THE PSYCHOPHYSIOLOGICAL REGULATION OF PACING BEHAVIOUR AND PERFORMANCE DURING PROLONGED ENDURANCE EXERCISE

by

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DECLARATION

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2. Venhorst A, Micklewright D, Noakes TD. Modelling the process of falling behind and its psychophysiological consequences. *Br J Sports Med* 2017; **0**: 1–6

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LIST OF ABBREVIATIONS

%	Percentage
χ^2	Chi-square
A.U.	Arbitrary Units
Δ AIC	Difference in Akaike information criterion
Δ AUC	Difference in area under the curve
°	Degrees
°C	Degrees Celsius
3D	Three-dimensional
4-DMS	Four-dimension mood scale
abs	Absolute
ACRISS	Action crisis scale
AD-ACL	Activation deactivation adjective check list
AG	Affect grid
AIC	Akaike information criterion
ANOVA	Analysis of variance
ART	Aligned rank transformation
AUC	Area under the curve
BASE	Baseline
CFI	Comparative fit index
CGM	Central governor model
CI	Confidence interval
cm	Centimetre
COMP	Competition
CR	Critical ratio
CR-10	Category ratio scale
CTRL	Control
DMT	Dual-mode theory
DOMS	Delayed onset of muscular soreness
DST	Dynamical systems theory

e	Error
EIMD	Exercise-induced muscle damage
EMG	Electromyogram
EVS	Empirical valence scale
FAS	Felt arousal scale
FS	Feeling scale
FSS	Flow state scale
g	Gram
GCT	Gate control theory
h	Hours
HPAA	Hypothalamo pituitary adrenal axis
HPLC	High pressure liquid chromatography
HTW	Hitting the wall
Hz	Hertz
IC	Insular cortex
INDV	Individual
INTV	Intervention
K_2-EDTA	Potassium-Ethylenediaminetetraacetic acid
kg	Kilogram
km	Kilometre
L	Litres
LMMF	Locomotor muscle fatigue
min	Minutes
ml	Millilitre
mm	Millimetre
MVC	Maximum voluntary contraction
NDST	Non-linear dynamical systems theory
NFI	Normed fit index
PANAS	Positive activation negative activation schedule
PCLOSE	p of close fit

PL	Performance level category
PM	Psychological momentum
PO	Power output
PPO	Peak power output
p-RPE	Rating of physical sensations
R^2	Total variance explained
RCP	Respiratory compensation point
RDS	Running discomfort scale
rel	Relative
RMSEA	Root mean square error of approximation
RPE	Rating of perceived exertion
rpm	Revolutions per minute
SCT	Social cognitive theory
SD	Standard deviation
SE	Standard error
s	Seconds
SEM	Structural equation modelling
SRMR	Standardised root mean square residual
TEA	Task effort and awareness
TRT	Task related thoughts
TT	Time trial
TUT	Task unrelated thoughts
VCO_2	Carbon dioxide production
V_E	Ventilatory volume
VO_2	Oxygen consumption
VO_{2peak}	Peak oxygen consumption
VT-1	First ventilatory threshold
W	Watts

THESIS ABSTRACT

THE PSYCHOPHYSIOLOGICAL REGULATION OF PACING BEHAVIOUR AND PERFORMANCE DURING PROLONGED ENDURANCE EXERCISE

Current models of exercise regulation almost solely rely on the Gestalt phenomenon of perceived exertion. This limits a more comprehensive understanding of how cause-effect relationships come to be and how perception-action coupling determines pacing behaviour and performance fatigability.

A three-dimensional framework of centrally regulated and goal-directed exercise behaviour is proposed, which differentiates between sensory-discriminatory, affective-motivational, and cognitive-evaluative processes hypothesised to underpin perceived fatigability. In short: (A) perceived physical strain and perceived mental strain are primary regulators of pacing behaviour necessary to align planned behaviour with current physiological state, (B) core affect plays a primary and mediatory role in performance regulation, and (C) the mindset- shift associated with an action crisis plays a primary role in volitional self-regulatory control and decision-making.

In study one, 23 cyclists of distinct performance levels engaged in 70-km individual and head-to-head competition time trials against a performance matched opponent. Sensory constructs were primarily associated with regulation of pacing behaviour. Affective and cognitive constructs acted as context-dependent modifiers and were primarily associated with regulation of performance.

A five-step structural equation modelling procedure was applied to assess the extent to which the observed data fit the hypothesised cause–effect relationships under the constraint of psychological duress: valence deterioration was found to mediate the relationship between falling-behind and action crisis; which in turn predicted increased non-adaptive endocrinological distress response; which in turn predicted performance decrement.

In study two, 22 highly-trained runners completed two self-paced 20-km treadmill time trials in a tapered condition and with locomotor muscle fatigue and exercise-induced muscle damage. The latter was associated with medium increases in markers of physiological distress and large alterations in perceived physical strain, affective

valence, and cognitive mindset. This indicates heuristic and rational antecedents in the goal-disengagement process.

Structural equation modelling confirmed the hypothesised dual-pathway model under the constraint of physical duress: haematological indicators of EIMD predicted (1) amplified physiological strain and non-adaptive endocrinological distress response and (2) increase in perceived physical strain, which mediated and predicted decrease in valence; which in turn predicted an increase in action crisis; and both physiological and perceptual effects predicted performance fatigability.

The proposed framework has the potential to enrich theory development in centrally regulated and goal-directed exercise behaviour by providing novel insights into and more complete account of the dynamic and complex processes in strain-perception-thinking-action coupling during prolonged endurance exercise.

“Treating people as machines may be scientifically and philosophically accurate, but it’s a cumbersome waste of time, if you want to guess what the person is going to do next. The economically useful way to model a person is to treat him as a purposeful goal-seeking agent with pleasures and pains, desires and intentions…”

Richard Dawkins

CHAPTER ONE

TOWARDS A THREE-DIMENSIONAL FRAMEWORK OF CENTRALLY REGULATED AND GOAL-DIRECTED EXERCISE BEHAVIOUR

ABSTRACT

The Central Governor Model (CGM) ignited a paradigm shift from concepts of catastrophic failure towards central regulation of exercise performance. However, the CGM has focused on the central integration of afferent feedback in homeostatic control. Accordingly, it neglected the important role of volitional self-regulatory control and the integration of affective components inherently attached to all physiological cues. Another limitation is the large reliance on the Gestalt phenomenon of perceived exertion. Thus, progress towards a comprehensive multi-dimensional model of perceived fatigability and exercise regulation is needed.

Drawing on Gate Control Theory (GCT) of pain, a three-dimensional framework of centrally regulated and goal-directed exercise behaviour is proposed, which differentiates between sensory, affective, and cognitive processes that shape the perceptual milieu during exercise.

It is suggested that: (A) perceived mental strain and perceived physical strain are primary determinants of pacing behaviour reflecting sensory-discriminatory processes necessary to align planned behaviour with current physiological state, (B) core affect plays a primary and mediatory role in exercise and performance regulation, and its underlying two dimensions - hedonicity and arousal - reflect affective-motivational processes triggering approach and avoidance behaviour, and (C) the mindset-shift associated with an action crisis plays a primary role in volitional self-regulatory control reflecting cognitive-evaluative processes between further goal-pursuit and goal-disengagement.

The proposed framework has the potential to enrich theory development in centrally regulated and goal-directed exercise behaviour by emphasising the multidimensional dynamic processes underpinning perceived fatigability and provides a practical outline for investigating the complex interplay between the psychophysiological determinants of pacing and performance during prolonged endurance exercise.

INTRODUCTION

Around the turn of the last century, a series of articles challenged the traditional reductionistic catastrophe models of exercise physiology, which hold that fatigue is the direct consequence of homeostatic failure in one or other physiological system.[1–5] In 2004/2005 another series of articles published in this journal introduced a novel alternative model of integrative control in exercising humans.[6–10] This Central Governor Model (CGM) has since ignited a paradigm shift from the concepts of peripheral or central catastrophic failure towards central regulation.[11,12]

The CGM postulates a complex psychophysiological model in which the brain integrates and interprets afferent feedback from myriads of homeostats in a teleoanticipatory feedforward fashion, specifically to maintain homeostasis and prevent catastrophic biological failure.[6–10] The CGM extends Ulmer's model of efferent control of skeletal muscle recruitment during exercise, and thus indirectly the control of muscular metabolic rate.[13]

Ulmer[13] proposed that a feedback control system must exist in concert with a 'programmer' that anticipates the finishing point of an exercise bout to maintain homeostasis and optimise performance. More specifically, the extent of neuromuscular recruitment and thus exercise intensity is determined in anticipation based on expected and remaining exercise duration or distance, previous experience, endogenous and exogenous reference signals, psychological state, and many other physiological variables.[14] Once exercise begins neural efferent motor command is continuously regulated based on interoceptive,[15–17] proprioceptive,[18] and exteroceptive[19] information in relation to predicted 'templates'[20] and homeostatic set-points.[21,22] Critically, motor command is further adjustable by a host of centrally-acting performance modifiers.[23,24]

Moreover, the CGM accounts for conscious layers of behavioural homeostatic control through the generation of the conscious perceptions of exertion and fatigue, which the model suggests arise from error differences between predicted exercise-induced changes and actual disturbances of physiological state.[25] According to Enoka and Duchateau,[26] human fatigue is a psychophysiological symptom underpinned by interactions between (A) performance fatigability – the observed decline in an objective

measure of endurance performance and (B) perceived fatigability – changes in the perceptions that regulate the integrity of the performer. It is therefore proposed that perceived fatigability has the teleological function of altering exercise behaviour by modifying or constraining the conscious desire to tap into a protected homeostatic reserve.[27] The subjective nature of perceived fatigability therefore allows for behavioural flexibility through volitional control of tolerable homeostatic deviations, but only within the boundaries set by subconscious homeostatic set-points and complex systems control in peripheral systems necessary to sustain life within the narrow permissible range.[21,28,29]

Despite the proposed coexistence of conscious and subconscious regulatory mechanisms in the maintenance of homeostasis, the criticism of the CGM is almost entirely based on a conceptually flawed conscious and subconscious dichotomy.[30] Edwards and Polman proposed that this dilemma could be resolved when decision-making in pacing is viewed on a continuum from subconscious to conscious processes with varying degrees of awareness.[31] However, given the hierarchical interdependency of subconsciousness and consciousness, this approach appears to be of limited theoretical scope and investigative utility in understanding the underlying mechanisms of observed pacing behaviour. Micklewright et al.[32] instead suggested to rather focus on intuitive and deliberative processes, which are more adaptive and flexible than trajectory perceived exertion centric models. Thus, dual-process theory may more completely account for the dynamic changes in the psychophysiological determinants of pacing and performance in novel, varying, and uncertain (i.e. 'real-world') conditions.

Regardless of early[3] and continued[10,23] acknowledgement of the importance of psychological-motivational performance modifiers in exercise regulation, the CGM has focused on the central integration of afferent sensory feedback in the teleoanticipatory feedforward regulation of homeostatic control. Consequently, it has neglected deliberative processes such as the important self-regulatory role of volition and moderating social-cognitive factors in the cognitive-evaluation of these somatosensory cues. The CGM further neglected intuitive processes such as the integration of affective-motivational components that are inherently attached to all interoceptive cues,[33–35] and which may provide a more parsimonious account of changes in

approach and avoidance behaviour than direct alteration through sensory afferent feedback signals.

Another limitation that the CGM shares with alternative psychobiological models of exercise regulation[30,31,36,37] is the large reliance on the Gestalt phenomenon of perceived exertion to explain something as multifaceted as centrally regulated and goal-directed exercise behaviour. The use of the one-item Rating of Perceived Exertion (RPE) scale clearly is an oversimplification of this complex psychophysiological construct,[38–41] ignores qualitatively distinct variations of perceived exertion, and is likely inadequate to explain exercise behaviour in its entirety.[42] The challenge therefore continues to be the examination of alterations in exercise behaviour from a dynamic systems perspective, rather than concentrating on any single system or variable in the search to understand the causes of fatigue and the limits of human endurance performance.[10]

Hence, progress towards a multidimensional concept of human exercise regulation is required that emphasises the integration of both sensory and affective cues as well as the cognitive interpretation thereof. By moving towards a comprehensive psychophysiological framework of centrally regulated and goal-directed exercise behaviour, researchers will be better equipped to investigate and interpret the complex and dynamic interplay of the psychophysiological determinants of pacing and performance during prolonged endurance exercise.

CONFINES, TRENDS, AND SUGGESTIONS

Borg[43] initially differentiated between perceived force, effort, exertion or resistance during short-term exercise dominated by muscular strain (thereafter predominantly used in kinesthesiology research) and perceived exertion, laboriousness, fatigue or perceived exhaustion during long-term exercise increasingly dominated by cardiorespiratory strain (thereafter predominantly used in exercise physiology research). However, Borg did not differentiate between the mental effort *invested into / required to continue with* the task and the overall perception of physical strain *caused by* the task (i.e. between perceived mental strain and perceived physical strain).[39,40] Thus, following Borg's seminal work, the measurement and interpretation of RPE data has been confounded by the interchangeable use of these two interrelated but

independent constructs. This continues to create confusion and debate about what the RPE actually measures.[44] Hutchinson et al.[39] aimed to operationalise these two independent constructs by distinguishing between the terms ‘effort’ and ‘exertion’, but this suggestion has received criticism based on semantic grounds.[45] Notwithstanding, there is a continued need to distinguish between these two constructs and to avoid further semantic debates it is instead referred to ‘perceived mental strain’ reflecting the subjective task difficulty and mental effort expended and ‘perceived physical strain’ denoting the overall perception of physical strain from central and peripheral sites.

The current debate about what RPE actually measures[44,46] was evoked by Marcora,[47] who, by drawing on the kinesthesiology literature, proposed that perception of effort and perceived exertion (terms used interchangeably) are independent of afferent feedback and instead arise solely from centrally generated corollary discharges of efferent motor command. In response, Smirmaul[48] suggested that exercise is ultimately regulated by the interaction of two distinct entities, a centrally generated sense of effort and afferent feedback from the body’s physiological condition essential for homeostatic control.

Thirty years earlier, the mostly overlooked contribution of Cafarelli to a symposium on perceived exertion at the American College of Sports Medicine came to the same conclusion.[49–54] Cafarelli[51] proposed that muscular contractions are sensed and controlled in a feedforward and feedback fashion, with the feedforward loop creating the sense of effort and the feedback loop providing information on velocity, force, and position. (See figure 1).

Kinesthesiological experiments may artificially isolate these two cues and carefully instructed participants appear able to attend to either one for appropriate judgement of force and effort.[55,56] By comparing and matching information of desired contractions with the overall state of the muscular system, the brain would be equipped with a protective mechanisms of great precision, the purpose of which is to maintain a reserve capacity for locomotion as required for survival.[51]

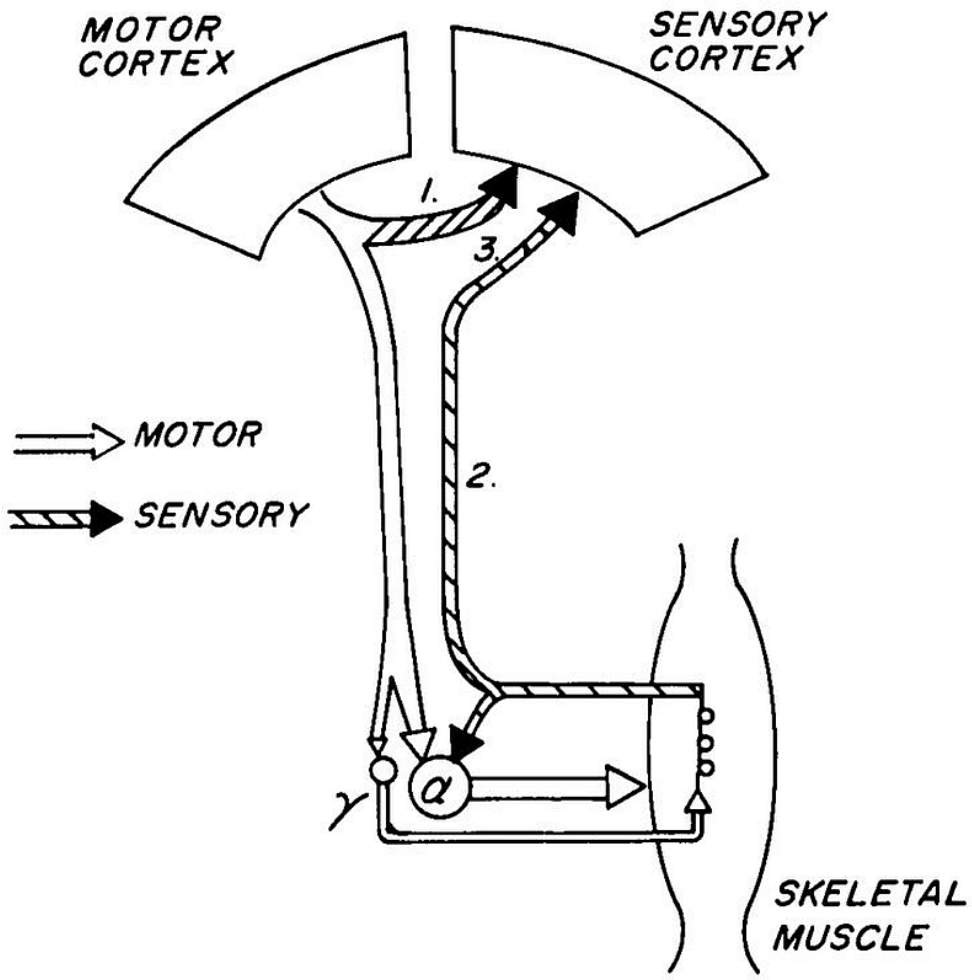


Figure 1: Schematic of three possible mechanisms for generating muscle sensations. 1. Feedforward: a feedforward loop generates the sense of effort, 2. Feedback: an afferent feedback loop provides information from peripheral receptors on velocity, force, and position, and 3. Feedforward + feedback: Afferent inflow from peripheral receptors is compared to a copy of motor outflow. Reprinted with permission from Cafarelli.[51]

Note: It is now more widely accepted that the sense of effort is generated upstream from the motor cortex, for example, the premotor and particularly the supplementary motor area.

