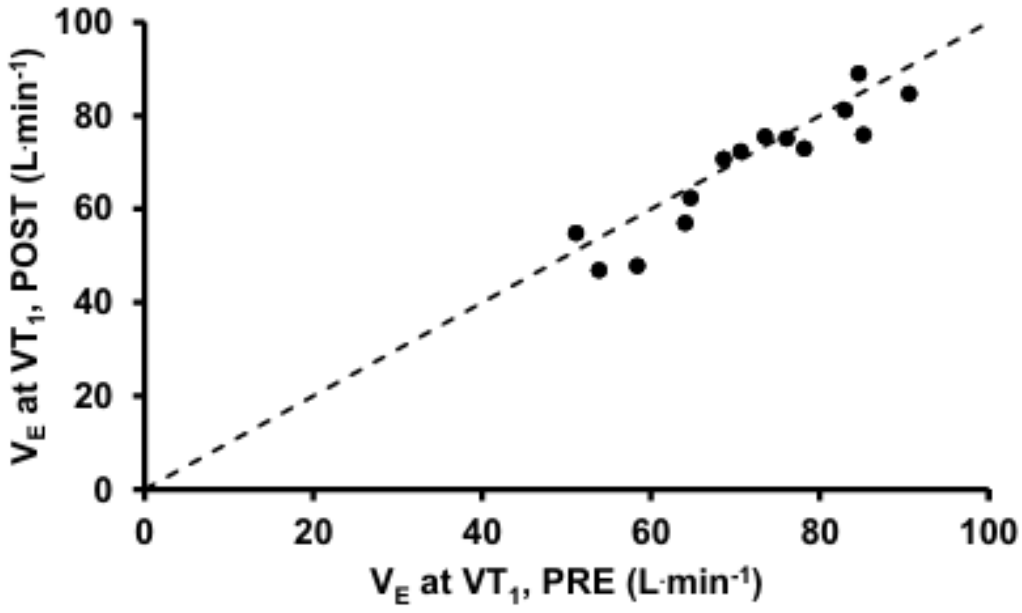
In 2024 Klaris et al. investigated the connection between durability and substrate utilization by taking measurements before and after a six-hour fatigue protocol, which served as a race simulation with a mixture of high and low intensity phases. Although the subjects ingested 90g of carbohydrate per hour the findings revealed an increase in the fat oxidation rate. Furthermore, a high correlation between the maximal fat oxidation rate (MFO) in fasted and fed state (at the beginning of the fatigue protocol) was found, no connection was observed to the MFO after the 6h protocol. These findings suggest that MFO in a rested state may not represent MFO in a fatigued state. In terms of durability the study suggested a tended significant correlation between MFO in a fatigued state and the decline in the MMP of a seven-minute time trial. Due to a poor number of subjects these findings must be verified by future studies.

Gallo et al. (2024) discovered a positive relationship between the durability of the aerobic threshold and the rates of fat oxidation during an initial incremental test. They argued that higher rates of fat oxidation prevented larger glycogen depletion and therefore enhances durability. Interestingly the aerobic threshold maintained its baseline value before decreasing in a non-linear fashion, which could be an indicator for glycogen depletion. Similar to Klaris et al. (2024), they did not attribute MFO to durability but instead pointed at a possible connection.

1.3.6 Monitoring of durability

Beside the challenge of the predicting durability, live monitoring of the new physiological landmark has drawn increasing interest. As durability portrays the shift between external and internal workload, the decoupling of heart rate (HR) has been suggested as a possible form of measurement. However, the HR-based variable DFA-a1 has been the only marker associated with fatigue related aerobic threshold impairment (Nuuttila et al., 2025). DFA-a1 could be a powerful tool in endurance-sports, as it has been shown to display changes in threshold intensities, which could be vital for pacing strategies (Gronwald et al., 2024).

In addition, the rate of ventilation ($\dot{V}_E$) has been brought forward as another potential parameter to track the anaerobic threshold after prolonged exercise. As shown by Stevenson et al. (2024) values of $\dot{V}_E$ remained stable at VT_1 and were not altered by fatigue (Figure 7). However, the practicability of the measuring devices and the day-to-day variation in exercise $\dot{V}_E$ might present a challenge for training implementation.



$\dot{V}_E$ = ventilation, VT_1 = first ventilatory threshold (aerobic threshold)

Fig 7: $\dot{V}_E$ at VT_1 before (PRE) and after (POST) prolonged cycling (Stevenson et al., 2024)

2 Methods

2.1 Design

The study followed a repeated measure design and consisted of three separate laboratory visits and seven test sessions. The test days where split apart by at least 48 hours to minimise the effect of fatigue. The first visit involved a preliminary test to identify performance related landmarks and concluded with a familiarisation of the 5-minute time trial. The second test day focused on actual performance variables in a fresh state, whilst the third visit included a fatigue protocol and a subsequent 5-min TT under fatigued conditions (Table 3).

2.2 Subjects

Passed on a power analysis of a study by Mateo-March et al. (2022) the reduction of MMP during the 5-minute time trial was expected to be around 4%. To obtain a correlation with 80% power, which is seen as the standard in sport science, at least seven subjects were needed. Participants were searched at the Austrian amateur racing scene, with the demand of a second threshold at approximately 5 $W \cdot kg^{-1}$.

The seven subjects had multiple years of training age and regularly participated in cycling races during the last years. On the first testing day a graded exercise test was used to identify performance related landmarks. The study protocol was approved by the Ethics committee of the University of Vienna on 27th of March 2024 and followed the principles as set out in the Declaration of Helsinki (number 01121). The subjects were received all the necessary information about the study and gave consent in written form.

2.3 Laboratory testing

All tests were carried out under laboratory conditions at sea level in a controlled environment (temperature = 20°C–25°C, humidity = 30%–65%). In addition, the subjects were placed in front of a fan to provide extra cooling when desired. They were advised to avoid any exhausting exercise within 24 hours and to restrain form caffeine intake in the last three hours before the tests. Furthermore, they were asked to arrive at the laboratory in a well hydrated state as drinking during the tests was not possible.

All tests were performed on the participant's own road bikes, which they use most for their habitual training. The bikes were mounted on an electromagnetically braked stationary trainer with a 10-Hz sampling rate (Cyclus2; RBM Elektronik-automation GmbH, Leipzig, Germany). The gas exchange of the respiratory gases was measured by a mobile gas analyser (MetaMax 3B, MetaMax 3B-R2, Cortex Biophysik GmbH, Leipzig, Germany). Before, during and after the tests blood lactate samples were taken from the earlobe and

analysed by an automated lactate analyser (Biosen C line, EKF Diagnostics, Barleben, Germany). All equipment was calibrated in advance by using the standard manufacturer-specified protocol. The focus of the testing days are described in Table 3.

Table 3: Study Design

Testing days			
Visit	Test	Main Output	Note
1	graded exercise test	VT1, VT2, MAP, $\dot{V}O_{2max}$	Start at 75 W with an increase of 25 W per Minute
	5min TT	MPO_{5min}	familiarisation
Split by at least 48 hours			
2	6min Baseline Test	GE	at VT1 every three minutes ($[La^-]_b$) was taken, to ensure steady state conditions
	5-second-sprint-test	P_{max}	in cadence-depended-mode with a resistance of 0.13 kp/kg
	5min TT	MPO_{5min}	Actual test in fresh state
Split by at least 48 hours			
3	Fatigue protocol	kJ/kg	Details in Fig 5 and Table 4
	5min TT	MPO_{5min}	Test in fatigued state

2.4 Protocols

The first visit was used for preliminary testing and familiarisation with the 5-minute time trial. After taking a rested blood lactate sample the subjects performed a graded exercise test (GXT) to identify the first and secondary ventilatory threshold (VT1 and VT2), maximal anaerobic power and $\dot{V}O_{2max}$. The initial resistance was set at 75 W with an increase of 25 W every minute. In addition to the analyses of the gas exchange and a heart rate monitor, blood lactate ($[La^-]_b$) samples were taken at the end of each stage. The test was carried out until volitional exhaustion at which point capillary blood was again drawn from the earlobe.

Based on the GXT the first and secondary ventilatory threshold were identified by an analysis of the recorded gas exchange. VT1 was set at the point where the ventilation rate ($\dot{V}E$) increased in contrast to the rate of oxygen consumption ($\dot{V}O_2$), whilst VT2 marked the shift between $\dot{V}E$ and rate of carbon dioxide ($\dot{V}CO_2$) exhalation. Both VT1 and VT2 were checked by the obtained blood lactate concentrations. The $\dot{V}O_{2max}$ was defined as the highest 30-s rolling average throughout the test.