However, within exercise physiology, the first attempts to delineate perceived physical strain from mental sense of effort were only made recently. Swart[57] modified the traditional 15-point RPE scale to include “only” the perceived physical symptoms (p-RPE) and developed a novel scale assessing task effort and awareness (TEA) that includes “only” the mental sense of effort. He reported that cyclists were able to differentiate between these two perceptual cues during submaximal workloads and maximal sprints, but not during incremental protocols to exhaustion and maximal time trial efforts.

Importantly, Swart[57] concluded that TEA is generated when the workload exceeds the one required to fit a predetermined RPE template. This implies that TEA is generated secondary to physiological disruption and therefore conflicts with the more accepted view that the sense of effort arises primary to physiological disruption via centrally generated corollary discharges.[58–61]

Swart[57] further concluded that "the direct consequence of the increasing sense of effort will be an altered behaviour, specifically a voluntary reduction in the exercise intensity. Conversely, exercise intensities that do not pose a threat to homeostatic control produce no or little sense of effort" (p. 47). A clear violation of this conclusion occurs during the endspurt, where the effort invested is maximal and the up till then protected physiological reserve becomes (partly) available, exactly because the final effort does not threaten the maintenance of homeostasis.[62] Instead, exercise intensity is voluntarily increased to allow the highest workloads performed during a prolonged time trial matching the maximum predetermined RPE template values. Task effort, which Swart[57] suggests to be the conscious representation of the 'hazard score',[a] should therefore be nil at the finish line, as is the hazard score, but the very opposite holds true for TEA scores. Besides these theoretical concerns, the TEA scale has additional psychometrical flaws such as placing the descriptor "unaware" inside the scale's range and measuring two constructs (task effort *and* awareness) with a single-item scale. However, clear empirical evidence against the TEA concept is missing and warrants further investigation.

In line with the original findings, other research using the p-RPE and TEA scales found that these two perceptual cues cannot easily be distinguished during self-paced bicycle time trials of different length (16.1 km vs 40 km).[63] However, this may partly be an artefact of the overly simple laboratory protocols used (i.e. simulated flat and windless courses). Both cues rose linearly in both trials suggestive of a regulatory role in the teleoanticipatory regulation of pacing behaviour. Perceptual scores were significantly higher during the shorter time trial, but no interaction or main effect for scale was

[a] The 'hazard score' is a simple index of the likelihood to change pace. It is calculated as the product of momentary RPE and relative exercise distance remaining and consequently is always nil at the finish line.[62]

observed, although the main effect for scales approached statistical significance (F=4.1; p=0.053). This finding therefore certainly does not suffice to demonstrate the inability of participants to differentiate between perceived mental strain and perceived physical strain as suggested in a recent review.[45] As originally described by Smirmaul,[48] future investigations should use (simulated) profiled courses, which may assist delineating these two distinct entities.

To the best of our knowledge, only one other study has investigated the role of sense of effort and physiological discomfort on self-selected power output in cyclists during normoxia and hypoxia trials.[64] The authors used modified CR-10 scales and 'clamped' the sense of effort at '3' during a 5 min submaximal ride, followed by 5 progressive sprints, and two maximal sprints clamped at an effort of '10'. Mental sense of effort was closely related to alterations in power output and quadriceps muscle activation, while localised perceived physiological discomfort ratings increased in parallel with physiological disturbances and were significantly elevated during the hypoxia trial.[64] This suggests that (A) perceived mental strain and perceived physical strain can be experimentally dissociated, (B) perceived mental strain accurately reflects power output during short-term sprints, and (C) perceived physical strain accurately reflects disruption of the physiological state.

These findings are in full agreement with Cafarelli's proposal of efferent feedforward control initiating subjective planned behaviour and, after a lag period, afferent feedback control providing objective information on the caused physiological disturbances. When combined and compared, predictive coding and error differences provide essential information to a Bayesian inference algorithm that is hypothesised to underpin perception, cognition, and action.[65]

However, while of great theoretical interest and congruent with a recent proposal for a combined feedforward and feedback 'sensory tolerance limit',[66] the meaning and practical value of this differentiation in healthy individuals is unclear and warrants further investigation.

More importantly, researchers interested in fatigue and the limits of human performance should be mindful to test what underpins perceived fatigability and must ultimately move beyond debating what the Borg scale measures. Hence, greater

eclecticism in the choice of measurement tools and underlying theoretical concepts is needed to advance theory development in centrally regulated and goal-directed exercise behaviour. Progress towards a multidimensional concept of perceived fatigability is proposed differentiating between the distinct inputs of sensory, affective, and cognitive processes that shape the perceptual milieu during exercise. By emphasising a dynamic systems approach, a broad framework is provided within which current models of exercise regulation can be interpreted, compared, and discussed more meaningfully.

A THREE-DIMENSIONAL FRAMEWORK OF PERCEIVED FATIGABILITY

According to Mauger,[67,68] a promising research area to search for a multidimensional framework of perceived fatigability is exercise-induced discomfort and the independent, but closely related, construct of pain. In 1965 Melzack and Wall[69] proposed the Gate Control Theory (GCT) of pain, which provides an example of a comprehensive theory accounting for affective and cognitive factors in the perception of pain. The GCT proposes that pain is not merely an afferent sensory sensation that directly elicits the production of pain sensations in the respective brain areas, but pain signals first must flow through a gate-like control mechanism in the dorsal horn of the spinal cord. The proposed neural gating circuits can receive efferent signals from the brain allowing the pain sensation to be filtered and modified. However, the extent of cognitive control over ascending sensory transmission is limited and depends on the intensities of ascending and descending impulses as well as on the myelination and diameter of neural fibre types.

A complex interaction is proposed between three dimensions underpinning the overall perception of pain: (A) the sensory-discriminatory dimension representing the perceptual information of location, quality, and intensity, (B) the affective-motivational dimension triggering the affective response and arousal of behavioural drive towards approach or avoidance, and (C) the cognitive-evaluative dimension exerting *intensity dependent* volitional control over the other two dimensions.[70]

Tenenbaum et al.[71] conceptualised the construct of pain to be closely related to perceived discomfort in runners and drew on the GCT to develop the Running Discomfort Scale (RDS). The initial version of the RDS was empirically inferred from

the narratives of ten runners, who had recently completed a challenging 9 km run. Eventually, eight lower-order factors emerged (proprioceptive symptoms, leg symptoms, respiratory difficulties, dryness and heat, head or stomach symptoms, task completion thoughts, disorientation, and mental toughness) and were collapsed into the three global dimensions.

Hutchinson and Tenenbaum[39] extended this line of research to an isometric handgrip strength and a stationary cycling task at different intensities. They conceptualised physiological discomfort (aches, fatigue, and pain) to represent the sensory-discriminatory dimension; mental toughness, determination, and concentration to represent the affective-motivational dimension; and perceived effort, perceived exertion, and task aversion to represent the cognitive-evaluative dimension. Significant dimension × time interactions effects in both studies showed that the three dimensions provide distinct inputs into the overall perception of effort and that these can be perceived distinctly.[39]

The same group of researchers recently proposed a three-dimensional framework of perceived effort.[41] They suggested that central and peripheral physical sensations underpin the sensory-discriminatory dimension; arousal or activation, positive or negative affect, state anxiety, and task motivation the affective-motivational dimension; and perceived exertion and task aversion the cognitive-evaluative dimension. By including a social-cognitive perspective of perceived exertion and exercise tolerance, the model allows for external and internal mediating factors to influence the perception of effort.[72,73] Importantly, the effect of these cognitive factors are proposed to be a function of exercise intensity being most influential at submaximal sustainable workloads, while interoceptive cues prevail at workloads that preclude the maintenance of a true physiological steady-state.[38,74,75]

However, the proposed subordinate structure was not based on sound theoretical considerations, but empirically inferred, which might explain why significant discrepancies emerged in comparison to the original defining aspects of the three dimensions within GCT. For example, it was suggested that mental toughness and determination underpinning task motivation constitute the affective-motivational dimension.[39,71] However, the will to continue striving in the face of adversity to attain an important goal (i.e. exerting volitional control over the desire to disengage from

further goal-pursuit) can better be described as a cognitive strategy and thus be related to higher cortical brain areas such as the lateral prefrontal cortex.[76,77] It therefore more appropriately subordinates the cognitive-evaluative dimension. Also, task aversion (i.e. the desire to disengage from further goal-pursuit) was suggested to represent the cognitive-evaluative dimension, but actually constitutes an affect laden aversive drive triggering avoidance behaviour and thus can better be related to the limbic system.[78,79] It therefore more appropriately subordinates the affective-motivational dimension.

In the following a modified lower-order structure is proposed based on: (A) Melzack's original descriptions of the three dimensions, (B) constructs that are imbedded in sound psychological theories, (C) constructs that are interrelated, but can be meaningfully delineated, (D) constructs that are relevant for exercise regulation, (E) constructs that can dynamically change and be repeatedly measured during exercise, and (F) modern neuroscientific evidence.

The Sensory-Discriminatory Dimension of Perceived Fatigability

Melzack[70] described the sensory-discriminatory dimension of pain to represent perceptual information on location, quality, and intensity, thereby allowing spatio-temporal analysis of pain. Within the context of exercise regulation, sensory-discriminatory information needs to include perceptual information of the physiological condition of the entire body necessary to maintain homeostasis and optimise performance.

An Exercise Physiology Perspective

Borg[50,80] conceptualised perceived exertion as a gestalt phenomenon and proposed it to be the single best indicator of somatic stress and physical strain integrating information from peripheral (i.e. muscles and joints) and central (i.e. cardiovascular and respiratory) sites as well as from the central nervous system. More specifically, Borg[81] considers emotions and motivation as well as cognitive variants such as memory and attention to be an integral part of the gestalt concept of perceived exertion. Pandolf[53,82] therefore proposed RPE to be a superordinate variable, which is consequently only loosely related to the underlying physiological variables. A multilevel model of subjective RPE ratings also explains why, despite great efforts, exercise

physiologists have failed to find a single most important sensory cue that is consistently and causally related to RPE.[83,84]

Subordinate localised ratings of perceived exertion for central and peripheral sites are generally stronger associated with physiological markers of physical strain, and the strength of this relationship increases as a function of exercise intensity.[85,86] This suggests that individuals can localise the source of sensory information and differentiate the magnitude of grouped perceptual cues reflecting cardiorespiratory and muscular strain during exercise.[84] Importantly, a single factor or system can become the dominant cue when approaching homeostatic failure.[53] This is facilitated by an attentional shift towards interoceptive cues at unsustainable workloads and towards the dominant cue once a factor or system approaches its limits.[86–89]

Sensory-discriminatory information is continuously sensed, integrated, and interpreted, thereby allowing anticipation of a potential threat to homeostasis early on in exercise.[90] Accordingly, it has been shown that information from various sites and systems, such as muscle glycogen content,[15] core temperature,[91] hypoxia,[92] and eccentric muscle loading,[18] are all related to alterations in pacing behaviour well in advance of critical homeostatic disturbances.

Neuroscientific support for spatio-temporal analysis of the physiological state of the entire body would require: (A) somatotopic organisation within the underlying neural structures, (B) continuous integration of afferent feedback in a hierarchically organised homeostatic, affective, and cognitive network, and (C) sensory cues must relate to the putative neural pathway as a function of stimulus strength.

Mechanosensory and proprioceptive information are transmitted by type I and II fibres and respectively via the spinothalamic and spinocerebellar tract, and relay to somatotopically organised nuclei of the thalamus and somatosensory cortex in a medial to lateral fashion.[93–95] In contrast, pain signals and information from metabo- and mechanoreceptors as well as the autonomic nervous system are conveyed by unmyelinated type III and IV fibres and are somatotopically organised in an anterior to posterior manner in nuclei of the thalamus and insular cortex.[35,94] The function of these separable type III and IV somatosensory afferents appears to be to reflexively increase autonomic responses facilitating exercise,[96] but also to limit intramuscular metabolic

disturbance via alteration of spinal motor neurons eventually contributing to central fatigue.[97,98]

The lamina I spinothalamic pathway is activated in direct relationship to the strength of the afferent input and in a continuous fashion.[94] It has hierarchically organised somatoautonomic reflex arcs at the spinal, brain stem, and thalamic level, which are essential for homeostatic control.[94,99] These arcs may act as complementary sensory and motor regions that subserve homeostasis with coordinated regulatory behaviour, connect to limbic system structures, and underpin consciousness.[99,100] The dorsal insular cortex (IC) is somatotopically organised and represents objective information on the physiological state of the entire body,[100] thereby allowing for modality identification and stimulus localisation.[35,101] The mid IC provides an integrative zone for affective and environmental information needed for homeostatic efficiency, which is then re-represented in the anterior IC.[100] The anterior IC in turn closely relates to the experience of subjective perceptions[34] and awareness,[102] as well as connects to the anterior cingulate and prefrontal cortices, thereby allowing crosstalk between affect and cognition.[94,103] In addition, afferent information from the sympathetic and parasympathetic nervous system are organised in an opponent fashion. While sympathetic inflow is predominantly represented to the right anterior IC, parasympathetic inflow is predominantly lateralised to the left anterior IC.[104] This intensity dependent frontal-brain asymmetry provides the neural basis for efficient homeostatic control based on energy management, affective responses, and cost-benefit analysis of goal-directed behaviour.[22,105]

Supportive evidence for a crucial role of this putative pathway described above comes from increased activation of the IC after fatiguing cycling exercise,[106,107] fatiguing handgrip task,[107,108] and during increased inspiratory breathing load.[109] Interestingly, the subjective perceptions are more accurate and less aversive in elite athletes suggesting a critical role for the IC in the complex integration of physiological, affective, and cognitive components underpinning exercise behaviour and performance.[109] Furthermore, non-invasive stimulation of the left temporal and insular cortices integrating predominantly information from the parasympathetic branch of the autonomic nervous system reduces heart rate and perceived exertion during submaximal, but not maximal, exercise intensities.[110]

A Kinesthesiology Perspective

While exercise physiologists largely focused on the physiological variables that give rise to perceived exertion, kinesthesiologists concluded that individuals rely on afferent inflow to sense movement, position, and tension, but on corollary discharges to perceive the senses of effort, force, and heaviness.[58,111,112]

The importance of central motor command in determining the sense of effort is most evident during force and weight matching tasks after induced fatigue or partial paralysis of the reference limb. These interventions induce greater perceived heaviness of a given weight and consequently require greater compensatory motor command and effort to lift the same. When subjects are instructed to match a weight lifted by the fatigued and/or paralysed reference limb with a variable weight by the non-fatigued and/or non-paralysed indicator limb, the perceived weight lifted with the weakened muscle is overestimated.[113] Similar matching errors do occur, when subjects are instructed to match isometric forces produced with the weakened reference limb.[114] Thus, subjects usually place more reliance on effort expended than on actually achieved tension.[115]

Supportive evidence also accumulated for a significant role of central motor command in the senses of movement, position, and tension.[116–119] Thus, efferent signals do contribute to the judgement of limb position in a graded level during absence of afferent feedback, for example during experimentally induced complete paralysis and anaesthesia,[120] and also, albeit to a smaller degree, in its presence, for example during experimentally induced paralysis only.[121]

On the other hand, Luu et al.[122] recently showed that muscle weakness of 60%, induced by high-force fatiguing exercise and low-force fatiguing exercise plus vibration of the muscle tendon respectively led to a 15% and 14% decrease in the sense of heaviness (i.e. the weight lifted by the fatigued muscle felt lighter). (See figure 2).

They further showed that after muscle force recovery from complete paralysis back to 40% of maximum voluntary contraction (MVC) perceived heaviness of a reference weight lifted with the weakened muscle was underestimated by 31%.[122] These findings are the opposite of the predictions of the central corollary discharge theory.