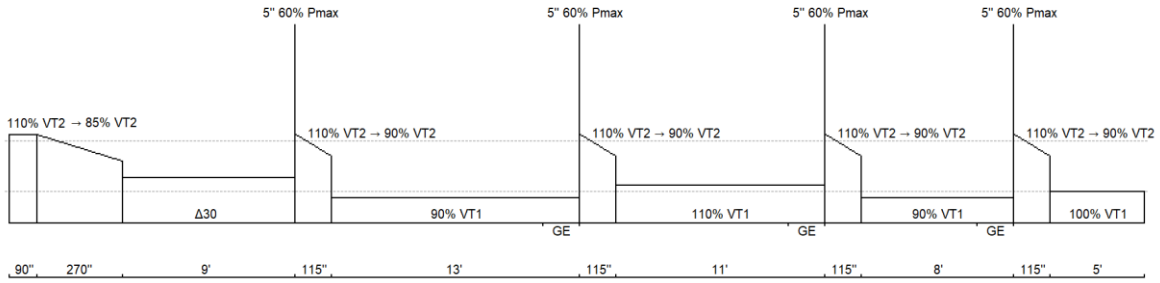
After the GXT subjects were asked to recover while riding at a low workload until $[La^-]_b$ dropped below 4 mmol/L. As soon as the requirement was reached the subjects were asked to perform a maximal 5-minute time trial effort. In order to simulate field-based efforts, the participants were able to manipulate their cadence and gear by using a virtual gear changer mounted to their handlebars. The start of the time trial was initiated by an increase of pedal cadence, whilst the subjects were able to increase power output as a function of cadence and pedal force. After the trial the preferred gear ratio was noted and repeated similar at the next test. The data collected form the test was not taken into account of the study but served as an orientation for the second effort, which was carried out at the second visit.

The second testing day contained three testing sessions. At first rested blood lactate samples were taken, before a trial at a fixed resistance was carried out to measure Gross efficiency (GE) at a fresh state. After a short warm-up at zero load the resistance was set at the power output related VT1 for six minutes. Every three minutes ($[La^-]_b$) was taken to ensure steady-state conditions, whilst GE was calculated from the last two minutes of the session. During the effort the subjects were not allowed to talk and advised to maintain an evenly cadence.

The session was followed up by a 5-second-sprint-test, which focused on the maximal power output (P_{max}). The test was performed in a cadence-depended-mode with a resistance of 0.13 kp/kg. The resistance was applied as soon as the subject exceeded a cadence of 70 rpm. During the sprint the subjects were verbally encouraged and advised to up the increase the cadence as much as possible.

After the sprint-test the subjects were asked to recover before the 5-minute time trial they had already performed on the first visit. A blood lactate concentration below 4 mmol/L was once again used as the key requirement to start the final session. After the settings were fitted toward the participant, the test was completed, whilst being verbally encouraged. During the time trial the gas exchange of the respiratory gases was measured by the previous described equipment. Physical exertion was ensured by comparisons to the $\dot{V}O_{2max}$ obtained in the GXT as well as blood lactate concentrations above 8 mmol/L.

On the third testing day the participants completed a one-hour fatigue protocol (Fig 8, Table 4), that was designed in advance to amount to a total energy expenditure (EE) of roughly 15 kJ/kg. The power output was based on percentage of VT1 and VT2 to provide comparable training loads between the subjects. Although the protocol was programmed using Zwift software (Zwift Inc., California, United States of America), the subjects had no visual control to their avatar. The objective of the fatigue protocol was to mimic common demands of road cycling. The first 90 seconds were performed above the second threshold at 110% VT2, before the intensity declined linearly for the following 4 and a half minutes to 85% VT2. The intense start continued with 9 minutes at Δ30 (VT1+(VT2-VT1) x 0,3) to put more strain on the subjects. After 15 minutes the first of four 2-minute-long intervals above VT2 started. Each of them consisted of a 5 second sprint at 60% of P_{max}, before the resistance decreased for the remaining time (115 s) from 110% to 90% VT2. Between the intervals the subjects completed intensities around VT1 (13 minutes @ 90% VT1, 11 minutes @110% VT1, 8 minutes @ 90% VT1). In these sections GE was calculated during the last two minutes of the low intensity segments to assure steady-state conditions. In order to verify the steady-state conditions blood lactate samples were taken from the earlobe. After the last interval the subjects completed the protocol by exercising for five minutes at VT1 before they repeated the 5-minute time trial in a fatigued state. Once again, the subjects were verbally encouraged during their efforts.



VT_1 = first ventilatory threshold, VT_2 = second ventilatory threshold

Δ30 = VT_1 + (VT_2- VT_1) x 1,3, P_{max} = maximal power output

Fig 8: Fatigue protocol

Table 4: Fatigue protocol

Time (min:s)	Duration	Intensity	Note
00:00–01:30	90 s	110% VT2	
01:30–06:00	270 s	Decreasing from 110% VT2 to 85% VT2	
06:00–15:00	9 min	Δ30 of VT1 to VT2 range	
15:00–15:05	5 s	60% P_{max}	
15:05–17:00	115 s	Decreasing from 110% VT2 to 90% VT2	
17:00–30:00	13 min	90% VT1	GE calculation from last two min
30:00–30:05	5 s	60% P_{max}	
30:05–32:00	115 s	Decreasing from 110% VT2 to 90% VT2	
32:00–43:00	11 min	110% VT1	GE calculation from last two min
43:00–43:05	5 s	60% Pmax	
43:05–45:00	115 s	Decreasing from 110% VT2 to 90% VT2	
45:00–53:00	8 min	90% VT1	GE calculation from last two min
53:00–53:05	5 s	60% P_{max}	
53:05–55:00	115 s	Decreasing from 110% VT2 to 90% VT2	
55:00–60:00	5 min	100% VT1	

immediate start of 5 min TT

2.5 Calculations

In order to analyse gas exchange the mean of every 30 seconds was portrait by one datapoint, which were then exported from the MetaSoft® Studio Software (Cortex Biophysik GmbH, Leipzig, Germany). GE and the fresh 5-min-TT effort were exported with 60 datapoints per minute, whilst GXT and the fatigue protocol were exported by 4 datapoints per minute.

GE was calculated as the ratio between the external load and the total energy expenditure (EE). To calculate total EE the following equation was used:

$EE = (0.550 \times \dot{V}CO_2) + (4.471 \times \dot{V}O_2)$ (Jeukendrup & Wallis, 2005).

The EE of the fatigue protocol was calculated by the equation kJ = (Watt x s)/1000. $\dot{V}O_{2max}$ and mechanical power output based parameters were portrait by absolute and relative values, with the latter being calculated by dividing body weight.

2.6 Statistics

The statistical analysis was performed using IBM SPSS Statistics (IBM, New York, United States of America). The variables of the thresholds (VT1 [w], VT1 [w/kg], VT2 [w], VT2 [w/kg]), maximal Sprint (Pmax [w]), maximal aerobic capacity ($\dot{V}O_{2max}$ [ml/min], $\dot{V}O_{2max}$ [ml/min/kg]), Efficiency (GE fresh [%], GE measurements during the fatigue protocol [%], ΔGE between the measurements [%]) and oxygen uptake kinetics (tau [s]) as well as the 5-min-TT efforts (fresh [w], fresh [w/kg], fatigued [w], fatigued [w/kg]) and its decline (ΔTT [w], ΔTT [w/kg], ΔTT [%]), were tested for normal distribution. The Shapiro–Wilk's significance test ($P > 0.05$) showed a normality in all variables except Pmax [w] and 5min-TT fresh [w/kg]. Correlations between the variables were assessed by using the pearson correlation coefficient. Calculations with Pmax [w] and 5min-TT fresh [w/kg] were performed by using spearman's rank correlation coefficient. The Significance was set at $P < 0.050$. Furthermore, a repeated measures ANOVA was performed, to analyse GE values.

3 Results

3.1 Physiological profile under fresh conditions

Seven subjects (age = 25.9 ± 3.2 yr, body mass = 68.8 ± 3.7 kg, height = 179.1 ± 3.6 cm, body mass index = 21.4 ± 0,9 $kg \cdot m^{-2}$) performed a graded exercise test under fresh conditions in order to identify performance related landmarks ($\dot{V}O_{2max}$ = 67.7 ± 4.6 $mL \cdot kg^{-1}$, VT1 = 2.9 ± 0.2 $W \cdot kg^{-1}$, VT2 = 5 ± 0.3 $W \cdot kg^{-1}$, P_{max} = 1112 ± 128 W). Based on these results an expected EE of 10.9 to 13.3 kJ/kg was calculated during the fatigue protocol. In a fresh state the average 5 min MPO of the subjects was 397 ± 28 W corresponding to 5.8 ± 0.3 $W \cdot kg^{-1}$. The subjects are portrayed in Table 5.

Table 5: Physiological profile of the subjects (n = 7)

	min	max	M ± SD
height [m]	1.72	1.82	1.79 ± 0.3
weight [kg]	64.5	73.7	68.8 ± 4
$\dot{V}O_{2max}$ [ml/min/kg]	64.2	75.0	69.5 ± 4.3
VT1 [W/kg]	2.5	3.1	2.87 ± 0.24
VT2 [W/kg]	4.6	5.4	4.95 ± 0.35
P_{max} [W]	993	1355	1112 ± 138
MPO_{5min} [W]	361	433	397 ± 30
MPO_{5min} [W/kg]	5.09	6.05	5.77 ± 0.33

3.2 Effects of the fatigue protocol

A paired t-test showed significant changes in MPO_{5min} when comparing values from the fresh and fatigued state PO ($t(6) = 5.43$; $p = 0.002$). After the fatigue protocol the subjects MPO_{5min} was reduced to 363 ± 18 W and 5.3 ± 0.3 $W \cdot kg^{-1}$, relative to BM. This implies a decrease in MPO_{5min} vales of 34 ± 15.2 W or 0.5 ± 0.2 $W \cdot kg^{-1}$ and results in a percentual loss of 8.3 ± 3.4% (Table 6). The changes in MPO_{5min} values graphicly displayed in Figure 9.

Table 6: Changes in MPO_{5min} values (n = 7)

	min	max	M ± SD
MPO_{5min} fresh [W]	361	433	397 ± 29
MPO_{5min} fresh [W/kg]	5.1	6.1	5.8 ± 0.3
MPO_{5min} fatigued [W]	338	391	363 ± 20
MPO_{5min} fatigued [W/kg]	4.7	5.7	5.3 ± 0.3
MPO_{5min} delta [W]	-58	-12	-33 ± 16
MPO_{5min} delta [W/kg]	-0.8	-0.2	-0.49 ± 0.22
MPO_{5min} delta [%]	-13.4	-3.3	-8.3 ± 3.7

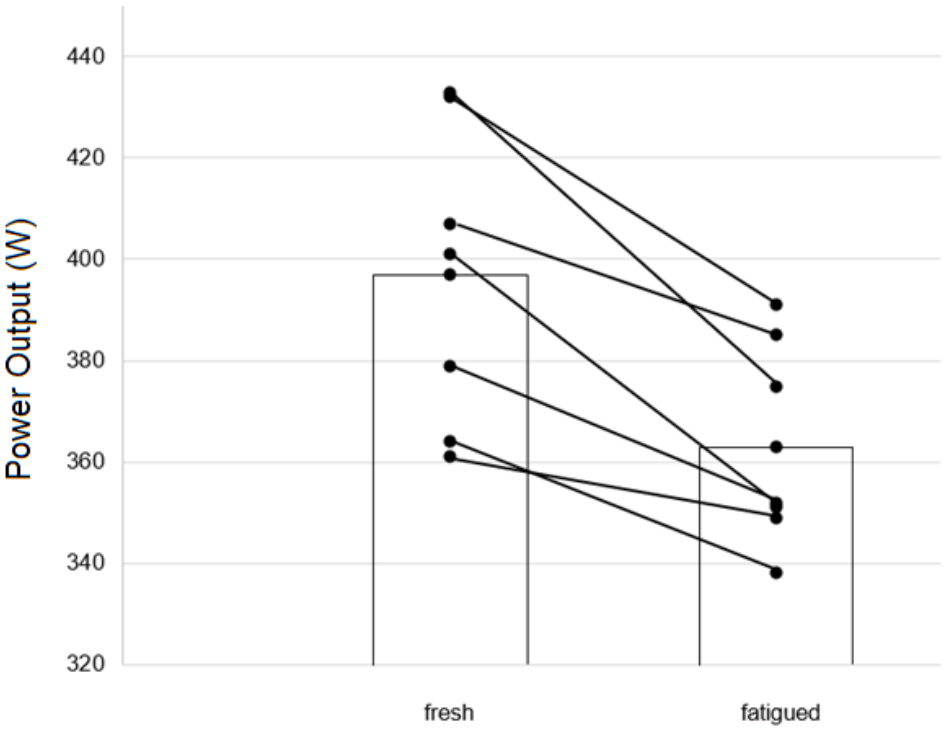


Fig 9: Change of MPO_{5min} after the fatigue protocol

In contrast, the changes of the $\dot{V}O_{2peak}$ values were not statistically significant. These accounts for both absolute values ($t(6) = 2.03$; $p = 0.089$) and values obtained relative to BM ($t(6) = 2.04$; $p = 0.087$). Furthermore, not all subjects showed a decrease in $\dot{V}O_{2max}$ values. On average $\dot{V}O_{2peak}$ decreased by 3.4 ± 4.1 $mL \cdot kg^{-1}$, which indicates a highly individual variation between the subjects. Two subjects increased their $\dot{V}O_{2peak}$ values during the 5 min time trial under fatigued conditions by approximately 1.5 $mL \cdot kg^{-1}$/min. The results are portrait in Table 7 and Figure 10.

Table 7: Changes in physiological landmarks (n = 7)

	min	max	M ± SD
$\dot{V}O_{2peak}$ fresh [ml/min/kg]	64.2	75	69.5 ± 4.3
$\dot{V}O_{2peak}$ fatigued [ml/min/kg]	60.7	70.5	66.1 ± 4.2
$\dot{V}O_{2peak}$ delta [ml/min/kg]	-11.5	1.6	-3.4 ± 4.4
GE [%]	20.7	22.5	21.6 ± 0.6
GE1 [%]	17.3	21.3	20 ± 1.3
GE2 [%]	18	23.8	21.3 ± 1.7
GE3 [%]	17.3	20.5	19.6 ± 1.1
GE delta [%]	-4	-1.1	-2 ± 1

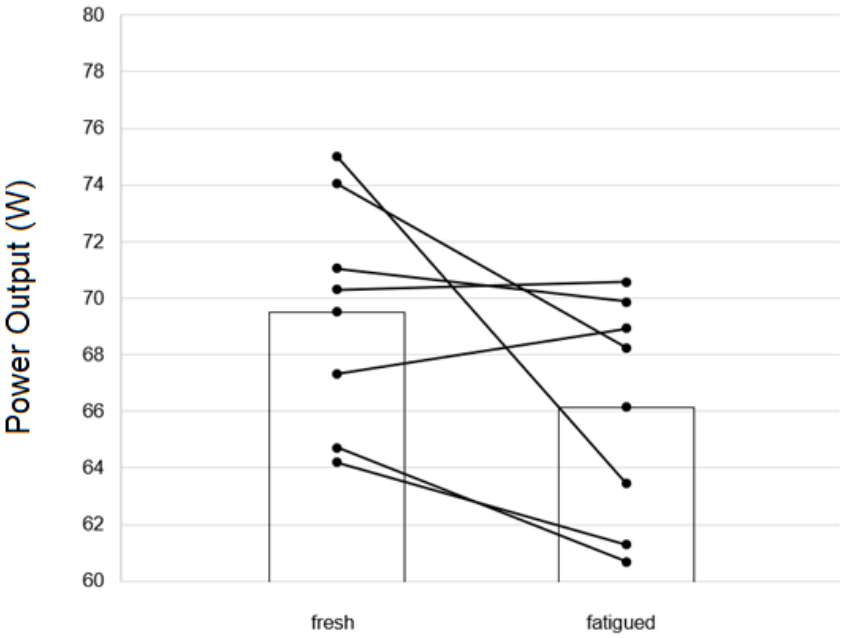
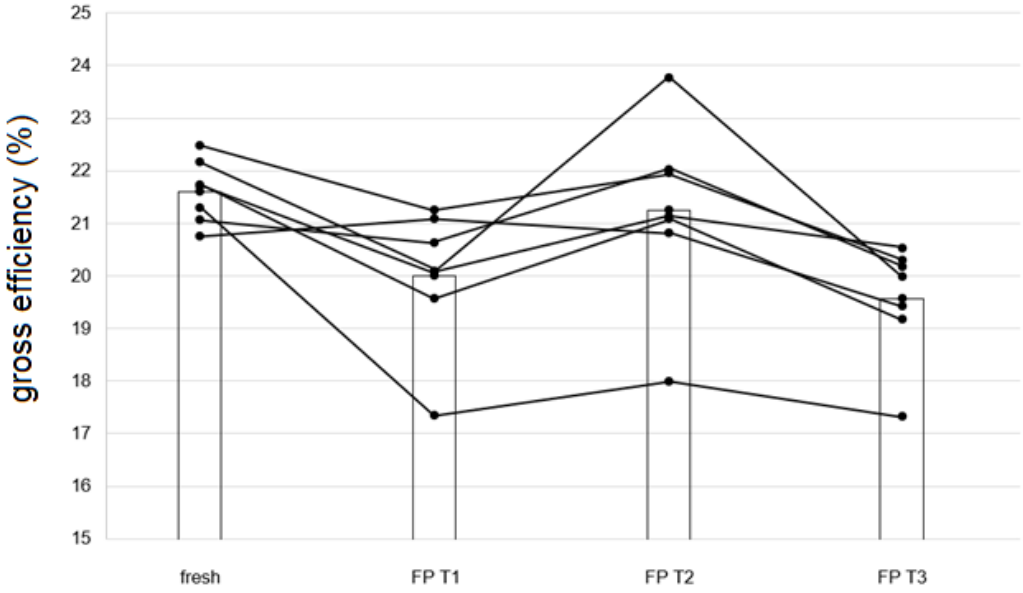


Fig 10: Fatigue-based changes of $\dot{V}O_{2peak}$

A repeated measures ANOVA showed a significant effect on GE caused by the fatigue protocol ($F(3,18) = 9{,}561$, $p < 0{,}001$). These effects were found during baseline GE and the third and last measurement at the end of the protocol (GE3), as well as during the later stages of the fatigue protocol (GE2 and GE3). All but one subject reached their maximal value under fresh conditions in the baseline test at VT1. After the first intense phase of the fatigue protocol the average GE dropped by 1.6 ± 1.3% whilst one subject showed an increase of 0.4%. Interestingly, GE rose during the second measurement during the fatigue protocol by an average of 1.2 ± 1.1% with only one exception, where a small decrease of 0.3% was observed. In the last measurement towards the end of the fatigue protocol all subjects experienced a reduction of GE by an average of 1.7 ± 0.9% (Figure 11). When comparing the fresh GE values with those taken in a fatigued state (roughly after accumulation of 12 kJ/kg) an average decrease of 2.0 ± 0.9% was observed (Table 5).