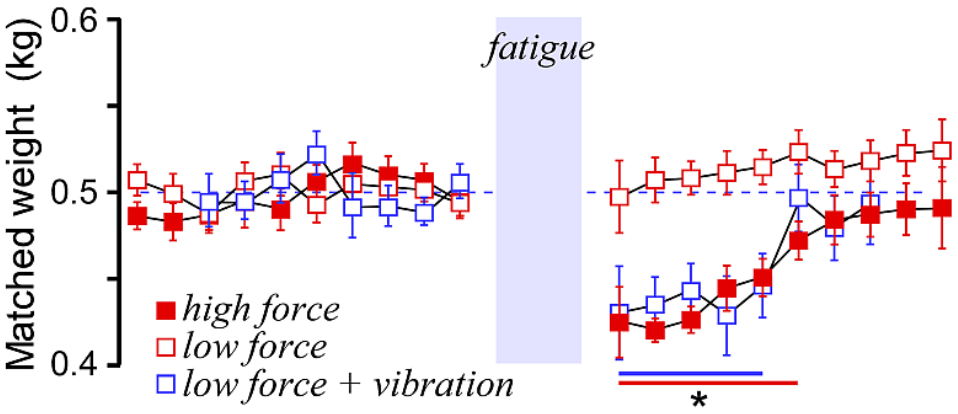


Figure 2: Effects of muscle fatigue on weight perception. Weights selected to match 500 g lifted with the reference thumb before (left) and after (right) fatiguing the reference muscle until its force was reduced to 40% of the initial MVC level. Data are means ± SEM (n = 16, and n = 10 for vibration data). After the high-force fatigue (filled red squares) subjects indicated that the reference weight felt lighter and this recovered over several minutes. After low-force fatigue (open red squares) to the same level of 40% maximal force output, they indicated that the weight felt the same as that lifted by the non-fatigued arm. After the same low-force fatigue, but with strong vibration of the muscle tendon (open blue squares), they indicated that the reference weight felt lighter as it had with the high-force fatigue. * = $p < 0.05$ from baseline by ANOVA. Reprinted with permission from Luu et al.[122]

During an isometric fatiguing contraction with the reference limb at 40% of MVC to task failure, these authors further showed in a force matching protocol that the perceived force only increased by 53%.[122] (See figure 3).

This is much less than would be expected if the effort-force relationship has a gain of approximately unity (i.e. fatiguing muscle force to half of its original force requires double the central motor command to generate the same force) and what is experienced by individuals with large-fibre sensory neuropathy.[122,123] After 40% of MVC could no longer be sustained despite maximum effort, perceived force declined in parallel with actual force, which is again inconsistent with the central corollary discharge theory.[122]

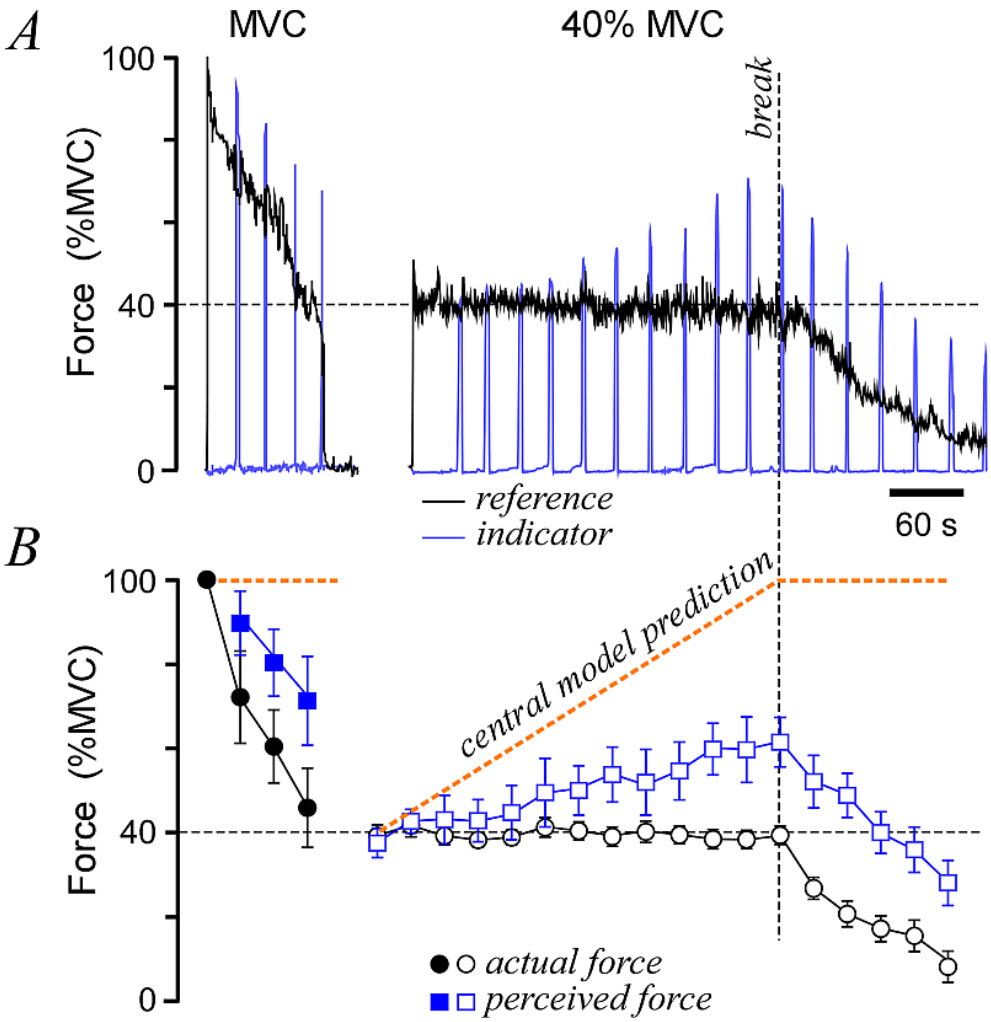


Figure 3: Perceived force exerted during an isometric fatiguing contraction. A: On the left, the black trace shows the declining force output of a continued maximal isometric contraction made with the reference thumb by one subject. The blue traces show intermittent isometric contractions made with the indicator thumb to match the reference force. On the right, the reference contraction is at 40% of a pre-test maximal force until it cannot be sustained. At that time (break), force output begins to decline. The forces indicated by the contralateral indicator thumb increase during the period of steady force output but decline steeply when the force output has become maximal. B: In the format of A are the group means ± SEM (n = 12) of the actual and perceived (indicated) force for each fatigue protocol. To allow for different times to reach the prescribed level of fatigue, time is scaled to the break point with relative equal-width bins averaged across subjects. The orange lines represent the prediction of perceived force exerted if perception is based on a central command with a gain of unity; i.e. halving maximal force output by muscle fatigue requires doubling the central command to maintain force output. Reprinted with permission from Luu et al.[122]

Similar results were provided by Brooks et al.,[124] who also concluded that the central command hypothesis alone cannot explain the perceptions of force and heaviness in their entirety and that a number of aspects are better interpreted in terms of a

peripheral afferent contribution to the senses of force and heaviness. Collectively, these results suggest that afferent signals from primary sensory intrafusal fibres of muscle spindles and Golgi tendon organs significantly contribute to the senses of force and heaviness. Thus, healthy individuals ultimately relate 'reafferent signals'[b] to efferent motor command in their perception and judgement of muscular effort, thereby challenging a feedforward and feedback dichotomy.

Despite widespread acceptance, neuroscientific evidence in support of corollary discharges from central motor command in humans is still limited. DeMorree et al.[60,125] showed that motor-related cortical potential amplitude detected on the scalp above the premotor and motor areas of the cortex is associated with sense of effort during submaximal dynamic elbow flexions and isometric knee extensions. However, the results are only correlational, of low spatial resolution, and the study designs do not permit differentiation between purely central or reafferent corollary discharges.

Using ischemic nerve block, Christensen et al.[126] provided evidence for increased interaction between the premotor cortex and primary somatosensory cortex during willed movement in the absence of proprioceptive feedback. In a later study, they evoked the sensation of movement in the absence of afferent feedback by transcranial magnetic stimulation of the primary motor and dorsal premotor cortex, with the latter being less affected by sensory and motor deprivation.[127] Collectively, these data suggest that the premotor cortex generates efference copies sent to the somatosensory cortex for comparison with reafferent feedback, thereby contributing to the sensation of movement. There also exists support for the reafference principle in central cardiovascular feedforward control.[128] Williamson has shown that this command activates thalamus and anterior cingulate and insular cortices,[129,130] with distinct regions for efference copies and afferent feedback in the latter.[131]

In conclusion, it is proposed that the reafference principle with its combined feedforward and feedback system provides the greatest precision in the maintenance

[b] Reafference is the anticipated sensory stimulation elicited by voluntary movements and is linked to efferent motor command. It thereby allows distinction between self-initiated sensory stimulation from external stimuli, so called exafference. The computed error difference between both signals underpins the reafference principle.[360,361]

of homeostasis during exercise regulation. It is further hypothesised that perceived mental strain and perceived physical strain are key variables used in the teleoanticipatory pacing algorithm and thus are primary regulators of pacing behaviour. Accordingly, it is suggested that the combined measurement of perceived mental strain and perceived physical strain will embody the underlying perceptual processes most accurately and will therefore represent the sensory-discriminatory dimension of perceived fatigability most appropriately.

The Affective-Motivational Dimension of Perceived Fatigability

The GCT describes pain as a distinctly hedonic experience that becomes overwhelming, demands immediate attention, disrupts ongoing behaviour and thought, and ultimately motivates behavioural change.[70] Therefore, "to consider only the sensory features of pain and ignore its motivational and affective properties, is to look at only part of the problem, and not even the most important part at that" (p.423).[70]

Melzack's[70] description of the affective-motivational dimension triggering the affective response and tendency of motivational drive towards approach or avoidance behaviour relates closely to LeDoux´s[79] 'survival circuit' concept, in which favourable biological functions and behaviours are initiated to allow the organism to survive and thrive by detecting and responding to challenges and opportunities. Survival circuits are likely involved in various homeostatic tasks such as defence, maintenance of energy and nutrients, fluid balance, thermoregulation, and reproduction. They can act without deliberate control unifying components of affect, motivation, reinforcement, and arousal to control behavioural responses as well as the related physiological adjustments. This is consistent with information reaching limbic structures such as the amygdala by means of a direct non-cortically mediated thalamo-amygdala pathway ('low road'), thus bypassing the indirect cortically mediated thalamo-cortico-amygdala pathway relaying at the somatosensory cortex ('high road').[132]

Interoceptive stimuli that are sufficiently intense to cause homeostatic disturbance can therefore directly arouse affective responses and initiate behavioural change. This supports the notion of the paramount importance of hedonicity in sensation, consciousness, emotion, and the determination of behaviour.[33,133,134] Thus, affect-laden responses to a homeostatic threat can create 'mental shortcuts', which require

neither cortically-mediated decision-making processes,[135] nor infinitely complex networks of hardwired reflexes in the maintenance of homeostasis.[136] This affect heuristic provided not only an evolutionary advantage by selecting the 'quick from the dead', but also an efficient mechanism to regulate homeostasis within its narrow permissible range. When homeostasis is threatened, subcortical homeostatic regulatory control pathways initiate behavioural change faster than cortically mediated voluntary changes, thereby providing a safe-guard mechanism, which protects from voluntary goal-directed behaviour potentially leading to homeostatic failure.

Furthermore, Cabanac[136] proposed that hedonicity is inherently attached to all interoceptive cues and functions as the 'common currency', thereby allowing conflicting motivational drives to be compared and ranked. The outcome of this permanent pre-rational computation of the algebraic sum of simultaneous positive and negative desires then motivates the final common path for a chosen behaviour.[137] Hence, hedonicity becomes the optimiser of behaviour by ranking priorities between clashing motivations, for example between the psychological need to satisfy a motif and the organism's physiological capacities.[138]

However, hedonicity is only one dimension of the non-reducible affective state in the construct of 'core affect', the other being arousal, which is required to prepare the organism for activation and to energise behavioural drive.[139,140] Core affect has been defined as a neurophysiological state that is consciously accessible as a simple, non-reflective feeling that is an integral blend of hedonic and arousal values.[141,142] It is phylogenetically (i.e. within the development of a species) and ontogenetically (i.e. within the development of an individual organism) the most basic affective construct, the others being emotion and mood.[143] For a comprehensive review on the crucial distinction between these constructs and their measurement see referenced textbooks.[144,145]

Critically, core affect is not only elicited and determined solely by afferent feedback pre-empting a potential threat to homeostasis, but rather is jointly influenced by interoceptive and cognitive factors. Ekkekakis proposed the Dual-Mode Theory (DMT) suggesting that the affective state changes as a consequence of the continuous interplay between these two general factors with the relative contribution shifting systematically as a function of exercise intensity.[146,147] Thus, affect is strongly

correlated with: (A) interoceptive cues at exercise intensities that preclude a physiological steady state and therefore pose a potential threat to homeostasis and (B) cognitive factors at exercise intensities proximal to the ventilatory/lactate thresholds that allow the maintenance of homeostasis by temporary adjustment of the physiological steady state to a higher level, thereby challenging the individual's endurance capacities.[148]

Additionally, below and above threshold intensities do respectively elicit homogenous perceptions of pleasure or displeasure, while inter-individual variability in affective responses predominates during exercise around threshold intensities.[147] Homogeneity in affective responses is considered adaptively useful. For example, the negative affective valence inherently attached to powerful perceptions of exertion, fatigue, and discomfort will ultimately prevent homeostatic failure by forcing behavioural change through phylogenetically more primitive and rigid subcortical pathways. Variability on the other hand is considered adaptively neutral. For example, the cognitive-evaluative trade-off between the respective benefits and risks of preference for and tolerance of interoceptive cues is primarily tied to more evolved and flexible cortical pathways.[146]

Neuroscientific evidence has been provided to support the plausibility of the core assumptions of DMT by showing that: (A) the homeostatic control system is highly sensitive to variations in the intensity of interoceptive information, (B) cortical and subcortical pathways are involved in a hierarchically organised homeostatic control system, and (C) appropriate gating mechanisms exist that redirect information via the subcortical pathways once exercise intensity precludes a physiological steady-state and homeostasis is threatened.[149] It is postulated that interoceptive information can reach the amygdala via several putative neural pathways from the spine, the nucleus of the solitary tract, the parabrachial nucleus, the periaqueductal grey, and the sensory thalamus, thereby 'short-cutting' the thalamo-cortico-amygdala pathway at various levels. Importantly, intensity-sensitive gating mechanisms at these critical locations can direct interoceptive information to either subcortical or cortical pathways depending on stimulus intensity and thus as a function of homeostatic threat.[149]

In conclusion, it is proposed that core affect is psycho-biologically linked to the sensory-discriminatory and cognitive-evaluative dimensions and that it plays a primary and mediatory role in centrally regulated and goal-directed exercise behaviour. It is further

hypothesised that core affect is a primary regulator of exercise performance. Accordingly, it is suggested that the two bipolar and independent dimensions hedonicity and arousal underpinning the construct of core affect most appropriately represent the affective-motivational dimension of perceived fatigability.

The Cognitive-Evaluative Dimension of Perceived Fatigability

Melzack described the cognitive-evaluative dimension to exert *intensity dependent* volitional control over the other two dimensions. This closely resembles Lehmann's description of a conscious 'skirmish' in performance regulation and protection of homeostatic reserve between the sum of negative feedback on the one hand and motivation and willpower on the other.[150] In line with the core assumptions of the CGM and DMT discussed previously, an increasing threat to homeostasis will strengthen the relationship between interoceptive cues and core affect, constrain the desire to tap into a protected reserve, and increasingly replace the motivational drive to engage in further goal-pursuit with a powerful affect-laden drive to disengage from further goal-pursuit, thereby leaving continued, but temporally limited, goal-striving to an act of volition or will.

From the above it becomes clear that static motivational theories solely relying on the motivational task of 'goal-setting' are insufficient to explain dynamic changes in observed exercise and pacing behaviour, and that long neglected theories on the volitional process of 'goal-striving' are required to enrich theory development in exercise science. In the early 1900's German will psychologists focused on the issue of volition[151–153] and made the crucial distinction between moving towards a chosen goal (i.e. goal-striving) and choosing a goal based on its expected value (i.e. goal-setting).[154] Still, motivational theories thereafter focused on achievement motivation and attempted to explain and predict crucial aspects of goal-striving such as work effort and performance outcome from aspects of goal-setting.[155,156] However, the motivational task of goal-setting (i.e. what a person wants to do) was more often than not unrelated to the self-regulatory and volitional task of goal-striving (i.e. what a person is actually doing).[157,158] The most comprehensive theory of goal-directed behaviour subsuming both goal-setting and goal-striving in a sequential order within one overarching theoretical model is Heckhausen's 'Rubicon model'[159] and in its extension Gollwitzer's mindset theory of action phases.[160,161]

The model assumes four action phases each characterised by a distinct mindset optimal for the specific task at hand. The pre-decisional action phase is characterised by a deliberative mindset, which facilitates mental contrasting of the desirability and feasibility of the wishes in an impartial and accurate manner. After a decision is made, individuals transit from deliberation to commitment, acquiring goal-intention, thereby metaphorically "crossing the Rubicon".[159] Individuals then move to the post-decisional, but still pre-actional phase characterised by an implemental mindset, which facilitates planning and implementation of intentions in a closed-minded and partial manner about when, where, and how to act. Individuals then initiate the actional phase characterised by an actional mindset in which cognitive tuning facilitates biased orientation towards goal-pursuit through 'illusionary optimism'. The post-actional phase is characterised by an evaluative mindset in which the individual analyses in an open-minded and impartial manner if the desired outcome of the goal-pursuit has been achieved. (For a summary see table 1).

However, barriers are a natural part of the goal-striving process,[162] particularly during prolonged endurance exercise, which at times can lead to the development of psychological crises. Schüler et al.[136] showed that a psychological crisis, related, but partially independent of a physiological crisis, can arise during a marathon. This is characterised by a disengagement impulse and increased thoughts about the costs and benefits of continuing and disengaging from the focal goal.[163]

According to Brandstätter et al.[163] individuals in such an 'action crisis' are caught between further goal-pursuit and goal-disengagement as a result of negatively valenced events related to setbacks and difficulties in goal-striving after considerable investments have already been made. This intrapsychic conflict shifts the cognitive orientation from an implemental or actional mindset facilitating volitional tasks such as goal-striving in the face of adversity to a deliberative mindset facilitating motivational tasks such as deliberating anew the desirability and feasibility of the focal or alternative goals.[164] Importantly, an action crisis is not a binary event inevitably leading to self-regulatory failure, but constitutes an antecedent in the temporally dynamic goal-disengagement process.[158]

Table 1: Summary of tasks and mindsets specific to the four action phases of Heckhausen's and Gollwitzer's mindset theory.

Mindsets	Deliberative	Implemental	Actional	Evaluative
Type	Motivational	Volitional	Volitional	Motivational
Action phase	Pre-decisional	Post-decisional-Pre-actional	Actional	Post-actional
Action task	Deliberating → deciding	Planning → initiating	Acting → attaining	Evaluating outcomes
Goals	Goal-setting	Goal-intention → Goal-implementation	Goal-pursuit-Goal-striving → Goal-attainment	Goal-achievement → Goal-evaluation
Cognitive orientation	Open-minded, unbiased, realism	Closed-minded, biased, optimism	Closed-minded, biased, optimism	Open-minded, unbiased, realism
Cognitive tuning	towards issues of desirability and feasibility	towards when, where and how; and to if-then	towards attaining chosen goal	towards analysis of outcome and desired consequences
Relationships	Decision-making, heuristic counterplea, action-outcome probability estimation	Focus, concentration, visualisation, positive illusion, self-verbalisation	Determination, illusionary optimism, commitment, persistence, "Flow"	
Practical application	Mental contrasting	Implementation intentions		

The intrapsychic conflict between further goal-pursuit and goal-disengagement also has physiological, affective, cognitive, and behavioural consequences.[164,165] In an action crisis, commitment to the focal goal is undermined[166] as alternative goals are being considered,[167] thereby reducing goal-shielding.[168] The associated mindset-shift further causes suboptimal cognitive tuning and information processing,[169] thereby attenuating the volitional bias of the implemental mindset.[164] This can lead to reduced task performance[163,164,170] as well as diminished psychological well-being and ill-health.[171–174]

Supportive neuroscientific evidence for the construct of an action crisis has been provided by Herrmann et al.,[175] who showed that individuals experiencing an action crisis have increased resting state functional connectivity between fronto-polar cortex and nucleus accumbens, while effective goal-striving has been related to decreased fronto-accumbal connectivity.[77] In addition, self-regulatory fatigue has been proposed to be the consequence of an imbalance between volitional 'top-down' control and 'bottom-up' motivational hedonic impulses.[175,176] Goal-disengagement might thus be the result of lateral prefrontal impairment, heightened activity in subcortical limbic structures such as the amygdala, or a combination thereof.[177–179]

Similarly, but more specific with regard to mental fatigue, Tops et al.[180,181] proposed a framework based on expected rewards and metabolic costs suggesting that an effort-reward imbalance leads to protective inhibition of self-regulation and motivation in further goal-pursuit. With regard to physical fatigue, Tanaka and Watanabe[182] provided neural substrates for two partially overlapping circuits suggesting that motor output is regulated by the balance between afferent feedback activating the inhibition system and motivational factors activating the facilitation system. Both models have in common that cortico-basal ganglia-thalamo-cortical reward circuits can facilitate goal-directed behaviour when homeostasis is challenged but maintained.[183] However, when homeostasis is threatened, the relative contribution of the powerful motivational drive of the limbic system signalling to 'disengage from further goal-pursuit' will oppose and ultimately overcome the relative contribution of the volitional prefrontal cortex drive to 'engage in further goal-pursuit', thereby protecting from homeostatic failure. The teleological function and evolutionary value of an action crisis may therefore be to

cause self-regulatory fatigue, thereby preventing too great a homeostatic disturbance and catastrophic biological failure.[184]

In conclusion, it is proposed that experiencing an action crisis in pursuit of a goal can negatively impact on self-regulation processes and goal-directed behaviour, thereby further aggravating the intra-psychic conflict in a vicious circle. It is further hypothesised that self-regulatory fatigue is related to a shift in the relative contribution of volitional control driving further goal-pursuit and sensory as well as affective impulses motivating goal-disengagement within a given situational context in psychobiological regulation. Accordingly, it is suggested that the shift from an implemental to a deliberative mindset characteristic of an action crisis appropriately represents the cognitive-evaluative dimension of perceived fatigability.

The Transactional Psychobiological Nature of the Endocrinological Distress Response

Lastly, Melzack[70] postulated "that the sequences of behaviour that characterise pain are determined by sensory, motivational, and cognitive processes acting on motor…and response mechanisms in the hypothalamus…" (p. 434). Pain was therefore suggested to impact on the hypothalamo-pituitary-adrenal axis (HPAA).

Acevedo and Ekkekakis[185] proposed an integrative framework for the transactional psychobiological nature of cognitive appraisals in the endocrinological distress response to stressful events that includes physiological, affective, and cognitive components. The core assumption is that the continuous cognitive appraisal of the perception of the psychophysiological demands can "dramatically alter the intensity and the emotional concomitants of the ensuing physiological activation" (p. 48).[185] Preliminary evidence linking specifically the construct of an action crisis to the psycho-neuro-endocrinological distress response comes from a study by Brandstätter et al.,[164] who found exacerbated salivary cortisol concentrations during a marathon in runners experiencing an action crisis two weeks prior to the race and after controlling for physiological distress. Perceived action crisis and dynamic change in salivary cortisol concentrations predicted slower marathon times, and salivary cortisol slope was a significant mediator in the relationship between action crisis and running performance. This suggests that an action crisis acts on the HPAA causing an exacerbated

endocrinological distress response and thus a debilitative psychophysiological milieu not conducive to exercise performance.

CONCLUSION

Given the reliance of current models of exercise regulation on the construct of perceived exertion and the current debate of what the RPE construct measures, it is suggested to progress towards a comprehensive multidimensional concept of perceived fatigability differentiating between the distinct inputs of sensory, affective, and cognitive processes that shape the perceptual milieu during exercise. A three-dimensional framework is proposed to investigate centrally regulated and goal-directed exercise behaviour. It is further suggested that: perceived mental strain and perceived physical strain forming part of the reafference principle appropriately represent the sensory-discriminatory dimension, the construct of core affect and its underlying two dimensions valence and arousal appropriately represents the affective-motivational dimension, and the shift from an implemental to a deliberative mindset associated with an action crisis appropriately represents the cognitive-evaluative dimension of perceived fatigability. When combined with a dynamical systems approach, the framework has the potential to enrich theory development in fatigue research as well as provides an unbiased practical framework for investigating the complex interplay between the psychophysiological determinants of pacing and performance during prolonged endurance exercise.

Reiterating the call from Frank E. Marino:[186] “While the nature of modern research often requires progressively greater specialisation and refinement of theories, opportunities must be taken to discuss fatigue from a holistic perspective so that we can integrate different components of fatigue into a modern understanding” (p. 66). This has the obvious potential to make fatigue research much more ‘fuzzy’ and much less ‘mechanical’, but it might be exactly this fuzzy approach that will allow us to gain a clearer understanding of the mechanisms underpinning centrally regulated and goal-directed exercise behaviour.

The subsequently conducted research studies are aimed at investigating the dynamic interdependencies between the three dimensions and their differential predictive role in observed pacing behaviour, performance, and markers of physiological distress. The

complex interplay between these multiple variables is tested under stressful psychological (head-to-head competition against a performance matched competitor) and physiological (running with isolated locomotor muscle fatigue and exercise-induced muscle damage) conditions that are relevant for ‘real-world’ endurance performance. Research studies further address the influence of moderating variables such as performance level and sex. In addition, formal tests of the direction and temporal precedence of hypothesised cause-effect relationships as well as the integrity of the hypothesised model structure in relation to observed data will be conducted and discussed.

CHAPTER TWO

THE PSYCHOPHYSIOLOGICAL DETERMINANTS OF PACING AND PERFORMANCE DURING PROLONGED ENDURANCE EXERCISE: A PERFORMANCE LEVEL AND COMPETITION OUTCOME COMPARISON

ABSTRACT

A three-dimensional framework of centrally regulated and goal-directed exercise behaviour emphasised the integration of distinct sensory-discriminatory, affective-motivational, and cognitive-evaluative dimensions that underpin perceived fatigability. This study aimed to capture the complex interdependencies and temporal dynamics in these processes, their interrelations with observed pacing behaviour, performance, and biochemical variables as well as their performance level and competition outcome dependent variances.

Twenty-three cyclists of distinct endurance performance level categories engaged in individual and head-to-head competition time trials against a performance matched opponent. Sensory, affective, and cognitive processes were respectively assessed with the constructs perceived physical strain and perceived mental strain, valence and felt arousal underpinning core affect, and action crisis characterised by a shift from an implemental to a deliberative mindset.

Performance level and competition outcome dependent variances and differential temporal dynamics in constructs were associated with alterations in pacing behaviour, performance, and physiological disturbance. Perceived physical and mental strain were primarily associated with observed pacing behaviour as necessary to align planned behaviour with current physiological state. Valence and arousal were primarily associated with differential responses in performance regulation. The mindset-shift associated with an action crisis was primarily associated with non-adaptive psycho-neuro-endocrinological distress response.

The proposed constructs are interdependent in a non-linear dynamic fashion, context-dependent, constraint-based, distinguishable by well-trained cyclists, and interrelated with observed pacing behaviour, performance, and physiological disturbance. The proposed framework provides a more comprehensive alternative to the Gestalt

concept of perceived exertion and more completely accounts for centrally regulated and goal-directed exercise behaviour.

INTRODUCTION

Exercise scientists and clinicians readily agree that fatigue is a multifactorial, interactive, and complex phenomenon.[187] However, progressive specialisation and multi-disciplinary approaches in the exercise sciences have led to fragmentation[188] as well as reductionism and dichotomies in models and methodologies. This consequently constrains the pursuit of a holistic understanding of fatigue.[186]

In line with the advent of integrative psychophysiological models of exercise regulation,[23,30,31,36,37,66] Enoka and Duchateau[26] proposed a unified taxonomy in the investigation of fatigue and its impact on human performance. Specifically, the authors emphasised the importance of interactions in modulating factors within and between two collective variables: performance fatigability and perceived fatigability. Thus, a deeper understanding of the limits of human performance and the fatigue process can only be gained by investigating the complex interdependence of homeostatic, psychological, and behavioural processes, and their interrelation with objective measures of performance.

Critically, the phenomenological experience of fatigue (qualia) and altered behaviour in response to exercise-induced fatigue are not the result of mechanistic linear interactions. Instead they are context-dependent and emerge from dynamic non-linear as well as circular bottom-up and top-down effects between molecular, organismic, and social levels.[189] Dynamical systems theory provides an opportunity to discuss the socio-psycho-biological fatigue phenomenon from a level- and model-independent perspective.[190] It is therefore particularly suited to investigate the psychophysiological determinants of pacing and performance under changing constraints imposed by fatigue.[191]

A non-linear dynamic model of attention focus has been proposed allowing a comprehensive understanding of thought dynamics during exercise to volitional exhaustion.[192] Under increasing constraints (time on task, exercise intensity, and accumulated exertion), metastable (i.e. spontaneous alternation between possible states) task-unrelated (TUT) and task-related (TRT) thought states emerge, which

eventually involuntarily stabilise on internal TRT (body monitoring, feeling and affect, and command and instruction).[193,194] Thus, exercise-induced alterations in observed pacing behaviour and performance are preceded by an inner dialogue between volition urging further goal-pursuit and fatigue urging goal-disengagement.[195] This self-regulatory process is fatigable itself and thus temporally limited,[196] eventually leading to the dissolution of the pursued goal.[197] Critically, trained individuals can sustain this volitional process longer than untrained individuals indicative of performance level dependent variances.[198]

In chapter one,[199] a three-dimensional framework of centrally regulated and goal-directed exercise behaviour was proposed, which emphasises the integration of distinct sensory-discriminatory, affective-motivational, and cognitive-evaluative processes that underpin perceived fatigability during exercise. It therefore provides a more comprehensive alternative to the related but independent Gestalt concept of perceived exertion, which is almost exclusively the sole psychophysiological construct used to explain fatigue-induced alterations in observed pacing behaviour and performance.

The review article concluded with the proposal and hypotheses that: (A) the constructs of perceived mental strain (i.e. the subjective task difficulty and effort expended) and perceived physical strain (i.e. the physical sensations caused by the task) appropriately represent the sensory-discriminatory dimension and play primary roles in the regulation of pacing behaviour, (B) the construct of core affect with its underlying two dimensions valence and arousal appropriately represents the affective-motivational dimension and plays a primary and mediatory role in the regulation of performance, (C) the mindset-shift associated with an action crisis appropriately represents the cognitive-evaluative dimension and plays a critical role in volitional self-regulatory control and the psycho-neuro-endocrinological distress response, and (D) the proposed three-dimensional framework provides a more comprehensive alternative to the Gestalt concept of perceived exertion and more completely accounts for dynamic changes in observed pacing behaviour and performance.

The aim of this research study was to capture the complex interdependencies and temporal dynamics of the above described constructs as well as their interrelations. It was hypothesised that alterations in perceived fatigability would be related to

alterations in observed pacing behaviour, performance, and biochemical variables in a systematic fashion and temporal order as outlined in detail in the preceding chapter. It further investigated performance level and competition outcome dependent variances during (A) a maximal self-paced 70-km individual bicycle time trial over a simulated profiled course and (B) under the constraint of a head-to-head competition against a performance matched opponent.

METHODS

Participants

Twenty-three cyclists, 11 at performance level 4 (PL4) and 12 at performance level 3 (PL3) were recruited for this study. The performance level categorisation of each participant was estimated based on race results and seed ranking by the local race event organiser, and verified according to their peak power output (PPO) and VO_{2peak} values as described in detail by DePauw et al.[200] Sample size estimation using G*Power 3 software (G*Power, Version 3.1.7, Kiel, Germany) and magnitude based inferences[201] indicated ten participants per group would be needed for performance level and competition outcome comparisons, respectively. All participants provided prior written informed consent to the procedures used in this study, which were approved by the institutional ethics committee and carried out in accordance with the Declaration of Helsinki**.**

Study Design

A between-group experimental research design was used. Participants completed four 70-km bicycle time trials at weekly to biweekly intervals. The first time trial took place after preliminary testing and acted as a submaximal familiarisation time trial. Before returning for their second visit participants were asked to log their training and diet for 48 hours prior to the baseline time trial ($BASE_{TT}$) and to prepare in a way that resembled their routine before an important race. They were then advised to repeat this ‘mini taper’ for the following experimental time trials. Participants then completed an individual time trial ($INDV_{TT}$) and a head-to-head competition time trial ($COMP_{TT}$) against a performance-matched opponent in a counterbalanced AB/BA crossover design.

Procedures

Preliminary Procedures

During the first laboratory attendance each participant had their age, stature, and body mass recorded. Body fat percentage was estimated using the seven skinfold method.[202]

An incremental cycling test with peak oxygen consumption (VO_{2peak}) was performed on an electronically braked Computrainer™ Pro cycle ergometer (RacerMate Inc., Seattle, USA) which was calibrated using a standardised protocol before and after a 15 min warm-up.[203] Starting flywheel resistance was set at 100 W and was continuously increased at a rate of 20 $W \cdot min^{-1}$ until volitional exhaustion, indicated by participant's inability to sustain a cadence of 60 rpm despite verbal encouragement. PPO was calculated by averaging the power output for the final minute of the test. During the test, breath-by-breath measurements of ventilation volume (V_E), oxygen uptake (VO_2) and carbon dioxide production (VCO_2) were recorded using an Oxycon™ Pro (Viasis, Hoechberg, Germany) calibrated in accordance with the manufacturer's instructions. Peak oxygen uptake was determined as the highest recorded VO_2 measurement averaged over 30 seconds and the first ventilatory threshold was determined according to methods by Gaskill et al.[204]

Time Trial Procedure

After a self-selected 15-min pre-race warm-up routine, participants completed each of the four 70-km time trials on their own bicycle mounted on a Computainer™ Pro cycle ergometer under stable climatic conditions (Temperature: 20.7 ± 1.1°C; Humidity: 38.8 ± 5.6%). Only course profile, gradient, and distance covered were displayed to participants during each time trial. Participants were told to complete the distance in the shortest possible time, but no verbal encouragement was given during the time trial itself. The time trial course included two simulated climbs of 7-km length at a gradient of 5% starting at 21 and 49 km, respectively, followed immediately by a downhill section of equal length and gradient. The gradients were restricted to 5% after extensive pilot testing to ascertain that the fastest and slowest participants had adequate gears to use during the downhill and uphill sections, respectively. A 3D-landscape of the 70-km time trial course was projected onto a screen in front of the

participants. A fan was placed 1.5 m in front of the participants and the fan level was adjusted in accordance with the course profile to partly simulate speed-related alterations in peripheral cooling. During the uphill sections fans were set on level one, during the flat sections on level two and during the downhill sections on level three, creating average wind speeds of 3.15, 3.80, and 4.25 $m \cdot s^{-1}$, respectively.