fresh = GE (gross efficiency) measurements at baseline (VT1)

FP T1 = GE measurements after 28 min of the fatigue protocol at 90% VT1

FP T2 = GE measurements after 41 min of the fatigue protocol at 110% VT1

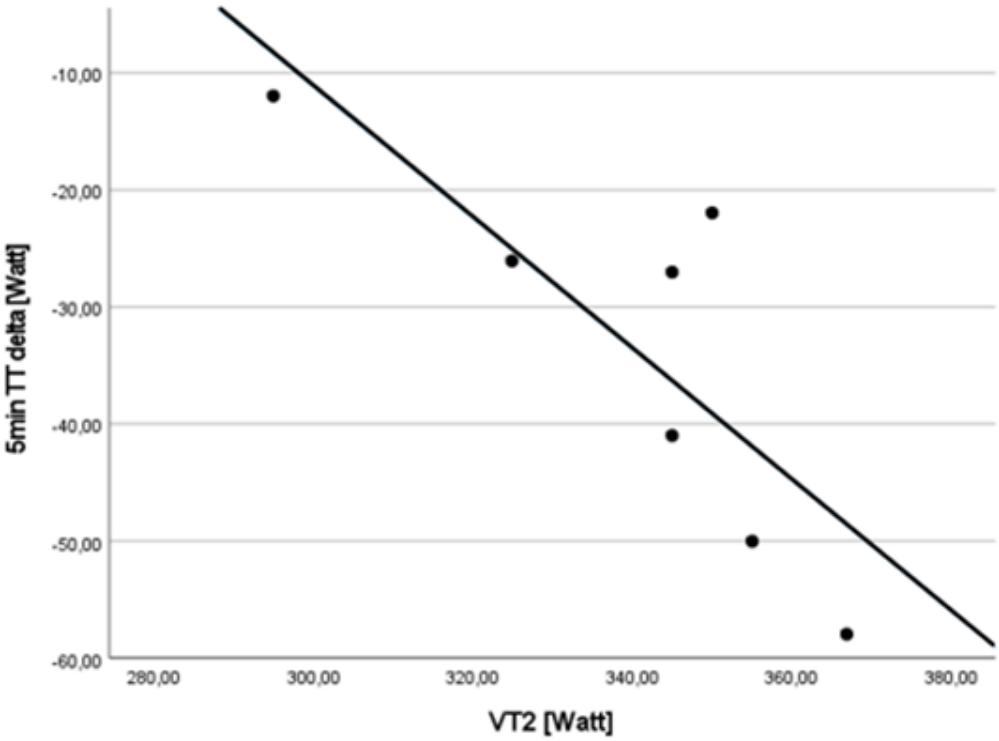
FP T2 = GE measurements after 51 min of the fatigue protocol at 90% VT1

Fig 11: Fatigue-based changes of GE during the fatigue protocol

3.3 Correlations among different physiological markers and fatigue

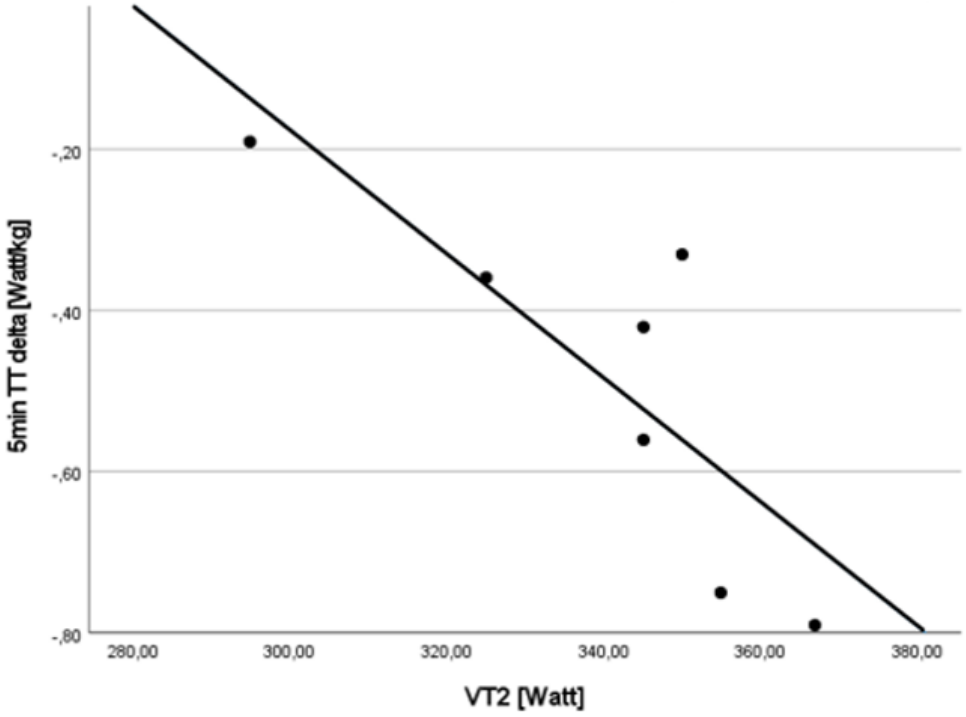
When analysing the relationship between laboratory parameters such as GE, $\dot{V}O_{2max}$ and VT1 no significant correlations were found with the drop in MPO_{5min} values. These results were observed in both absolute and relative values. Absolute PO associated with VT2 showed a significant negative correlation with reduction in MPO_{5min} (Table 8). Absolute (r = -0,806, p = 0,029), relative (r = -0,816, p = 0,025) and percentage vales (r = -0,794, p = 0,033) of the 5 min TT Delta (ΔMPO_{5min}) seem to correlate with absolute VT2 vales (W), which has not been observed at relative VT2 values ($W \cdot kg^{-1}$) (Table 9). Figures 12, 13 and 14 shows the corelations between ΔMPO_{5min} and VT2 values.

measurements after



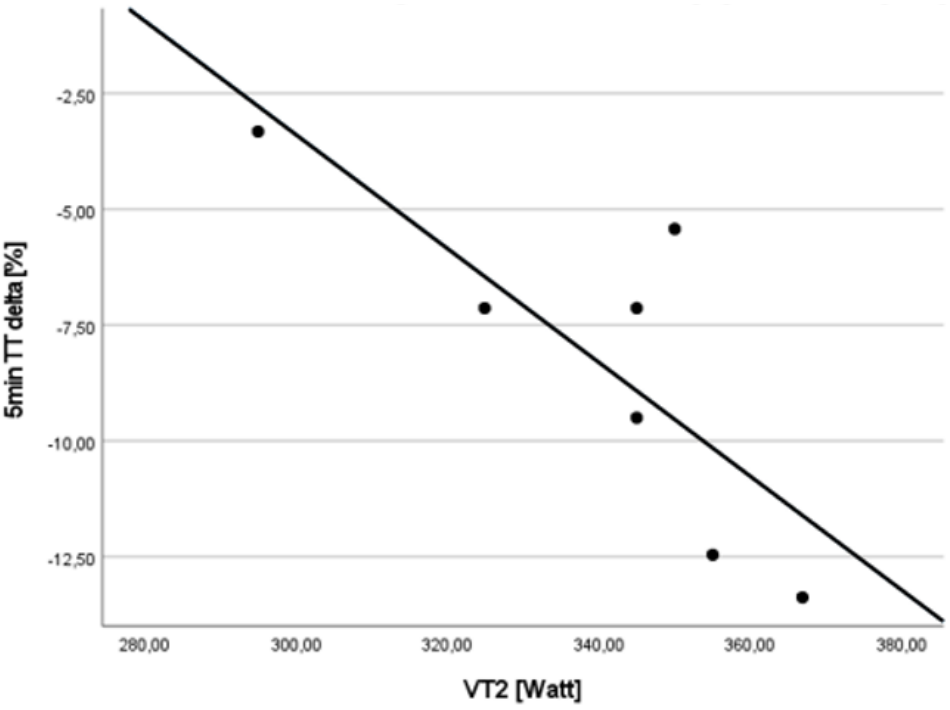
5min TT delta = decline in absolute MPO_{5min}, VT_2 = second ventilatory threshold

Fig 12: Corelations between absolute ΔMPO_{5min} and VT2



5min TT delta = decline in relative MPO_{5min}, VT_2 = second ventilatory threshold

Fig 13: Corelations between relative ΔMPO_{5min} and VT2



5min TT delta = percentual decline MPO_{5min}, VT_2 = second ventilatory threshold

Fig 14: Corelations between percentage ΔMPO_{5min} and VT2

Table 8: Correlations between ΔMPO_{5min} *and fresh physiological landmarks (n = 7)*