Participants were permitted to consume water ad libitum and a commercially available carbohydrate drink (Enduren™ Endurance Energy Drink) was given at a rate of 60 $g \cdot h^{-1}$. All experimental time trials commenced at 8 am to control for diurnal variations.

During each time trial, power output, cadence, speed, and distance covered were recorded and stored continuously at 34 Hz using Computrainer™ Coaching Software v1.6.432 (RacerMate Inc., Seattle, USA). Heart rate was recorded at two second intervals throughout each trial by means of a Suunto® T6 Heart Rate Monitor (Suunto Oy, Vantaa, Finland). Performance and heart rate data were subsequently analysed with TrainingPeaks™ analysis software (WKO edition+, Version 3.0, Lafayette, CO, USA). All continuously captured data were averaged into 7-km bins before statistical analyses were performed.

Finishing times of the maximal $BASE_{TT}$ were used to match participants for the $COMP_{TT}$. A cut-off criterion of maximum 2% time difference was used as this performance variance is unexplained by physiological processes.[205] Since it was impossible to find only exclusively matched pairs of two using this strict criteria, it was necessary for five participants to complete an additional $COMP_{TT}$ (Two won both trials, one lost both trials, and two won and lost one trial each). Eventually, 23 participants completed the study and an overall of 14 head-to-head competitions were investigated.

The matched participants performed the $COMP_{TT}$ in pairs of two next to each other in the bicycle laboratory. Each rider's position and progression over the simulated course, gradient, distance covered, and distance between competitors were projected onto the split screen in front of the two participants. Prior to the test, participants were given the primary goal to win the competition and the secondary goal to beat their own previous personal best. No verbal encouragement was given during the time trial itself. Care was taken to ensure that participants did not know each other personally. Thus, both

competitors were placed in a competitive environment under challenging conditions with uncertain outcome, closely resembling the defining characteristics of 'real-world' competition.

Measurements

Perceptual Measures

During each time trial, participants provided ratings on the following four single-item scales presented in random order at 7-km intervals.

The 15-point (6-20) Borg scale with modified instructions to include only the subjective perception of physical sensations *caused by* the task was used to approximate perceived physical strain. Participants were instructed to focus on location, quality, and intensity of central (i.e. cardiorespiratory), peripheral (i.e. muscles and joints), and other (e.g. thermal) physical sensations before returning an overall perceived physical strain score.

The Task Effort and Awareness scale (TEA) was used to approximate perceived mental strain and administered in accordance with published guidelines.[57] Participants were instructed to focus only on the subjective awareness of the mental effort *invested into / required to continue with* the task before returning a perceived mental strain score.

A three-tiered justification process for measurement selection in affect research has been recommended.[144,145] Firstly, the construct of core affect was chosen as it encompasses the global domain of affective states in a more holistic fashion than mood and emotion, and because distinct affective states during maximal self-paced $INDV_{TTs}$ and head-to-head competitions cannot be predicted. Secondly, from the available conceptual models of core affect, the circumplex model of affect was chosen, as it allows the independent assessment of the two orthogonal and bipolar dimensions valence and arousal.[206] Thirdly, the independence and bipolarity of the two dimensions have been psychometrically tested and confirmed.[207] To investigate alterations in exercise behaviour it is paramount to measure these variables repeatedly over time, whilst interfering as little as possible with the athlete's focal state. Thus, the 11-point (--5 'very bad' to 0 'neutral' to +5 'very good') Feeling Scale (FS) and the 6-point (1 'low

arousal' to 6 'high arousal') Felt Arousal Scale (FAS) were chosen to approximate dynamic changes in valence and arousal, respectively.[38,208]

The Action Crisis Scale (ACRISS) was administered during the 30-min recovery period to retrospectively measure the extent to which participants experienced an intra-psychic conflict between further goal-pursuit and goal-disengagement.[174] ACRISS comprises six items: conflict, setbacks, implemental disorientation, rumination, disengagement impulses and procrastination. All items were rated on a 5-point Likert type scale ranging from 1 'no agreement' to 5 'very much agreement'. The ACRISS was administered for seven sections of the profiled TT course (3 flat, 2 uphill, and 2 downhill). The internal consistency estimate of reliability was acceptable with mean Cronbach´s alpha of 0.77 ± 0.07.

Haematological Measures

Prior to the $INDV_{TT}$ and $COMP_{TT}$, a superficial antecubital forearm vein was cannulated by a qualified physician. Samples were taken for assessment of blood concentrations in: (A) cortisol and norepinephrine at rest, at the start of each TT, every 14-km during each TT, at the end of each TT, and every 10 min during the 30-min recovery period, (B) lactate at each 14-km interval during each TT, and (C) haemoglobin and haematocrit at rest, at the finish, and after 30-min of recovery. The degree of haemoconcentration was calculated and all blood samples were subsequently corrected for plasma volume changes.[209]

Blood samples were placed into four different pre-chilled Vacutainers, respectively containing: (A) Lithium-Heparin for the analysis of norepinephrine, (B) serum clot activator for the measurement of cortisol, (C) potassium oxalate and sodium fluoride for the measurement of lactate concentrations, and (D) K_2-EDTA for the measurement of haemoglobin and haematocrit. All samples were inverted 5 times and kept on ice until processed further. Blood samples were then centrifuged at 3000 rpm for 10 min, the plasma/serum pipetted off and stored at -80°C for subsequent analysis.

Plasma catecholamine concentrations were determined using the Waters High Pressure Liquid Chromatography (HPLC) system (Waters®, Milford, MA, USA) based on the method by McDonald et al.[210] Chromatographic data were captured using Waters Breeze software. Serum cortisol concentrations were analysed using an

automated chemiluminescence system (ADVIA Centaur®, Siemens Health Diagnostics, Munich, Germany). Plasma lactate concentrations were determined using glucose oxidase method (YSI 2300 STAT PLUS, Ohio, USA). Haematocrit and haemoglobin were determined by Metropolis Pathology Laboratories using conventional methods.

An overall of respective 2.78% and 4.14% of all blood samples taken during the $INDV_{TT}$ and $COMP_{TT}$ were missing due to blood clotting, insufficient sample, or measurement errors. Missing data points were considered missing completely at random and thus replaced by means of multiple imputation procedure.[211]

Statistical Analysis

Parameters collected during the preliminary procedures were compared using independent samples t-tests between performance level groups, and paired samples t-tests between winners and losers of the head-to-head competition. Between-group effect sizes were calculated using Cohen's d (trivial <0.20, small 0.21– 0.60, moderate 0.61–1.20, large 1.21–2.00, very large 2.01–4.00 and near perfect >4.00). Confidence intervals and magnitude based inferences for meaningful differences between time trial performances were derived from p-values in accordance with Hopkins.[212] A two-way mixed ANOVA with repeated measures was used to compare changes in parameters over time between performance level and competition outcome groups. A Greenhouse-Geisser epsilon adjustment was made when sphericity was violated. Aligned rank transformation procedure (ARTool software 1.5.1, Washington, USA) was used to perform non-parametric factorial analysis on all perceptive data.[213] All ANOVA effect sizes were calculated as partial eta squared (η_p^2). All data are presented as mean ± one standard deviation and an alpha level of <0.05 (two-tailed) was used to indicate statistical significance.

RESULTS

Part One: Performance Level Comparison

Participant Characteristics

Anthropometric characteristics and performance parameters of participants (n=23) are listed in table 2.

Table 2: Performance level comparison of anthropometric and performance data.

	PL4 (n=11)	PL3 (n=12)	p-value	Cohen´s d
Descriptive data				
Age (years)	34 ± 8.0	36 ± 6	n.s.	-
Stature (cm)	179 ± 6.0	180 ± 5	n.s.	-
Body mass (kg)	72.2 ± 7.3	75.3 ± 6.8	n.s.	-
Percentage body fat (%)	8.1 ± 2.3	10.3 ± 2.7	0.002	0.89
Training time ($h \cdot wk^{-1}$)	10.9 ± 1.7	8 ± 2	0.02	1.48
Performance test data				
abs PPO (W)	396 ± 36	359 ± 31	0.013	1.14
rel PPO ($W \cdot kg^{-1}$)	5.5 ± 0.3	4.8 ± 0.1	<0.001	3.52
abs VO_{2peak} ($L \cdot min^{-1}$)	4.71 ± 0.49	4.22 ± 0.31	0.008	1.23
rel VO_{2peak} ($ml \cdot min^{-1} \cdot kg^{-1}$)	65.3 ± 3.2	56.3 ± 4.4	<0.001	2.37
abs PO @ VT-1 (W)	275 ± 24	254 ± 23	0.038	0.93
rel PO @ VT-1 (W·kg-1)	3.8 ± 0.3	3.4 ± 0.1	<0.001	1.05
70 km TT time				
$INDV_{TT}$ time (h:min:s)	2:06:35 ± 0:05:43	2:16:52 ± 0:09:00	0.004	1.40

Abbreviations: abs = absolute; rel = relative; PPO = peak power output; VO_{2peak} = peak oxygen consumption; VT-1 = First ventilatory threshold; $INDV_{TT}$ = Individual time trial; Data represented as mean ± SD.

Time Trial Times

Times taken to complete the $INDV_{TT}$ were 2h:06min:35s ± 0h:05min:43s and 2h:16min:52s ± 0h:09min:00s for PL4 and PL3 cyclists, respectively. The average time difference was 7.52%. The mechanistic inference for this difference between performance level groups was very likely substantially positive (98.1% very likely substantially positive, 1.9% very unlikely trivial and 0.0% most unlikely substantially negative).

Performance Data

Power, power to weight, and power to VT-1

There was no significant time × group interaction effect for absolute power output ($F_{3.0, 63.5}$=0.2; p=0.873; η_p^2=0.013). However, absolute power output was significantly higher in PL4 cyclists throughout the $INDV_{TT}$ (main group effect: $F_{1, 21}$=7.0; p=0.015; η_p^2=0.251). There further was a significant main time effect with lower absolute power output during the downhill sections ($F_{3.0, 63.5}$=51.9; p<0.001; η_p^2=0.712). (See figure 4 a)).

There was no significant time × group interaction effect for relative power output ($F_{3.0, 62.7}$=0.2; p=0.811; η_p^2=0.015). However, relative power output was significantly greater in PL4 cyclists throughout the $INDV_{TT}$ and the main group effect was stronger when compared to absolute power output ($F_{1, 21}$=30.6; p<0.001; η_p^2=0.593). There also was a significant main time effect with lower relative power output during the downhill sections ($F_{3.0, 62.7}$=53.5; p<0.001; η_p^2=0.718). (Not shown).

PL4 cyclists not only produced absolute and relative higher power outputs, but also performed at a significantly higher relative exercise intensity when compared to the first ventilatory threshold (main group effect: $F_{1, 21}$=4.7; p=0.042; η_p^2=0.182). In line with absolute and relative power output, relative exercise intensity was significantly lower during the downhill sections (main time effect: $F_{3.1, 64.7}$=55.5; p<0.001; η_p^2=0.725). No significant time × group interaction effect was observed ($F_{3.1, 64.7}$=0.1; p=0.967; η_p^2=0.004). (See figure 4 b)).

Split time

There was a significant time × group interaction effect for 7-km interval split times ($F_{2.0, 41.2}$=9.3; p=0.001; η_p^2=0.306). The investigation of simple (main) group effects showed that PL4 cyclists were significantly faster during all flat and uphill sections ($F_{1, 21}$>5.7; p<0.026; η_p^2>0.214). However, there were no significant split time differences during the downhill sections ($F_{1, 21}$<3.3; p>0.083; η_p^2<0.136). Split times

changed significantly over time and according to course profile in both performance level groups with simple (main) time effects ($F_{1.5, 15.2}$=661.7; p<0.001; η_p^2=0.985) and ($F_{2.0, 21.5}$=473.2; p<0.001; η_p^2=0.977) for PL4 and PL3, respectively. (See figure 4 c)).

Heart rate

There was neither a significant time × group interaction effect for heart rate ($F_{2.0, 41.8}$=0.2; p=0.848; η_p^2=0.008), nor a significant main group effect ($F_{1, 21}$=0.2; p=0.662; η_p^2=0.009). However, there was a significant main time effect in accordance with course profile ($F_{2.0, 41.8}$=20.4; p<0.001; η_p^2=0.492). (Not shown).

Cadence

There was neither a significant time × group interaction effect for cadence ($F_{3.8, 78.9}$=0.4; p=0.809; η_p^2=0.018), nor a significant main group effect ($F_{1, 21}$=0.4; p=0.554; η_p^2=0.017). However, there was a significant main time effect with lower cadences during the uphill sections and higher cadences at the start and during the downhill sections ($F_{3.8, 78.9}$=33.3; p<0.001; η_p^2=0.614). (Not shown).

Perceptual Data

All perceptive data were additionally analysed with a non-parametric factorial procedure using common analysis of variance after align rank transform (ART). The results are provided in squared brackets after the parametric test results in parenthesis.

Perceived Physical Strain (p-RPE)

A significant time × group interaction effect for p-RPE was observed, when a two-way mixed ANOVA with repeated measures was applied ($F_{4.2, 87.9}$=3.1; p=0.018; η_p^2=0.129). A non-parametric factorial procedure (ART) using common analysis of variance after align rank transform, provided supporting results [$F_{4.2, 88.2}$=2.9; p=0.024; η_p^2=0.122]. Investigation of simple (main) group effects showed that there were no significant differences between performance level groups during all flat and uphill sections ($F_{1, 21}$<1.0; p>0.326; η_p^2<0.046). However, there was a significant difference in p-RPE with PL4 cyclists scoring significantly lower during the downhill sections

($F_{1, 21}$>5.5; p<0.028; η_p^2>0.209). Ratings of p-RPE increased significantly over time and according to course profile in both performance level groups with simple (main) time effects of ($F_{3.2, 32.0}$=40.6; p<0.001; η_p^2=0.802) and ($F_{3.4, 37.7}$=28.1; p<0.001; η_p^2 =0.719) for PL4 and PL3, respectively. (See figure 4 d)).

Perceived Mental Strain (TEA)

Neither a time × group interaction effect ($F_{9.189}$=0.9; p=0.553; η_p^2=0.040) [ART: $F_{9, 189}$=1.4; p=0.188; η_p^2=0.063], nor a main group effect ($F_{1, 21}$=0.6; p=0.461; η_p^2=0.026) [ART: $F_{9, 189}$=0.4; p=0.540; η_p^2=0.018] were found for TEA scores. However, there was a significant main time effect with a steady increase in TEA scores over time and lower scores during the downhill sections ($F_{9, 189}$=32.1; p<0.001; η_p^2 =0.605) [ART: $F_{9, 189}$=27.5; p<0.001; η_p^2=0.567]. (See figure 4 e)).

Valence (FS)

No significant time × group interaction effect was observed for hedonicity ($F_{9, 189}$=0.7; p=0.560; η_p^2=0.033) [ART: $F_{3.7, 77.1}$=0.8; p=0.504; η_p^2=0.038]. However, there was a significant main group effect with PL4 cyclists experiencing greater pleasure throughout the trial and better maintenance of positive hedonic tone than PL3 cyclists, who perceived progressively less pleasure in the last third of the time trial course ($F_{1, 21}$=4.5; p=0.045; η_p^2=0.178) [ART: $F_{1, 21}$=5.4; p=0.031; η_p^2=0.203]. Parametric mixed ANOVA revealed a significant main effect for time ($F_{3.4, 71.6}$=4.0; p=0.008; η_p^2 =0.159), which was not confirmed by non-parametric factor analysis [ART: $F_{3.7, 77.7}$=1.9; p=0.112; η_p^2=0.086]. (See figure 4 f)).

Felt Activation (FAS)

A significant time × group interaction effect for felt activation was observed ($F_{5.7, 119.9}$=2.6; p=0.022; η_p^2=0.111) [ART: $F_{5.2, 110.2}$=2.8; p=0.019; η_p^2=0.117]. Investigation of simple (main) group effects showed that there were no significant differences between performance level groups during the first 42 km of the course ($F_{1, 21}$<1.1; p>0.316; η_p^2<0.048). However, after approaching significance at km 49

($F_{1, 21}$=4.2; p=0.054; η_p^2=0.166), there was a significant difference in felt activation with PL3 cyclists scoring significantly higher during the remainder of the course ($F_{1, 21}$>5.4; p<0.030; η_p^2>0.204). Felt activation changed significantly over time in both performance level groups with simple (main) time effects of ($F_{4.7, 46.9}$=6.8; p<0.001; η_p^2 =0.406) and ($F_{9, 99}$=15.3; p<0.001; η_p^2=0.581) for PL4 and PL3, respectively. (See figure 4 g)).

Action Crisis (ACRISS)

There was neither a significant time × group interaction effect for action crisis ($F_{3.8, 79.0}$=0.7; p=0.578; η_p^2=0.033) [ART: $F_{9, 189}$=0.4; p=0.883; η_p^2=0.018], nor a significant main group effect in either group ($F_{1, 21}$=0.1; p=0.770; η_p^2=0.004) [ART: $F_{1, 21}$=0.0; p=0.903; η_p^2=0.001]. Also, no significant main time effect was found in either group ($F_{3.8, 79.0}$=1.3; p=0.270; η_p^2=0.059) [ART: $F_{9, 189}$=1.2; p=0.288; η_p^2 =0.056]. (See figure 4 h)).

Haematological Data

Lactate

There was no significant time × group interaction effect for blood lactate concentrations ($F_{2.2, 45.3}$=0.7; p=0.498; η_p^2=0.034). However, in line with PL4 cyclists performing at higher relative exercise intensities, there was a significant main group effect with PL4 cyclists having higher blood lactate concentrations throughout the $INDV_{TT}$ ($F_{1, 21}$=4.8; p=0.039; η_p^2=0.187). There also was a significant main time effect with the lowest blood lactate concentrations measured in the middle flat section and the highest lactate values reached at the finish ($F_{2.2, 45.3}$=16.5; p<0.001; η_p^2=0.440). (See figure 4 i)).

Norepinephrine

There was a trend for a significant time × group interaction effect ($F_{3.6, 76.2}$=2.1; p=0.094; η_p^2=0.091), as well as for a significant main group effect for blood

norepinephrine concentrations ($F_{1, 21}$=3.0; p=0.097; η_p^2=0.125), with PL4 cyclists having higher blood norepinephrine concentrations throughout the trial, but not during the pre and post-trial periods. Comparing the areas under the curve (AUC) between the performance level groups with an independent t-test showed a significant group difference (t=2.1; p=0.045; d=0.89). The main time effect reached a statistically significance level ($F_{3.6, 76.2}$=2.2; p<0.001; η_p^2=0.646) with a sharp logarithmic increase to a plateau and quick recovery. (See figure 4 j)).

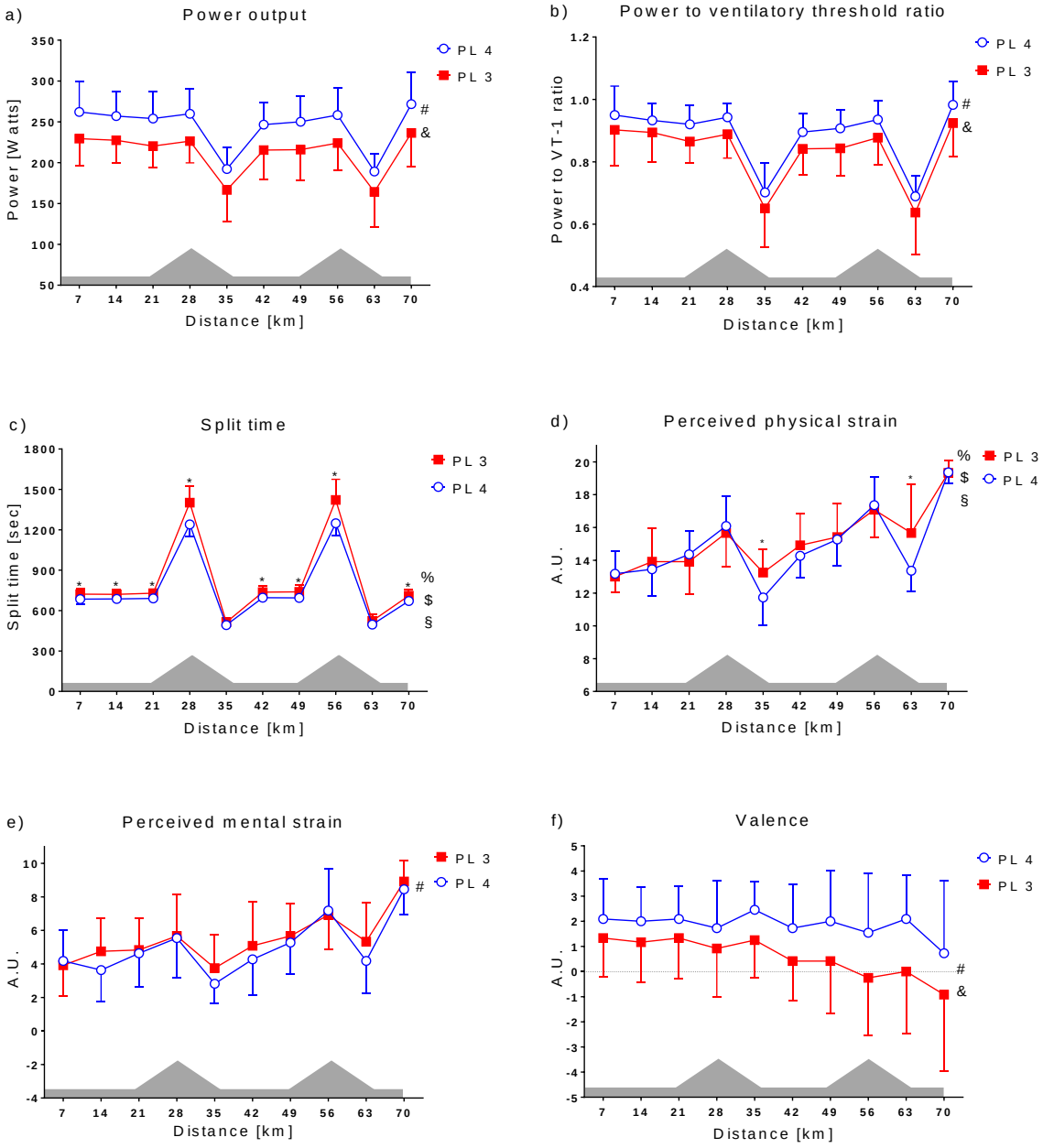
Cortisol

Neither a significant time × group interaction effect for blood cortisol concentrations ($F_{1.8, 38.9}$=0.4; p=0.618; η_p^2=0.021), nor a significant main group effect were observed ($F_{1, 21}$=0.2; p=0.681; η_p^2=0.008). However, there was a significant main time effect ($F_{1.8,}$ 38.9=2.2; p<0.001; η_p^2=0.544) with blood cortisol concentrations continuously rising and peaking at 20 to 30 min into the recovery period. (See figure 4 k)).

Glucose

Neither a significant time × group interaction effect ($F_{2.5, 52.2}$=2.2; p=0.107; η_p^2=0.096), nor main group effect ($F_{1, 21}$=0.0; p=0.876; η_p^2=0.001) for blood glucose concentrations was found. Nevertheless, there was a significant main time effect with an increase in blood glucose concentrations towards the finish line ($F_{2.5, 52.2}$=8.8; p<0.001; η_p^2=0.295). Euglycaemia was maintained throughout the trial. (Not shown).

A summary of graphical depictions of performance, perceptive, and haematological data for performance level comparisons is provided in figure 4.



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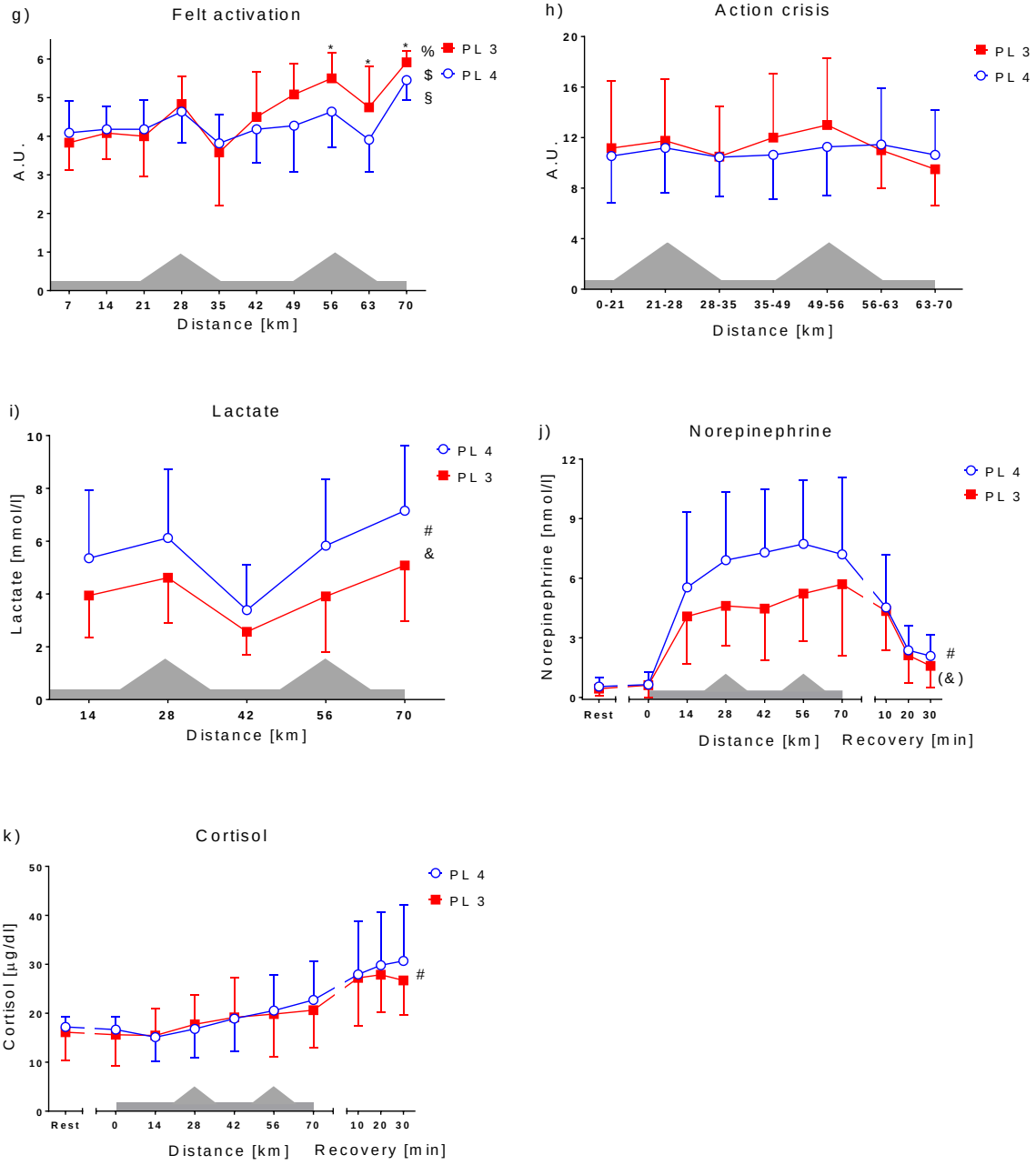


Figure 4: Performance level comparison of performance, perceptive, and haematological data.

Note the differences in the x-axes of action crisis and haematological parameters due to different sampling times.

Abbreviations: # = main time effect; & = main group effect; (&) = group effect, when comparing areas under the curve with an independent t-test; % = time x group interaction effect; $ = simple (main) time effect for winners; § = simple (main) time effect for losers; * = simple (main) group effect

Correlations

Zero-order correlations among the major study variables for performance level comparison are provided in table 3.

Table 3: Zero-order correlations between classical predictors of endurance performance, slopes of major study variables, and individual time trial time.

	1	2	3	4	5	6	7	8
1. abs PPO	-							
2. rel PPO	-.46*	-						
3. abs VO_{2peak}	-.78**	.36	-					
4. rel VO_{2peak}	-.20	.76**	.54**	-				
5. p-RPE slope	-.25	-.25	-.03	.06	-			
6. TEA slope	-.17	-.04	-.30	-.18	-.58**	-		
7. FAS slope	-.44*	-.49*	-.44*	-.45*	.51*	.44*	-	
8. Cortisol slope	.19	-.21	.28	-.08	.56**	.13	.08	-
9. $INDV_{TT}$ time	-.88**	-.55**	-.71**	-.35	-.35	.28	.50*	-.12

Abbreviations: abs = absolute; rel = relative; PPO = peak power output; p-RPE = physical-Rating of perceived exertion; TEA = Task effort and awareness; FAS = Felt arousal scale; $INDV_{TT}$ =Individual time trial.
** Correlation is significant at the 0.01 level (2-tailed). * Correlation is significant at the 0.05 level (2-tailed).

Summary of Performance, Perceptive, and Haematological Data

A summary of statistical findings in main performance, perceptive, and haematological variables for performance level comparison is provided in table 4.

Table 4: Summary of performance level comparison in performance, perceptive, and haematological variables.

	Time x Group Interaction			Group Main Effect			Time Main Effect			Comment
	F	*p*	h_p^2	*F*	*p*	h_p^2	*F*	*p*	h_p^2	
Performance data										
Power	0.2	0.873	0.01	7.0	0.015	0.25	51.9	<0.001	0.71	PL4 > PL3; Change with course profile
Power to weight	0.2	0.811	0.02	30.6	<0.001	0.59	53.5	<0.001	0.72	PL4 > PL3; Change with course profile
Power to VT-1	0.1	0.967	0.00	4.7	0.042	0.18	55.5	<0.001	0.73	PL4 > PL3; Change with course profile
Split time	9.3	0.001	0.31		N/A			N/A		PL4 faster in flat/uphill, but not downhill sections
Perceptual data										
p-RPE	3.1	0.018	0.13		N/A			N/A		PL4 lower in downhills, but not flat/uphill sections
TEA	0.9	0.553	0.04	0.6	0.461	0.03	32.1	<0.001	0.61	Scalar increase and change with course profile
FS	0.7	0.560	0.03	4.5	0.045	0.18	4.0	0.008	0.16	PL4 > PL3; Decline towards finish
FAS	2.6	0.022	0.11		N/A			N/A		PL3 greater arousal from second climb onwards
ACRISS	0.7	0.578	0.03	0.1	0.77	<0.001	1.3	0.27	0.06	
Haematological data										
Lactate	0.7	0.498	0.03	4.8	0.039	0.19	16.5	<0.001	0.44	PL4 > PL3; Decrease in middle flat section
Cortisol	0.4	0.618	0.02	0.2	0.681	0.01	38.9	<0.001	0.54	Linear increase into recovery
Norepinephrine	2.1	0.094	0.09	3	0.097	0.13	2.2	<0.001	0.65	AUC: PL4 > PL3 (t=2.1; p=0.045; d=0.89)

Note: N/A = a significant interaction effect renders the analysis of main group and main time effects obsolete. Abbreviations: PL4 = Performance level 4; PL3 = Performance level 3; VT-1 = First ventilatory threshold; p-RPE = physical-Rating of perceived exertion; TEA = Task effort and awareness; FS = Feeling scale; FAS = Felt arousal scale; ACRISS = Action crisis scale, AUC = Area under curve.

Part Two: Competition Outcome Comparison

Participant Characteristics

Anthropometric characteristics and performance parameters of the respective winners and losers of 14 head-to-head competitions are listed in table 5.

Table 5: Competition outcome comparison of anthropometric and performance data.

	Winners (n=14)	Losers (n=14)	p-value	Cohen´s d
Descriptive data				
Age (years)	33 ± 6.0	37 ± 6	n.s.	-
Stature (cm)	179 ± 5.0	180 ± 6	n.s.	-
Body mass (kg)	72.1 ± 6.2	75.1 ± 7.1	n.s.	-
Percentage body fat (%)	7.5 ± 3.0	10.6 ± 2.1	0.002	1.51
Training time ($h \cdot wk^{-1}$)	9.1 ± 2.9	9.4 ± 1.8	n.s.	-
Performance data				
abs PPO (W)	376 ± 39	381 ± 45	n.s.	-
rel PPO ($W \cdot kg^{-1}$)	5.2 ± 0.6	5.1 ± 0.3	n.s.	-
abs VO_{2peak} ($L \cdot min^{-1}$)	4.45 ± 0.45	4.52 ± 0.55	n.s.	-
rel VO_{2peak} ($ml \cdot min^{-1} \cdot kg^{-1}$)	62 ± 7.1	60.1 ± 4.5	n.s.	-
abs PO @ VT-1	267 ± 29	264 ± 28	n.s.	-
rel PO @ VT-1	3.7 ± 0.4	3.5 ± 0.2	n.s.	-
70km TT time				
$COMP_{TT}$ time (h:min:s)	2:08:17 ± 0:08:56	2:14:53 ± 0:08:58	*0.062*	*0.74*

Abbreviations: abs = absolute; rel = relative; PPO = peak power output; VO_{2peak} = peak oxygen consumption; VT-1 = First ventilatory threshold; $COMP_{TT}$ = Competition time trial; Data represented as mean ± SD.

Time Trial Times

The average time difference in the $BASE_{TT}$, used to match cyclists for the head-to-head competition, and the $INDV_{TT}$ was 0.92% and 1.06%, respectively. When compared to cyclists' own $INDV_{TT}$ time, winners were able to improve their performance during the $COMP_{TT}$ on average by 2.24%, while losers showed a performance decrement of 1.69%. The true magnitude of winning the head-to-head competition was most likely substantially positive (99.7% most likely substantially positive, 0.3% most unlikely trivial and 0.0% most unlikely substantially negative), while losing was likely substantially negative to performance (0.5% very unlikely substantially positive, 17.1% unlikely trivial and 82.4% likely substantially negative).