		VT1		VT2		$\dot{V}O_{2max}$		GE
		[W]	[W/kg]	[W]	[W/kg]	[ml/min]	[ml/min/kg]	[%]
ΔMPO_{5min} [W]	Pearson's r	-0.417	-0.036	-0.806**	-0.324	-0.448	-0.023	-0.065
	P value	0.352	0.938	0.029	0.478	0.313	0.961	0.890
ΔMPO_{5min} [W/kg]	Pearson's r	-0.397	-.081	-0.816*	-0.414	-0.409	-0.065	-0.092
	P value	0.379	0.864	0.025	0.356	0.362	0.890	0.844
ΔMPO_{5min} [%]	Pearson's r	-0.343	0.011	-0.794*	-0.351	-0.393	0.003	-0.050
	P value	0.452	0.982	0.033	0.441	0.383	-0.050	0.915

* The correlation is significant at the 0.050 level (2-tailed)

** The correlation is significant at the 0.010 level (2-tailed)

Table 9: Correlations between changes in delta values (n = 7)

		ΔGE [%]	$\Delta\dot{V}O_{2max}$ [ml/min/kg]	ΔMPO_{5min} [W]	ΔMPO_{5min} [W/kg]	ΔMPO_{5min} [%]
ΔGE [%]	Pearson's r	1	-0.135	0.113	0.077	0.165
	P value		0.773	0.810	0.869	0.723
$\Delta\dot{V}O_{2max}$ [ml/min/kg]	Pearson's r		1	0.558	0.517	0.528
	P value			0.193	0.235	0.223
ΔMPO_{5min} [W]	Pearson's r			1	0.993**	0.993**
	P value				<0.001	<0.001
ΔMPO_{5min} [W/kg]	Pearson's r				1	0.994**
	P value					<0.001
ΔMPO_{5min} [%]	Pearson's r					1
	P value					

* The correlation is significant at the 0.050 level (2-tailed)
** The correlation is significant at the 0.010 level (2-tailed)

3.4 Correlations between various physiological landmarks

Moreover, both VT1 and VT2 correlated significantly with MPO_{5min} values. Relative VT1 ($W \cdot kg^{-1}$) and relative $\dot{V}O_{2max}$ values ($mL \cdot kg^{-1}$) show a significantly positive correlation ($r = 0,925$, $p = 0,003$). Furthermore, a significant relationship between fatigued GE values and relative VT1 was observed: GE2 ($r = 0,88$, $p = 0,009$), GE3 ($r = 0,926$, $p = 0,003$) and GE delta ($r = 0,813$, $p = 0,026$).

4. Discussion

The main finding of the study was that the drop in MPO of a 5 min time trial is not related to traditional laboratory parameters such as GE, $\dot{V}O_{2max}$ and VT1. These findings are in line with previous studies by Klaris et al. (2024), Ørtenblad et al. (2024) and Gallo et al. (2024). Interestingly a significant inverse correlation between absolute VT2 power values and drop in 5-min TT MPO was found, which could support the hypothesis of Spragg et al., (2023) that aerobic fitness per se is associated with higher durability. However, in contrast to their study similar correlation with VT1 and $\dot{V}O_{2max}$, which are parameters of aerobic fitness as well, were not found.

4.1 Effects of fatigue on time trials

In accordance with previous work MPO values seem to be significantly altered by accumulation of fatigue. The present study found a mean drop of 34 W or 0.5 W·kg^{-1} in MPO_{5min} vales which relates to a decrease of 8.3%. These findings are in line with a study by Mateo-March et al., (2022), who found a significant and progressive decrease in various MPO values (1 s, 1 min, 5 min, 20 min, 60 min and 120 min) after 15 kJ/kg. Similar effects of fatigue on MPO trials are reported by Van Erp et al., (2021), Valenzuela et al., (2022) and Ørtenblad et al., (2024).

Interestingly, the effects of fatigue on MPO values have been found by using different approaches, which further strengthens the premise. Whilst Van Erp et al., (2021) and Mateo-March et al., (2022) carried out a retrospective study by analysing PO data from training and competition, Valenzuela et al., (2022) and Ørtenblad et al., (2024) used a fixed fatigue protocol, that provided similar outcomes. The present study also relied on a fixed fatigue protocol but tapped into different exercise intensity domains. Although the protocols differed between the studies, EE portrait by kJ/kg was used to compare results.

When analysing the fatigue induced loss in MPO_{5min} appears to be highly individual as it varied between 13.4% and 3.3%. However, there seems to be no significant correlation between the magnitude of MPO_{5min} values and the drop in power output. The differences in power output reduction can be assigned to the individuality of durability and have already been described by the previews mentioned studies.

4.2 Effects of fatigue on physiological landmarks

When analysing $\dot{V}O_{2max}$ and GE before and after the fatigue protocol two main findings can be presented. On average, the $\dot{V}O_{2max}$ values in fresh conditions, obtained during the latter stages of 5-min TT, were on average higher by 3.4 mL·kg^{-1}. However, these changes cannot be seen as statistically significant, as the varied between a 1.6 mL·kg^{-1} increase and a 11.5

$mL \cdot kg^{-1}$ decrease across the participants. Notably, two of the seven subjects achieved higher $\dot{V}O_{2max}$ values in the 5-min TT carried out after the fatigue protocol.

These findings are in line with previous work. Ørtenblad et al., (2024) compared $\dot{V}O_{2max}$ values during a 6-min TT and found a 7% decrease. Clark et al., (2018) as well reported a drop in $\dot{V}O_{2max}$ values after two hours of heavy-intensity exercise. However, as a limitation the latter study noted that the $\dot{V}O_{2max}$ values were obtained by two different protocols, by using an GTX in fresh and an end-power test under fatigued conditions. Further research of fatigued induced alterations in $\dot{V}O_{2max}$ values has already been done by Wingo et al., (2005), who stated overheating as a potential explanation, however body core temperature was not measured in the current study. As mentioned previously, neither $\dot{V}O_{2max}$ nor its alteration caused by the fatigue protocol showed a correlation with the loss of the 5-min TT power output.

In the present study all subjects experienced a drop in GE, when comparing baseline values in fresh conditions with those taken in the later stages of the fatigue protocol. Although a repeated measures ANOVA showed that GE was significantly altered by fatigue, the changes however do not correlate with the changes in the MPO_{5min}. Another interesting finding was the influence of power-output on GE. These findings are in line with studies by Samozino et al., (2006) and Leirdal and Ettema (2009), who found an increase in efficiency with increasing workrates. In the present study baseline GE was calculated from VT1 values, whilst GE measurements during the fatigue protocol were taken from workrates at 90 and 110% of VT1. All but one subjects experienced a drop in GE when comparing baseline values to the first measurement during the fatigue protocol, which was taken at 90% VT1 after a period of partly highly intense work above VT2. Whether this drop was caused by the 10% lower workrate or the fatigue induced decline of GE can therefore only be hypothesised. The theory that different workrates might have influenced the results can be supported by the fact that GE values increased in all but one subject during the second measurement, which was taken at 110% VT1. When comparing the GE values taken from the first and last measurement of the fatigue protocol, which were performed at the same work rate of 90% VT1 an average decline of 0.4% can be noted. However, the two measurements were taken at an interval of 21 min, where roughly a third of the total kJ accumulation took place.

Previous work by Spragg et al., (2023) found a relation between higher GE baseline values and increased durability and argued, that a higher baseline may also result in a smaller decline of GE over time. Based on this recent study the first claim could not be replicated as no significant connection between baseline GE and the decrease in MPO_{5min} was found. Furthermore, the four separate GE measurements do not support the hypothesis that a

higher GE might be associated with a smaller decrease over time. In fact, the subject with the lowest GE initial value experienced the smallest loss of efficiency.

4.3 Limitations

In this section some limitations of the present study should be acknowledged, that may have affected to previously mentioned outcomes. The most influential decision about the study design can be assumed to be the lack of fuelling and hydration that the probands have experienced during the fatigue protocol. Due to interim GE measurements the subjects were advised to retain their respiratory mask throughout the entire testing phase. This sparks valid criticism as durability and physical performance in general is heavily influenced by carbohydrate ingestion in order to maintain carbohydrate availability (Peeters et al., 2025, Alghannam et al., 2016). Therefore, the emptying of the carbohydrate stores might have influenced the magnitude of MPO_{5min}. The same can be assumed for the lack of hydration.

It can also be questioned if the fatigue protocol served as a valid race simulation. First of all, the duration of one hour might be too short to generate novel knowledge about durability, that could actually be used in day-to-day training and racing. As shown by Van Erp et al. (2021) the growing decrease in MPO vales appears to relate to the magnitude of previous done workload. The subjects of the present study were given a total workload of around 15 kJ/kg, which sits at the lower end of the spectrum, when observing the effects of durability. Secondly, although the present fatigue protocol was performed as a mixture of different intensities, it can be questioned if it mimics the demands of road cycling in an adequate way.

4.4 Novel insights into durability

When reviewing previous work in the field of durability the problem of quantifying the newly promoted landmark of endurance sports becomes apparent. Durability represents one's ability to counteract fatigue and shows itself in comparison with values obtained in a fresh or unfatigued state. Different study designs therefore only represent a small portion of the phenomenon. In other words, the point of view matters.