Performance Data

Power, Power to Weight, and Power to VT-1

There was neither a significant time × group interaction effect for absolute power output ($F_{3.3,\ 84.7}$=2.0; p=0.119; η_p^2=0.071), nor a significant main group effect between the winners and losers of the head-to-head competition despite winners having greater absolute power output from 14 km onwards. ($F_{1,\ 26}$=2.0; p=0.169; η_p^2=0.072). There was a significant main time effect with lower absolute power output during the downhill sections ($F_{3.3,\ 84.7}$=62.3; p<0.001; η_p^2=0.702). (See figure 5 a)).

There also was no significant time × group interaction effect for relative power output ($F_{3.2,\ 82.4}$=0.3; p=0.811; η_p^2=0.013). However, in contrast to absolute power output, relative power output was significantly greater in winning cyclists throughout the $INDV_{TT}$ ($F_{1,\ 26}$=21.2; p<0.001; η_p^2=0.449). There was a significant main time effect with the highest values at the start and significantly lower values during the downhill sections ($F_{3.2,\ 82.4}$=58.0; p<0.001; η_p^2=0.690). (Not shown).

The time × group interaction effect of relative exercise intensity in relation to the first ventilatory threshold was just outside the significance level cut-off ($F_{3.6,\ 94.7}$=2.5; p=0.051; η_p^2=0.089). However, the main group effect was significant with winners performing at a higher relative intensity from 14 km onwards ($F_{1,\ 26}$=7.6; p=0.011; η_p^2 =0.226). Relative exercise intensity was greatest at the start of the trial and dropped thereafter, particularly during the downhill sections ($F_{3.6,\ 94.7}$=68.1; p<0.001; η_p^2=0.724). (See figure 5 b)).

Split Time

A significant time × group interaction effect for 7-km interval split times was observed ($F_{1.6,\ 42.7}$=5.9; p=0.009; η_p^2=0.184). Winning cyclists were significantly faster during the second uphill and the final flat section to the finish line. (Simple (main) group effects: $F_{1,\ 26}$>5.3; p<0.029; η_p^2>0.170). However, there were no significant split time differences over the remaining sections of the course ($F_{1,\ 26}$<3.4; p>0.078; η_p^2<0.115).

Split times changed significantly over time and according to the course profile in both performance level groups with simple (main) time effects of ($F_{1.3, 17.1}$=448.8; p<0.001; η_p^2=0.972) and ($F_{1.6, 20.7}$=636.6; p<0.001; η_p^2=0.980) for winners and losers, respectively. (See figure 5 c)).

Accumulated Gap to Lead

Furthermore, a highly significant time × group interaction effect was found for the accumulated difference between the leader of the competition and the chaser ($F_{1.3, 32.8}$=47.0; p<0.001; η_p^2=0.644). On average, winning cyclists significantly pulled away from their competitors from 14 km onwards (Simple (main) group effect: $F_{1, 26}$=5.9; p=0.023; η_p^2=0.184) and thereafter they continuously increased their lead to the finish (Simple (main) group effects: $F_{1, 26}$>12.4; p<0.002; η_p^2>0.322). Accumulated split time differences changed significantly over time for both winners and losers with the leader pulling away and the chaser falling behind. Simple (main) time effects were ($F_{1.3, 16.6}$=24.6; p<0.001; η_p^2=0.654) and ($F_{1.1, 16.2}$=22.6; p<0.001; η_p^2 =0.635) for winners and losers, respectively. (See figure 5 d)).

Accumulated Time Difference to INDV$_{TT}$

There was a significant time × group interaction effect when the accumulated time difference to the cyclist's own INDV$_{TT}$ performance was compared between the winners and the losers ($F_{1.4, 37.4}$=10.0; p=0.001; η_p^2=0.290). During the first 21 km there were no significant simple (main) group effects for accumulated time improvement in either winners or losers when compared to their own INDV$_{TT}$ ($F_{1, 26}$<3.2; p>0.085; η_p^2 <0.109), respectively. Thereafter, winning cyclists accumulated significantly and increasingly more time improvement to their own INDV$_{TT}$ time than the losers, which were on par with their INDV$_{TT}$ split times till 49 km before slowing down in the last third of the course ($F_{1, 26}$>7.5; p<0.011; η_p^2>0.224). Thus, accumulated time difference to the INDV$_{TT}$ changed significantly over time for both winners and losers, albeit in antagonistic directions. Simple (main) time effects of ($F_{1.7, 22.4}$=10.8; p=0.001; η_p^2 =0.454) and ($F_{1.3, 17.0}$=4.7; p=0.037; η_p^2=0.263) were found for winners and losers, respectively. (See figure 5 e)).

Heart Rate

There was neither a significant time × group interaction effect for heart rate ($F_{2.3, 59.1}$=0.6; p=0.583; η_p^2=0.022), nor a significant main group effect ($F_{1, 26}$=0.4; p=0.535; η_p^2=0.015). However, heart rate changed significantly over time (time main effect: $F_{2.3, 59.1}$=9.2; p<0.001; η_p^2=0.260). (Not shown)).

Cadence

A significant time × group interaction effect for cadence was observed ($F_{3.9, 101.5}$=3.1; p=0.021; η_p^2=0.106). The winners used significantly higher cadences during the two downhill sections and the second uphill section (Simple (main) group effects: $F_{1, 26}$>4.4; p<0.045; η_p^2>0.146). However, there were no significant simple (main) group differences in cadence during the first uphill and the flat sections of the course i.e. reaching ($F_{1, 26}$<3.1; p>0.088; η_p^2<0.108). Cadence changed significantly in winners and losers and according to the course profile, (simple (main) time effect for winners ($F_{3.4, 43.8}$=22.5; p<0.001; η_p^2=0.634) and losers ($F_{3.4, 44.8}$ =31.0; p<0.001; η_p^2=0.704), respectively. (Not shown)). However, winners consistently chose higher cadences during the downhill sections during all trials pointing towards dispositional factors.

Perceptual Data

All perceptive data were additionally analysed with a non-parametric factorial procedure using common analysis of variance after align rank transform (ART). The results are provided in squared brackets after the parametric test results in parenthesis.

Perceived Physical Strain (p-RPE)

There was neither a significant time × group interaction effect for p-RPE scores ($F_{4.0, 105.1}$=0.3; p=0.868; η_p^2=0.012) [ART: $F_{4.0, 103.0}$=0.3; p=0.843; η_p^2=0.013], nor a significant main group effect ($F_{1, 26}$=0.1; p=0.760; η_p^2=0.004) [ART: $F_{1, 26}$=0.1; p=0.760; η_p^2=0.004]. However, p-RPE significantly increased over time and in accordance with the course profile (main time effect: $F_{4.0, 105.1}$=35.6; p<0.001; η_p^2=0.578) [ART: $F_{4.1, 107.5}$=29.8; p<0.001; η_p^2=0.534]. (See figure 5 f)).

Perceived Mental Strain (TEA)

Neither a significant time × group interaction effect ($F_{4.3, 112.1}$=0.6; p=0.640; η_p^2=0.024) [ART: $F_{4.3, 111.3}$=0.5; p=0.729; η_p^2=0.020], nor a significant main group effect were observed for TEA scores ($F_{1, 26}$=1.1; p=0.313; η_p^2=0.039) [ART: $F_{1, 26}$=1.0; p=0.328; η_p^2=0.037]. There was a significant main time effect with an increase over time and in line with the course profile ($F_{4.4, 112.1}$=16.6; p<0.001; η_p^2=0.390) [ART: $F_{4.4, 114.5}$=16.1; p<0.0001; η_p^2=0.382]. (See figure 5 g)).

Valence (FS)

A significant time × group interaction effect was found for hedonicity ($F_{4.2, 110.0}$=4.0; p=0.004; η_p^2=0.134) [ART: $F_{5.3, 138.3}$=4.1; p=0.001; η_p^2=0.137] with winners being able to maintain positive hedonic tone throughout the trial, while losers became increasingly negative. There were no significant simple (main) group effects in hedonicity till the end of the second flat section despite a trend for dropping to 'neutral' from the first climb onwards in losers only ($F_{1, 26}$<3.4; p>0.076; η_p^2<0.116). From the second climb onwards, winners and losers significantly differed from each other with losers increasingly experiencing displeasure (Simple (main) group effects: $F_{1, 26}$>12.2; p<0.002; η_p^2>0.320). Simple (main) time effects revealed a differential response in hedonicity over time between winners and losers with winners being able to maintain positive hedonic tone ($F_{2.6, 33.2}$=0.8; p=0.501; η_p^2=0.056), while losers' experience changed significantly from pleasure to displeasure ($F_{4.7, 60.9}$=11.8; p<0.001; η_p^2=0.475). (See figure 5 h)).

Felt Activation (FAS)

There was neither a significant time × group interaction effect for felt activation ($F_{5.4, 139.9}$=0.8; p=0.527; η_p^2=0.031) [ART: $F_{5.3, 137.1}$=0.7; p=0.659; η_p^2=0.025], nor a significant main group effect ($F_{1, 26}$=0.0; p=0.963; η_p^2<0.001) [ART: $F_{1, 26}$=0.0; p=0.927; η_p^2<0.001]. However, felt activation changed significantly over time increasing during

the uphill sections and particularly towards the finish line ($F_{5.4,\ 139.9}$=9.6; p<0.001; η_p^2 =0.270) [ART: $F_{5.5,\ 141.9}$=9.3; p<0.001; η_p^2 =0.262]. (See figure 5 i)).

Action Crisis (ACRISS)

A significant time × group interaction effect was observed for action crisis ($F_{2.3,\ 60.0}$=8.5; p<0.001; η_p^2 =0.245) [ART: $F_{3.1,\ 79.4}$=8.3; p<0.001; η_p^2 =0.242]. There were no significant simple (main) group effects in experienced action crisis over the first flat section ($F_{1,\ 26}$<1.3; p>0.259; η_p^2 <0.049). Thereafter, groups started to differ significantly after the first downhill (Simple (main) group effects: $F_{1,\ 26}$>4.6; p<0.041; η_p^2 >0.151). Simple (main) time effects revealed a differential response in experienced action crisis over time between winners and losers. While winners experienced no significant change in experienced action crisis throughout the course ($F_{1.8,\ 23.7}$=2.1; p=0.154; η_p^2 =0.136), losers significantly and increasingly struggled with ongoing conflict of further goal-pursuit and goal-disengagement from the first climb and even more so from the second climb onwards ($F_{2.5,\ 32.7}$=7.4; p=0.001; η_p^2 =0.362). (See figure 5 j)).

Haematological Data

Lactate

Neither a significant time × group interaction effect for blood lactate concentrations ($F_{2.3,\ 61.0}$=1.1; p=0.345; η_p^2 =0.040), nor a significant main group effect was found ($F_{1,\ 26}$=1.2; p=0.281; η_p^2 =0.045). However, there was a significant main time effect with the highest blood lactate concentrations found in the first flat section and the first climb after which blood lactate concentrations decreased ($F_{2.3,\ 61.0}$=14.7; p<0.001; η_p^2 =0.361). (Not shown).

Cortisol

There was a significant time × group interaction effect for blood cortisol concentrations ($F_{2.0,\ 51.0}$=8.9; p=0.001; η_p^2 =0.255). Winners had significantly higher blood cortisol concentrations at rest and at the start (Simple (main) group effect: $F_{1,\ 26}$>7.7; p<0.010;

η_p^2>0.228). There was no significant simple (main) group effect for the first 14 kilometres ($F_{1, 26}$=0.3; p=0.561; η_p^2=0.013). Thereafter, blood cortisol concentrations were significantly higher in losers and continuously increased throughout the remainder of the trial (Simple (main) group effects: $F_{1, 26}$>5.4; p<0.028; η_p^2>0.173). Simple (main) time effects revealed a significant increase in blood cortisol concentrations over time, albeit to a lower degree in winners ($F_{2.2, 28.8}$=9.7; p<0.001; η_p^2=0.429) than in losers ($F_{1.7, 22.7}$=32.5; p<0.001; η_p^2=0.715). (See figure 5 k)).

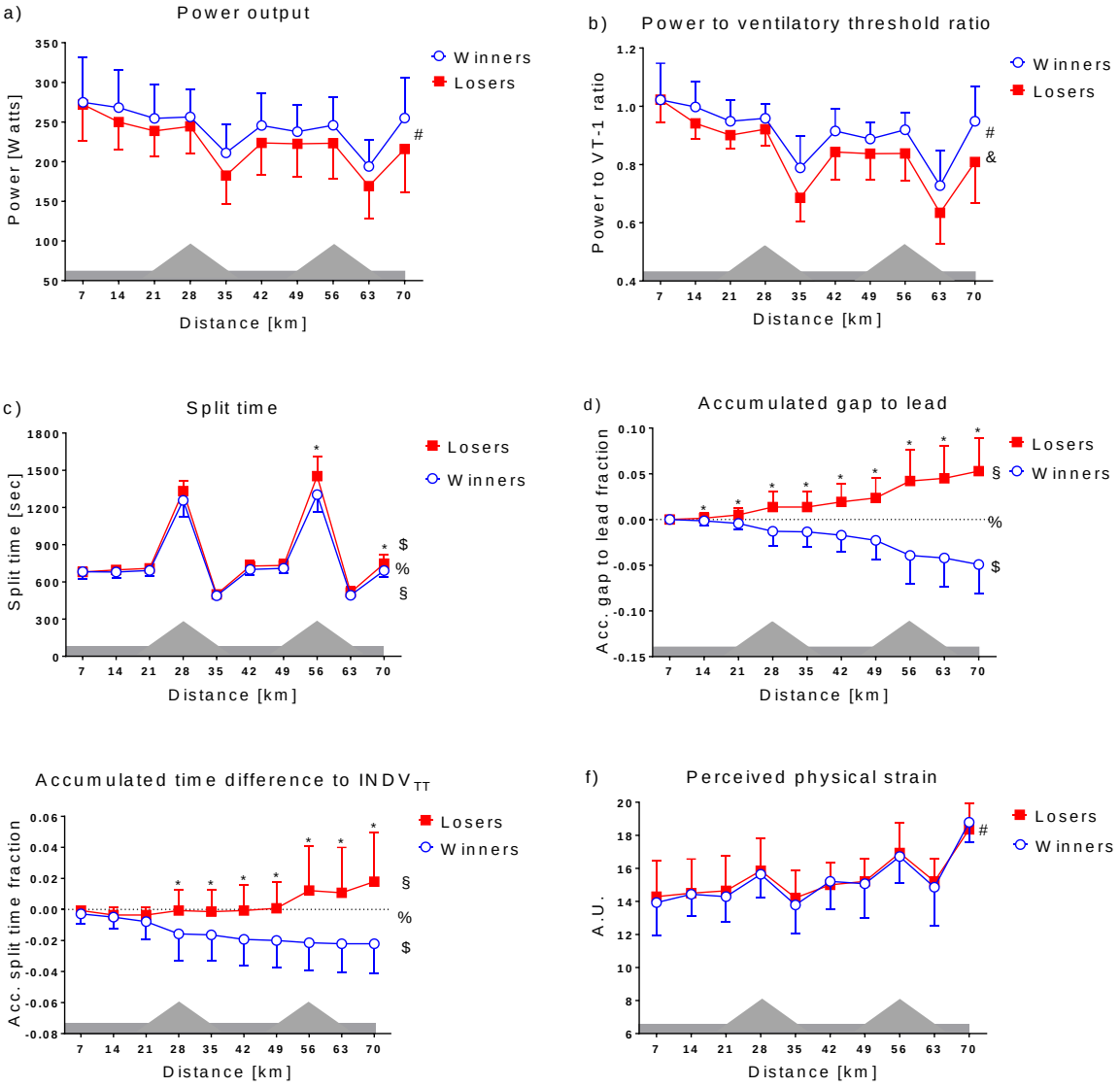
Norepinephrine

Equality of variance of blood norepinephrine concentrations was violated at the first four sampling points. Thus, additional non-parametric analysis was conducted. There was a trend towards, but no significant time × group interaction effect for blood norepinephrine concentrations when using parametric analysis ($F_{2.6, 67.6}$=2.3; p=0.098; η_p^2=0.080). In contrast non-parametric analysis revealed significant results, albeit with small effect size only [ART: $F_{3.6, 92.7}$=2.7; p=0.041; η_p^2=0.094]. However, there was a significant main group effect with losers showing greater blood norepinephrine concentrations throughout the 70-km $COMP_{TT}$ ($F_{1, 26}$=4.9; p=0.036; η_p^2=0.158), which was confirmed by non-parametric factorial procedure [ART: $F_{1, 26}$=5.1; p=0.032; η_p^2=0.164]. There also was a significant change over time in both groups with a sharp logarithmic increase towards the first climb followed by a plateau and rapid decline during recovery ($F_{2.6, 67.6}$=39.1; p<0.001; η_p^2=0.601) [ART: $F_{4.0, 7105.1}$=59.5; p<0.001; η_p^2=0.696]. (See figure 5 l)).

Glucose

Neither a significant time × group interaction effect ($F_{2.8, 74.1}$=0.6; p=0.618; η_p^2=0.022), nor main group effect ($F_{1, 26}$=0.0; p=0.999; η_p^2<0.001) for blood glucose concentrations was found. Nevertheless, there was a significant main time effect with peak blood glucose concentrations measured at the top of the first climb ($F_{2.8, 74.1}$=7.1; p<0.001; η_p^2=0.215). (Not shown)).