As of now, durability serves as a collective term that describes observable effects of fatigue. These include alterations in threshold concepts, drifts in physiological landmarks, loss of efficiency and the decline in MPO. This presents a problem with validity as a collective term might not be suited to describe a specific measurement. As an example, it could be hypothesized that fatigue might have a different effect on the aerobic threshold compared to the anaerobic threshold. In this case durability needs to be separated in two different branches in order to identify training adaptations. The problem can also be displayed by the

present study, which begs the question if changes in MPO values correlate with changes in threshold concepts such as CP. Therefore, the meaningfulness of these alterations in daily training or competing becomes questionable. Furthermore, it should be noted that the reliability of durability represents another unknown factor.

Besides the fact that durability lacks a clear definition, the protocols to determine its effects also vary to a great degree. Most studies under laboratory conditions have looked at the effect of fatigue caused by prolonged exercise at constant work rates at the moderate and heavy intensity domain. These long-duration protocols at steady-state conditions have been shown to lead to alterations in the moderate and heavy intensity transition (Stevenson et al. 2022, Gallo et al., 2024) and influence MPO values up to the severe intensity domain (Valenzuela et al., 2022; Ørtenblad et al., 2024). Noticeably, fewer studies have chosen fatigue protocols that tap into the servere intensity domain (Spragg et al., 2023; Klaris et al., 2024). This is surprising when considering the nature of fatigue. As described in the introduction CP represents an important turning point in fatigue composition as it represents the transition from the aerobic to the anaerobic metabolism (Poole et al., 2016). Interestingly, both protocols have led to the conclusion that laboratory parameters cannot predict durability, which has only been contradicted by Spragg et al., (2023). The present study, which had an anaerobic portion that should not be neglected, is in line with most of the previous done work.

Although both study designs have presented similar findings, the importance of CP on fatigue and therefore durability should not be underestimated. Klaris et al. (2024) reported that both accumulated work (kJ/kg) and time above lactate threshold showed an influence on fatigue. These assumptions are in line with findings by Spragg et al. (2024), who found that intensity seems to be the bigger contributor. Based on the present study these findings cannot be verified but 5-minute MPO vales on average declined by 0.5 $W \cdot kg^{-1}$ (8%) after accumulation of 12,4 kJ/kg mechanical work.

To gather a better understanding on durability future studies should look deeper into the effects of intensity on fatigue. Currently durability serves as a description of an observable phenomenon, which arises the question whether different studies can be compared in a meaningful way.

5. Conclusion and practical application for coaches and practitioners

The present study highlights the individuality of fatigue resistance. Both coaches and athletes should be encouraged to look at the bigger picture of performance, rather than only focusing on metrics that only represent one's ability in fresh conditions. It should therefore always be a part of physiological profiling, as it can make the difference between winning and losing. Furthermore, it can be hypothesised, that the importance of durability rises with increasing levels of fatigue, making it the key variable of endurance sports. Athletes therefore should not be blinded by their abilities in fresh state.

With the importance of durability clarified, its trainability and testability come into focus. Coaches are asked to construct training program targeting improvements in this subject. These might implement specific training sessions in a semi- or completely fatigued state, that would otherwise be described in fresh conditions. As workloads might differ, finding the right intensity at different levels of fatigue presents a challenging task. Furthermore, an appropriate testing battery needs to be implemented to show improvements in durability.

Another practical application of durability can be suggested in race strategies. Athletes with high fatigue resistance can use this ability to their advantage by making races harder. This might turn the race into their favour as shortcomings in their fresh-physiological profile could be made up for.

Lastly, it can be concluded that physiological profiles in a steady state might not be meaningful in scouting, as durability might be overlooked. Once ability in actual races can be deemed more important than their diagnostical output. Finally, it should be once again highlighted that durability cannot be predict by laboratory parameters in a fresh state.

Literature

Achten, J., Gleeson, M., & Jeukendrup, A. E. (2002). Determination of the exercise intensity that elicits maximal fat oxidation. *Medicine and science in sports and exercise*, *34*(1), 92–97. https://doi.org/10.1097/00005768-200201000-00015

Alghannam, A. F., Jedrzejewski, D., Tweddle, M. G., Gribble, H., Bilzon, J., Thompson, D., Tsintzas, K., & Betts, J. A. (2016). Impact of Muscle Glycogen Availability on the Capacity for Repeated Exercise in Man. *Medicine and science in sports and exercise*, *48*(1), 123–131. https://doi.org/10.1249/MSS.0000000000000737

Almquist, N. W., Hansen, J., & Rønnestad, B. R. (2023). Development of Cycling Performance Variables and Durability in Female and Male National Team Cyclists: From Junior to Senior. *Medicine and science in sports and exercise*, *55*(11), 2053–2063. https://doi.org/10.1249/MSS.0000000000003232

Allen, D. G., Lamb, G. D., & Westerblad, H. (2008). Skeletal muscle fatigue: cellular mechanisms. *Physiological reviews*, *88*(1), 287–332. https://doi.org/10.1152/physrev.00015.2007

Ament, W., & Verkerke, G. J. (2009). Exercise and fatigue. *Sports medicine (Auckland, N.Z.)*, *39*(5), 389–422. https://doi.org/10.2165/00007256-200939050-00005

Ansdell, P., Thomas, K., Hicks, K. M., Hunter, S. K., Howatson, G., & Goodall, S. (2020). Physiological sex differences affect the integrative response toexercise: Acuteandchronicimplications.ExperimentalPhysiology,105, 2007–2021. https://doi.org/10.1113/EP088548

Bassett, D. R., Jr, & Howley, E. T. (2000). Limiting factors for maximum oxygen uptake and determinants of endurance performance. *Medicine and science in sports and exercise, 32*(1), 70–84. https://doi.org/10.1097/00005768-200001000-00012

Bossi, A. H., Timmerman, W. P., & Hopker, J. G. (2020). Energy Expenditure Equation Choice: Effects on Cycling Efficiency and its Reliability. *International journal of sports physiology and performance*, *15*(2), 288–291. https://doi.org/10.1123/ijspp.2018-0818

Bouchard, C., An, P., Rice, T., Skinner, J. S., Wilmore, J. H., Gagnon, J., Pérusse, L., Leon, A. S., & Rao, D. C. (1999). Familial aggregation of VO(2max) response to exercise training: results from the HERITAGE Family Study. *Journal of applied physiology (Bethesda, Md. : 1985)*, *87*(3), 1003–1008. https://doi.org/10.1152/jappl.1999.87.3.1003

Burnley, M., & Jones, A. M. (2018). Power-duration relationship: Physiology, fatigue, and the limits of human performance. *European journal of sport science, 18*(1), 1–12. https://doi.org/10.1080/17461391.2016.1249524

Burnley, M., Vanhatalo, A., & Jones, A. M. (2012). Distinct profiles of neuromuscular fatigue during muscle contractions below and above the critical torque in humans. *Journal of applied physiology (Bethesda, Md. : 1985)*, *113*(2), 215–223. https://doi.org/10.1152/japplphysiol.00022.2012

Chávez Guevara, I. A., & Amaro-Gahete, F. J. (2025). Methodological issues related to maximal fat oxidation and FATmax reproducibility: a narrative review. *International journal of obesity (2005)*, 10.1038/s41366-025-01861-y. Advance online publication. https://doi.org/10.1038/s41366-025-01861-y

Cheuvront, S. N., Carter, R., 3rd, Kolka, M. A., Lieberman, H. R., Kellogg, M. D., & Sawka, M. N. (2004). Branched-chain amino acid supplementation and human performance when hypohydrated in the heat. *Journal of applied physiology (Bethesda, Md. : 1985)*, *97*(4), 1275–1282. https://doi.org/10.1152/japplphysiol.00357.2004

Clark, I. E., Vanhatalo, A., Bailey, S. J., Wylie, L. J., Kirby, B. S., Wilkins, B. W., & Jones, A. M. (2018). Effects of Two Hours of Heavy-Intensity Exercise on the Power-Duration Relationship. *Medicine and science in sports and exercise*, *50*(8), 1658–1668. https://doi.org/10.1249/MSS.0000000000001601

Cordeiro, L. M. S., Rabelo, P. C. R., Moraes, M. M., Teixeira-Coelho, F., Coimbra, C. C., Wanner, S. P., & Soares, D. D. (2017). Physical exercise-induced fatigue: the role of serotonergic and dopaminergic systems. *Brazilian journal of medical and biological research = Revista brasileira de pesquisas medicas e biologicas*, *50*(12), e6432. https://doi.org/10.1590/1414-431X20176432

Coyle, E. F., Sidossis, L. S., Horowitz, J. F., & Beltz, J. D. (1992). Cycling efficiency is related to the percentage of type I muscle fibers. *Medicine and science in sports and exercise*, *24*(7), 782–788.

Davies, C. T., & Thompson, M. W. (1986). Physiological responses to prolonged exercise in ultramarathon athletes. *Journal of applied physiology (Bethesda, Md. : 1985)*, *61*(2), 611–617. https://doi.org/10.1152/jappl.1986.61.2.611

Díaz, J. J., Fernández-Ozcorta, E. J., & Santos-Concejero, J. (2018). The influence of pacing strategy on marathon world records. *European journal of sport science*, *18*(6), 781–786. https://doi.org/10.1080/17461391.2018.1450899

Dideriksen, J. L., Enoka, R. M., & Farina, D. (2011). Neuromuscular adjustments that constrain submaximal EMG amplitude at task failure of sustained isometric contractions. *Journal of applied physiology (Bethesda, Md. : 1985)*, *111*(2), 485–494. https://doi.org/10.1152/japplphysiol.00186.2011

Enoka, R. M., & Stuart, D. G. (1992). Neurobiology of muscle fatigue. *Journal of applied physiology (Bethesda, Md. : 1985)*, *72*(5), 1631–1648. https://doi.org/10.1152/jappl.1992.72.5.1631

Fernandes, G. D., & Maldonado, V. (2024). In memory of Kelvin Kiptum: a reflection on his record-breaking marathon and the future outlook for a sub 2-h race from a drafting perspective. *European journal of applied physiology*, *124*(8), 2379–2388. https://doi.org/10.1007/s00421-024-05458-7

Gallo, G., Faelli, E. L., Ruggeri, P., Filipas, L., Codella, R., Plews, D. J., & Maunder, E. (2024). Power output at the moderate-to-heavy intensity transition decreases in a non-linear fashion during prolonged exercise. *European journal of applied physiology*, *124*(8), 2353–2364. https://doi.org/10.1007/s00421-024-05440-3

Gronwald, T., Horn, L., Schaffarczyk, M., & Hoos, O. (2024). Correlation properties of heart rate variability for exercise prescription during prolonged running at constant speeds: A randomized cross-over trial. *European journal of sport science*, *24*(11), 1539–1551. https://doi.org/10.1002/ejsc.12175

Hettinga, F. J., De Koning, J. J., de Vrijer, A., Wüst, R. C., Daanen, H. A., & Foster, C. (2007). The effect of ambient temperature on gross-efficiency in cycling. *European journal of applied physiology*, *101*(4), 465–471. https://doi.org/10.1007/s00421-007-0519-3

Hill, A. V. (1925). The Physiological Basis of Athletic Records. *The Scientific Monthly*, *21*(4), 409–428.

Holloszy, J. O., & Kohrt, W. M. (1996). Regulation of carbohydrate and fat metabolism during and after exercise. *Annual review of nutrition*, *16*, 121–138. https://doi.org/10.1146/annurev.nu.16.070196.001005

Horowitz, J. F., Sidossis, L. S., & Coyle, E. F. (1994). High efficiency of type I muscle fibers improves performance. *International journal of sports medicine*, *15*(3), 152–157. https://doi.org/10.1055/s-2007-1021038

Iannetta, D., Zhang, J., Murias, J. M., & Aboodarda, S. J. (2022). Neuromuscular and perceptual mechanisms of fatigue accompanying task failure in response to moderate-, heavy-, severe-, and extreme-intensity cycling. *Journal of applied physiology (Bethesda, Md. : 1985)*, *133*(2), 323–334. https://doi.org/10.1152/japplphysiol.00764.2021

Jamnick, N. A., Pettitt, R. W., Granata, C., Pyne, D. B., & Bishop, D. J. (2020). An Examination and Critique of Current Methods to Determine Exercise Intensity. *Sports medicine (Auckland, N.Z.)*, *50*(10), 1729–1756. https://doi.org/10.1007/s40279-020-01322-8

Jeukendrup, A. E., Craig, N. P., & Hawley, J. A. (2000). The bioenergetics of World Class Cycling. *Journal of science and medicine in sport*, *3*(4), 414–433. https://doi.org/10.1016/s1440-2440(00)80008-0

Jeukendrup, A. E., & Wallis, G. A. (2005). Measurement of substrate oxidation during exercise by means of gas exchange measurements. *International journal of sports medicine, 26* Suppl 1, 28–37. https://doi.org/10.1055/s-2004-830512

Joyner M. J. (1991). Modeling: optimal marathon performance on the basis of physiological factors. *Journal of applied physiology (Bethesda, Md. : 1985)*, *70*(2), 683–687. https://doi.org/10.1152/jappl.1991.70.2.683

Joyner, M. J., & Coyle, E. F. (2008). Endurance exercise performance: the physiology of champions. *The Journal of physiology*, *586*(1), 35–44. https://doi.org/10.1113/jphysiol.2007.143834

Klaris, M. B., Cubel, C., Bruun, T. R., Stampe, D., Rørvik, S., Fischer, M., Bonne, T., Christensen, P. M., Piil, J. F., & Nybo, L. (2024). Performance and Fatigue Patterns in Elite Cyclists During 6 h of Simulated Road Racing. *Scandinavian journal of medicine & science in sports*, *34*(7), e14699. https://doi.org/10.1111/sms.14699

Leirdal, S., & Ettema, G. (2009). Freely chosen pedal rate during free cycling on a roller and ergometer cycling. *European journal of applied physiology*, *106*(6), 799–805. https://doi.org/10.1007/s00421-009-1087-5

Lepers, R., Maffiuletti, N. A., Rochette, L., Brugniaux, J., & Millet, G. Y. (2002). Neuromuscular fatigue during a long-duration cycling exercise. *Journal of applied*

physiology (Bethesda, Md. : 1985), *92*(4), 1487–1493. https://doi.org/10.1152/japplphysiol.00880.2001

Mateo-March, M., Valenzuela, P. L., Muriel, X., Gandia-Soriano, A., Zabala, M., Lucia, A., Pallares, J. G., & Barranco-Gil, D. (2022). The Record Power Profile of Male Professional Cyclists: Fatigue Matters. *International journal of sports physiology and performance*, *17*(6), 926–931. https://doi.org/10.1123/ijspp.2021-0403

Matomäki, P., Heinonen, O. J., Nummela, A., Laukkanen, J., Auvinen, E. P., Pirkola, L., & Kyröläinen, H. (2023). Durability is improved by both low and high intensity endurance training. *Frontiers in physiology*, *14*, 1128111. https://doi.org/10.3389/fphys.2023.1128111

Maunder, E., Seiler, S., Mildenhall, M. J., Kilding, A. E., & Plews, D. J. (2021). The Importance of 'Durability' in the Physiological Profiling of Endurance Athletes. Sports medicine (Auckland, N.Z.), 51(8), 1619–1628. https://doi.org/10.1007/s40279-021-01459-0

McGuire, D. K, Levine, B. D, Williamson, J. W, Snell, P. G, Blomqvist, C. G, Saltin, B, & Mitchell, J. H (2001). A 30-year follow-up of the dallas bed rest and training study: II. Effect of age on cardiovascular adaptation to exercise training. Circulation, 104(12), 1358–1366. https://doi.org/10. 1161/hc3701.096099

Meyler, S., Bottoms, L., & Muniz-Pumares, D. (2021). Biological and methodological factors affecting V̇O2max response variability to endurance training and the influence of exercise intensity prescription. *Experimental physiology*, *106*(7), 1410–1424. https://doi.org/10.1113/EP089565

Monod, H., & Scherrer, J. (1965). The work capacity of a synergic muscle group. *Ergonomics, 8*(3), 329–338. https://doi.org/10.1080/00140136508930810

Montero, D., & Diaz-Canestro, C. (2019). Skeletal Muscle O_2 Diffusion and the Limitation of Aerobic Capacity in Heart Failure: A Clarification. *Frontiers in cardiovascular medicine*, *6*, 78. https://doi.org/10.3389/fcvm.2019.00078

Moritani, T., Nagata, A., deVries, H. A., & Muro, M. (1981). Critical power as a measure of physical work capacity and anaerobic threshold. *Ergonomics*, *24*(5), 339–350. https://doi.org/10.1080/00140138108924856

Muriel, X., Mateo-March, M., Valenzuela, P. L., Zabala, M., Lucia, A., Pallares, J. G., & Barranco-Gil, D. (2022). Durability and repeatability of professional cyclists during

a Grand Tour. *European journal of sport science*, *22*(12), 1797–1804. https://doi.org/10.1080/17461391.2021.1987528

Noakes T. D. (2007). The central governor model of exercise regulation applied to the marathon. *Sports medicine (Auckland, N.Z.)*, *37*(4-5), 374–377. https://doi.org/10.2165/00007256-200737040-00026

Nuuttila, O. P., Laatikainen-Raussi, V., Vohlakari, K., Laatikainen-Raussi, I., & Ihalainen, J. K. (2025). Durability in recreational runners: effects of 90-min low-intensity exercise on the running speed at the lactate threshold. *European journal of applied physiology*, *125*(3), 697–705. https://doi.org/10.1007/s00421-024-05631-y

Nybo, L., & Secher, N. H. (2004). Cerebral perturbations provoked by prolonged exercise. *Progress in neurobiology*, *72*(4), 223–261. https://doi.org/10.1016/j.pneurobio.2004.03.005

Olds, T., Norton, K., Craig, N., Olive, S., & Lowe, E. (1995). The limits of the possible: models of power supply and demand in cycling. *Australian journal of science and medicine in sport*, *27*(2), 29–33.

Ørtenblad, N., Westerblad, H., & Nielsen, J. (2013). Muscle glycogen stores and fatigue. *The Journal of physiology*, *591*(18), 4405–4413. https://doi.org/10.1113/jphysiol.2013.251629

Ørtenblad, N., Zachariassen, M., Nielsen, J., & Gejl, K. D. (2024). Substrate utilization and durability during prolonged intermittent exercise in elite road cyclists. *European journal of applied physiology*, *124*(7), 2193–2205. https://doi.org/10.1007/s00421-024-05437-y

Peeters, W. M., Barrett, M., & Podlogar, T. (2025). What is a cycling race simulation anyway: a review on protocols to assess durability in cycling. *European journal of applied physiology*, *125*(6), 1527–1548. https://doi.org/10.1007/s00421-025-05725-1

Poole, D. C., Burnley, M., Vanhatalo, A., Rossiter, H. B., & Jones, A. M. (2016). Critical Power: An Important Fatigue Threshold in Exercise Physiology. *Medicine and science in sports and exercise*, *48*(11), 2320–2334. https://doi.org/10.1249/MSS.0000000000000939

Puchowicz, M. J., Mizelman, E., Yogev, A., Koehle, M. S., Townsend, N. E., & Clarke, D. C. (2018). The Critical Power Model as a Potential Tool for Anti-doping. *Frontiers in physiology*, *9*, 643. https://doi.org/10.3389/fphys.2018.00643

Romijn, J. A., Coyle, E. F., Sidossis, L. S., Gastaldelli, A., Horowitz, J. F., Endert, E., & Wolfe, R. R. (1993). Regulation of endogenous fat and carbohydrate metabolism in relation to exercise intensity and duration. *The American journal of physiology*, *265*(3 Pt 1), E380–E391. https://doi.org/10.1152/ajpendo.1993.265.3.E380

Robergs, R. A., Ghiasvand, F., & Parker, D. (2004). Biochemistry of exercise-induced metabolic acidosis. *American journal of physiology. Regulatory, integrative and comparative physiology*, *287*(3), R502–R516. https://doi.org/10.1152/ajpregu.00114.2004

Sahlin, K., & Seger, J. Y. (1995). Effects of prolonged exercise on the contractile properties of human quadriceps muscle. *European journal of applied physiology and occupational physiology*, *71*(2-3), 180–186. https://doi.org/10.1007/BF00854977

Samozino, P., Horvais, N., & Hintzy, F. (2006). Interactions between cadence and power output effects on mechanical efficiency during sub maximal cycling exercises. *European journal of applied physiology*, *97*(1), 133–139. https://doi.org/10.1007/s00421-006-0132-x

Sarzynski, M. A., Ghosh, S., & Bouchard, C. (2017). Genomic and trans criptomic predictors of response levels to endurance exercise training. Journal of Physiology, 595, 2931–2939. https://doi.org/10.1113/ JP272559

Skiba, P. F., Chidnok, W., Vanhatalo, A., & Jones, A. M. (2012). Modeling the expenditure and reconstitution of work capacity above critical power. *Medicine and science in sports and exercise*, *44*(8), 1526–1532. https://doi.org/10.1249/MSS.0b013e3182517a80

Smyth, B., Maunder, E., Meyler, S., Hunter, B., & Muniz-Pumares, D. (2022). Decoupling of Internal and External Workload During a Marathon: An Analysis of Durability in 82,303 Recreational Runners. *Sports medicine (Auckland, N.Z.)*, *52*(9), 2283–2295. https://doi.org/10.1007/s40279-022-01680-5

Spragg, J., Leo, P., Giorgi, A., Gonzalez, B.M. and Swart, J. (2024), The intensity rather than the quantity of prior work determines the subsequent downward shift in the power duration relationship in professional cyclists. *Eur J Sport Sci, 24*(4),449-457. https://doi.org/10.1002/ejsc.12077

Spragg, J., Leo, P., & Swart, J. (2023). The Relationship between Physiological Characteristics and Durability in Male Professional Cyclists. *Medicine and science in sports and exercise*, *55*(1), 133–140. https://doi.org/10.1249/MSS.0000000000003024

Stevenson, J. D., Kilding, A. E., Plews, D. J., & Maunder, E. (2022). Prolonged cycling reduces power output at the moderate-to-heavy intensity transition. *European journal of applied physiology*, *122*(12), 2673–2682. https://doi.org/10.1007/s00421-022-05036-9

Stevenson, J. D., Kilding, A. E., Plews, D. J., & Maunder, E. (2024). Prolonged exercise shifts ventilatory parameters at the moderate-to-heavy intensity transition. *European journal of applied physiology*, *124*(1), 309–315. https://doi.org/10.1007/s00421-023-05285-2

Tornero-Aguilera, J. F., Jimenez-Morcillo, J., Rubio-Zarapuz, A., & Clemente-Suárez, V. J. (2022). Central and Peripheral Fatigue in Physical Exercise Explained: A Narrative Review. *International journal of environmental research and public health*, *19*(7), 3909. https://doi.org/10.3390/ijerph19073909

Unhjem R. J. (2024). Changes in running economy and attainable maximal oxygen consumption in response to prolonged running: The impact of training status. *Scandinavian journal of medicine & science in sports*, *34*(5), e14637. https://doi.org/10.1111/sms.14637

Väisänen, D., Ekblom, B., Wallin, P., Andersson, G., & Ekblom-Bak, E. (2024). Reference values for estimated VO_2max by two submaximal cycle tests: the Åstrand-test and the Ekblom-Bak test. *European journal of applied physiology*, *124*(6), 1747–1756. https://doi.org/10.1007/s00421-023-05398-8

Valenzuela, P. L., Alejo, L. B., Ozcoidi, L. M., Lucia, A., Santalla, A., & Barranco-Gil, D. (2022). Durability in Professional Cyclists: A Field Study. *International journal of sports physiology and performance*, *18*(1), 99–103. https://doi.org/10.1123/ijspp.2022-0202 Van Erp, T., & Sanders, D. (2021). Demands of professional cycling races: Influence of race category and result. *European journal of sport science*, *21*(5), 666–677. https://doi.org/10.1080/17461391.2020.1788651

Van Erp, T., Sanders, D., & Lamberts, R. P. (2021). Maintaining Power Output with Accumulating Levels of Work Done Is a Key Determinant for Success in Professional Cycling. Medicine and science in sports and exercise, 53(9), 1903–1910. https://doi.org/10.1249/MSS.0000000000002656

Wingo, J. E., Lafrenz, A. J., Ganio, M. S., Edwards, G. L., & Cureton, K. J. (2005). *Cardiovascular drift is related to reduced maximal oxygen uptake during heat stress. Medicine and science in sports and exercise, 37*(2), 248–255. https://doi.org/10.1249/01.mss.0000152731.33450.95

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Deutsches Abstract

In den letzten Jahren wurde Durability als vierter Eckpfeiler des Ausdauersports vorgeschlagen. Im Gegensatz zu den etablierten Schwellenmodellen, der maximalen Sauerstoffaufnahme ($\dot{V}O_{2max}$) und der Effizienz, liegt der Fokus hier auf den physiologischen Veränderungen, die durch Ermüdung entstehen. Wie frühere Studien gezeigt haben, verschieben sich die Parameter, die den Ausdauersport beeinflussen, während längerer Belastung, was zu einem Leistungsverlust führt. Dies unterstreicht die Bedeutung der Durability, welche die individuelle Widerstandsfähigkeit gegenüber Ermüdung beschreibt.

Der Einfluss von Durability auf die etablierten physiologischen Kenngrößen ist jedoch noch nicht vollständig geklärt. Ziel der vorliegenden Studie ist es, die Verbindung zwischen dem Verlust der maximalen 5-Minuten-Leistung (ΔMPO_{5min}) und den physiologischen Markern wie VT1, VT2, $\dot{V}O_{2max}$ und der Bruttoeffizienz (GE) unter erholten Bedingungen zu untersuchen. Darüber hinaus wurde ein möglicher Zusammenhang zwischen dem ΔMPO_{5min} und dem Rückgang der $\dot{V}O_{2max}$, sowie der GE-Werte analysiert.

Sieben gut trainierte Amateurradsportler absolvierten einen Rampenprotokoll sowie einen GE-Test, um ihr physiologisches Profil zu bestimmen. Basierend auf diesen Werten absolvierten sie zwei 5-minütige Zeitfahren, bei denen die durchschnittliche Leistung gemessen wurde. Während der erste Versuch im nicht-ermüdeten Zustand stattfand, erfolgte der zweite Versuch nach einem einstündigen Ermüdungsprotokoll, das zu einer Vorbelastung von etwa 15 kJ/kg führte. Anschließend wurden Korrelationen zwischen den Werten, sowie eine Varianzanalyse mit Messwiederholung (ANOVA) für die GE-Werte untersucht.

Die Ergebnisse zeigten keine signifikanten Korrelationen zwischen dem Abfall der MPO_{5min}-Werte und der GE, $\dot{V}O_{2max}$ oder VT1. Die absolute Leistung an der VT2 zeigte jedoch eine signifikante negative Korrelation mit der Reduktion der MPO_{5min}. Zudem ergab die ANOVA einen signifikanten negativen Effekt des Ermüdungsprotokolls auf die GE-Werte.

Zusammenfassend legen die Ergebnisse nahe, dass Durability nicht durch die bereits etablierten Marker des Ausdauersports vorhergesagt werden kann. Dies stärkt die Hypothese, dass Durability als ein eigenständiger vierter Eckpfeiler betrachtet werden muss, der weiterer Untersuchung bedarf.