Graphical depictions of performance, perceptive, and haematological data for competition outcome comparison are summarised in figure 5.



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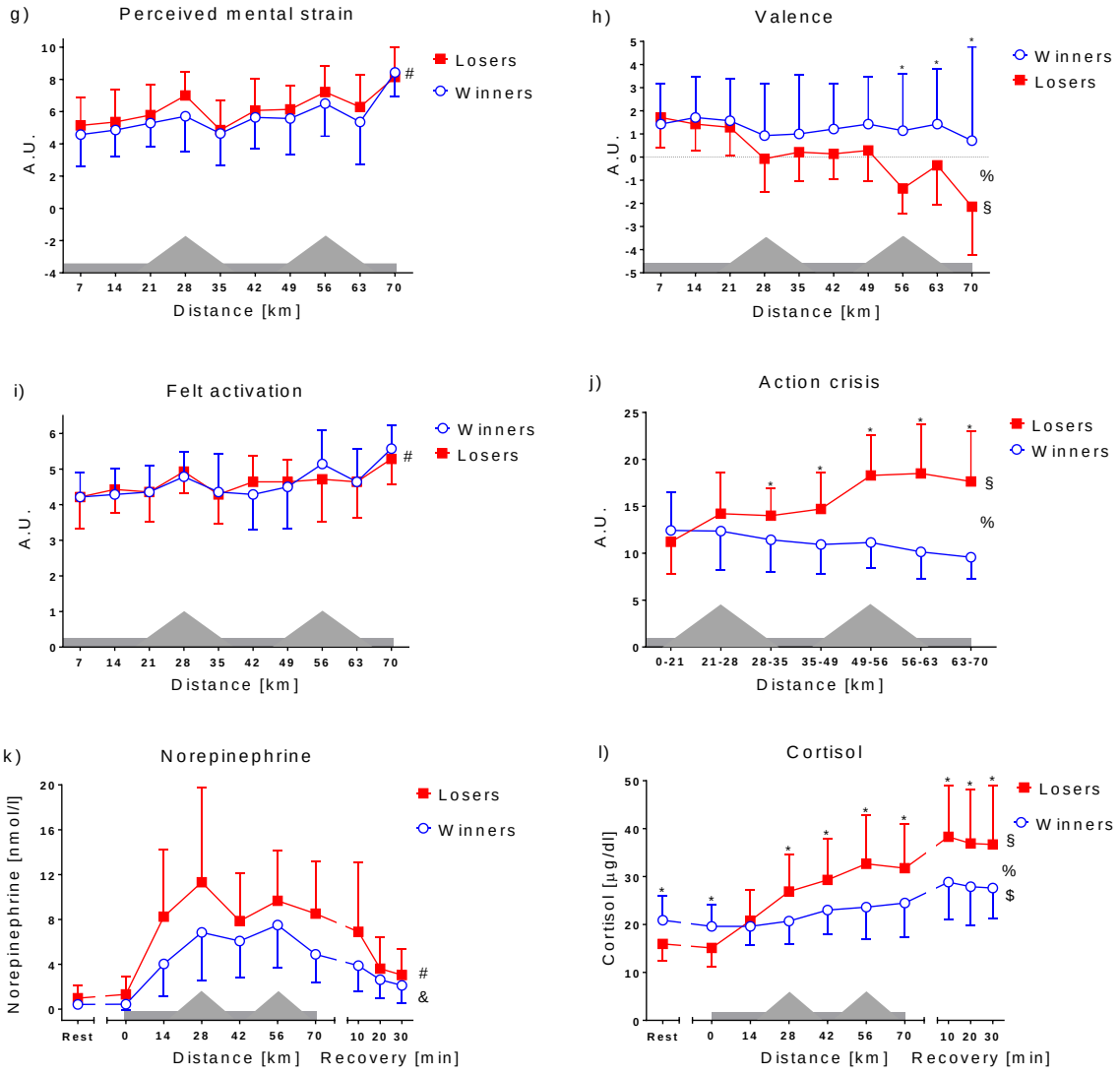

Figure 5: Competition outcome comparison of performance, perceptive, and haematological data.

Note the differences in the x-axes of action crisis and haematological parameters due to different sampling times.

Abbreviations: # = main time effect; & = main group effect; % = time x group interaction effect; $ = simple (main) time effect for winners; § = simple (main) time effect for losers; * = simple (main) group effect

Summary of Performance, Perceptive, and Haematological Data

A summary of statistical findings in main performance, perceptive, and haematological variables for competition outcome comparison is provided in table 6.

Table 6: Summary of competition outcome comparison in performance, perceptive, and haematological variables.

	Time x Group Interaction			Group Main Effect			Time Main Effect			Comment
	F	*p*	h_p^2	*F*	*p*	h_p^2	*F*	*p*	h_p^2	
Performance data										
Power	2.0	0.119	0.07	2.0	0.169	0.07	62.3	<0.001	0.70	Fast start and change with course profile
Power to weight	0.3	0.811	0.01	21.2	<0.001	0.45	58.0	<0.001	0.69	W > L; Fast start and change with course profile
Power to VT-1	2.5	0.051	0.09	7.6	0.011	0.23	68.1	<0.001	0.72	W > L; Fast start and change with course profile
Split time	5.9	0.009	0.18		N/A			N/A		W faster in final uphill and flat section only
Gap to lead	47.0	<0.001	0.29		N/A			N/A		Increasing gap between W and L
Difference to $INDV_{TT}$	10.0	0.001	0.64		N/A			N/A		W faster and L slower compared to their $INDV_{TT}$
Perceptual data										
p-RPE	0.3	0.868	0.01	0.1	0.760	0.00	35.6	<0.001	0.58	Scalar increase and change with course profile
TEA	0.6	0.640	0.02	1.1	0.313	0.04	16.6	<0.001	0.39	Scalar increase and change with course profile
FS	4.0	0.004	0.13		N/A			N/A		L increasingly more negative than W
FAS	0.8	0.527	0.03	<0.1	0.963	<0.001	9.6	<0.001	0.27	Scalar increase and change with course profile
ACRISS	8.5	<0.001	0.25		N/A			N/A		L increasingly greater action crisis than W
Haematological data										
Lactate	1.1	0.345	0.04	1.2	0.281	0.05	14.7	<0.001	0.36	High at start and decrease after first climb
Cortisol	8.9	0.001	0.26		N/A			N/A		L greater increase in cortisol than W
Norepinephrine	2.3	0.098	0.08	4.9	0.036	0.16	39.1	<0.001	0.60	L > W; Logarithmic increase

Note: N/A=a significant interaction effect renders the analysis of main group and main time effects obsolete.
Abbreviations: L = Losers; W = Winners; VT-1 = First ventilatory threshold; p-RPE = physical-Rating of perceived exertion; TEA = Task effort and awareness; FS = Feeling scale; FAS = Felt arousal scale; ACRISS = Action crisis scale.

Detailed results of three-way (time × trial × competition outcome) repeated measures ANOVA are provided in supplementary material one.

DISCUSSION

Part One: Performance Level Comparison

The main findings were: (A) perceived physical strain showed performance level dependent differences related to observed pacing behaviour and strong correlation with endocrinological distress response, (B) PL3 cyclists showed beginning deterioration in core affect and its underlying two dimensions valence and arousal towards the finish, and (C) well-trained cyclists paced themselves in a way that prevented the development of an action crisis during individual time trials.

First, the simulated profiled course prompted subtle differences in pacing behaviour during the downhill sections between the two performance level groups (see figure 4 c)). The lack of significant differences in split time during downhill sections coincided with greater reductions in perceived physical strain in PL4 cyclists (see figure 4 d)). In addition, the dynamic change in perceived physical strain (i.e. p-RPE slope) was strongly correlated with the dynamic change in the endocrinological distress response (i.e. cortisol slope) (see table 3). The latter is in line with findings from Christian et al.,[64] who showed that dynamic changes in perceived physical discomfort accurately reflected the physiological disturbances *caused by* constant-effort cycling and progressive 4-sond sprints in well-trained cyclists in normoxia and hypoxia. The authors further observed greater perceived difficulty breathing during hypoxia trials in accordance with reduced arterial oxygen saturation. Together, these findings point towards the accuracy of perceived physical strain in the psychophysiological integration of afferent feedback from peripheral and central homeostats.[214,215] Thus, by facilitating the awareness of the physiological condition of the entire body, perceived physical strain becomes a key variable in the central regulation of pacing behaviour.[68]

However, Christian et al.[64] also found that power output and associated quadriceps muscle activation changed in parallel with ‘clamped’ subjective efforts *invested into* submaximal exercise and brief progressive sprints, and independently from peripheral sensations of discomfort. These findings are consistent with findings from

Swart et al.,[57] who also found that the dynamic changes in the subjective awareness of the mental sense of effort and perceived physical strain were distinct from each other during submaximal exercise and intermittent sprints (i.e. p-RPE slopes were steeper than TEA slopes) but not during incremental exercise to exhaustion and maximal time trials. Thus, these related but independent perceptual cues are clearly distinguishable due to their different temporal dynamics.[48] The current study used maximal self-paced time trials and as expected perceived physical and mental strain scaled with time and according to course profile, and were not significantly discernible ($F_{5.0,\ 219.7}$=1.6; p=0.153; η_p^2=0.036) (see figure 4 d) and e)).[57,63]

Collectively, these findings may seem inconsistent at first but fully support the hypothesis that pacing behaviour is centrally regulated in an ongoing transition between efferent feedforward *and* afferent feedback control.[51,90] By necessity, anticipatory feedforward control is always imperfect,[135] while afferent feedback from homeostats providing information about the physiological disturbances caused by the goal-directed behaviour always lags behind.[48] Thus, pacing behaviour is improved when the effort invested into the task aligns with the current physiological ability to provide the desired work rate.[51] This makes perceived mental strain and perceived physical strain essential regulatory components of this sensory-discriminatory process.

Second, despite racing at greater absolute and relative exercise intensities (see figure 4 a) and c)), and consequently greater metabolic disturbance (see figure 4 i)), PL4 cyclists experienced more pleasure more constantly throughout the $INDV_{TT}$ than PL3 cyclists, who felt increasingly less pleasure over the final third of the course (see figure 4 f)). This suggests a greater preference for and tolerance of somatosensory cues in PL4 cyclists and indirectly points towards the influence of psychological moderators in the exercise-intensity dependent relationship between interoceptive and cognitive factors in affective responses.[147,216] This finding is further consistent with observations by Renfree et al.,[217] who showed differences in affective state from onset of exercise between the faster and slower of two 20-km bicycle time trials. The authors hypothesised that positive affect influences the degree of physiological disruption that can be tolerated. In the current study, PL4 cyclists indeed showed a physiologically 'tough' response characterised by greater blood norepinephrine without concomitant greater blood cortisol concentrations[139]

(see figure 4 j) and k)), and increased tolerance of metabolic stress indicated by greater blood lactate concentrations (see figure 4 i)).

PL4 cyclists further showed optimised arousal maintenance at elevated albeit submaximal levels, while PL3 cyclists felt increasingly greater arousal coinciding with reductions in pleasure in the last third of the $INDV_{TT}$ (see figure 4 g)). This finding agrees with a correlational inverted-U relationship between physiological arousal and performance and is supported by the observed correlations between dynamic change in arousal (i.e. FAS slope) and markers of cardiorespiratory performance, peak power output, and $INDV_{TT}$ time (see table 4). This suggests that greater aerobic capacity facilitates better somatic arousal maintenance and subsequent time trial performance. Thus, observed performance level dependent differences in valence and arousal support a primary role of core affect in the regulation of performance.[217]

Third, despite the beginning deterioration in core affect in PL3 cyclists, this did not (yet) lead to the development of an intra-psychic conflict between further goal-pursuit and goal-disengagement, i.e. an action crisis (see figure 4 h)). The physiological disturbance and deterioration in core affect in PL3 cyclists were apparently not severe enough to undermine self-regulatory action control[218] and to elicit the mindset-shift associated with an action crisis.[164] PL3 cyclists were therefore capable of shielding their implemental mindset facilitating goal-striving in the face of adversity from a deliberative mindset facilitating re-evaluation of the desirability and feasibility of the pursued or alternative goals.[168] Thus, well-trained cyclists pace themselves within their volitional and self-regulatory capacities, thereby preventing the development of an action crisis. This contrasts with previous findings in moderately-trained marathon runners, who were found to experience a peak in cost-benefit thinking and action crisis around the 30-km mark before resolving closer to the finish line.[163,170] Collectively, this points towards context-dependent, constraints-based, and non-linear interdependencies between sensory-discriminatory, affective-motivational, and cognitive-evaluative processes underpinning perceived fatigability.

Part Two: Competition Outcome Comparison

The main findings were: (A) perceived physical and mental strain scaled with time and according to course profile in winners and loser alike, (B) losers showed deterioration

in core affect and experienced the development of an action crisis, and (C) losers showed an exacerbated distress response and performance decrement relative to individual performance capabilities.

First, perceived physical strain and perceived mental strain scaled with time and course profile in winners and losers alike (see figure 5 f) and g)). The scalar properties suggest that perceived physical strain and perceived mental strain are key variables used in the central regulation of pacing behaviour as they provide the central nervous system with essential information about the physiological state and the ability to expend the desired effort.[51] Critically, the absence of differential responses in perceived physical strain and perceived mental strain occurred despite increased and decreased performance and subsequent physiological demands in winners and losers, respectively. This dissociation points toward the crucial role of affective-motivational and cognitive-evaluative performance modifiers in efferent feedforward and afferent feedback control.[14,23] Thus, perceived physical strain and perceived mental strain can be distorted and it is hypothesised that the alteration of this protective psychophysiological mechanism allows for behavioural flexibility and contextual performance adjustments within the boundaries necessary to maintain homeostasis.[199]

Second, losers showed significant reductions in valence compared to winners as well as to their own $INDV_{TT}$. Winners successfully maintained hedonic tone and perceived even more pleasure in the final third of the $COMP_{TT}$ compared to their own $INDV_{TT}$ (see figure 5 h)). The observed deterioration in valence in losers was associated with increased accumulated gap to lead and time difference to $INDV_{TT}$ performance (see figure 5 d) and e)). The high degree of physiological disruption due to the fast-start evoked by the competitive environment and the failed attempt to stay in contact with the leader can perfectly be conceived to *antecede* deterioration in core affect. Winners on the other hand sustained the faster pace relative to losers as well as their own $INDV_{TT}$ (see figure 5 c) and e)) and maintained hedonic tone despite arguably greater physiological demands. This differential response between winners and losers points toward the crucial role of psychological moderators in response to respectively pulling ahead or falling behind another competitor.[219]

Third, the differential response in core affect was associated with a significant increase in perceived action crisis in losers only (see figure 5 j)). This intrapsychic conflict

between further goal-pursuit and goal-disengagement is characterised by a shift from an implemental mindset cognitively tuned towards the 'how' of behaviour to a deliberative mindset cognitively tuned towards the 'why' of behaviour.[161] By definition, an action crisis results from negatively valenced events related to setback and failures in goal-striving and after considerable investments have already been made.[163] This suggests that the dynamic change in core affect is the driving force by which attentional focus is shifted towards the unique psychophysiological demands of the exercise task at hand, cognitive re-evaluation is initiated, and pacing strategy is redrawn.[220] Thus, deterioration in core affect *antecede*s concomitant changes in perceived action crisis and indicates that an action crisis is a process and not a discrete singular event.[158] The associated mindset-shift undermines the volitional bias in effective goal-striving by unbiased deliberation about alternative goals, thereby reducing focal goal-commitment.[167] Thus, experiencing an action crisis over time contributes to self-regulatory fatigue,[184,196] is a highly stressful event in goal-striving,[173] and is debilitative to performance.[163–165,170]

Last, the current study found greater sympatho-adrenal and hypothalamo-pituitary-adrenal responses in losers compared to winners (see figure 5 k) and l)). When compared to their own $INDV_{TT}$, winners had similar blood norepinephrine and cortisol concentrations despite improved performance, while losers' blood norepinephrine and cortisol concentrations were elevated by respectively ≈40% and ≈50% despite reduced performance. This indicates that the aggravated endocrinological distress response in losers was due to psychological factors and undoubtedly can be considered physiologically 'weak' and non-adaptive.[139,221] Similar results were provided by Brandstätter et al.,[164] who, after controlling for physiological demands, found greater salivary cortisol secretion in runners experiencing an action crisis prior to a marathon. Together, these findings add to research investigating dual-stress challenges showing unfavourable exacerbated responses in stress hormones, cardiorespiratory and cardiovascular variables as well as increased effort perception.[222,223] Collectively, these responses constitute a psychophysiological milieu that is not conducive to high performance and provide a psychobiological link between cognitive appraisal processes and performance decrement.[185] Thus, the observed non-adaptive psycho-neuro-endocrinological distress response is proposed to be the *consequence* of experiencing an action crisis and to *antecede* subsequent performance decrement.

In conclusion, it is hypothesised that core affect and action crisis play primary and mediatory roles in performance regulation. More specifically, it is hypothesised that the psychophysiological responses to falling behind a performance matched opponent occur in the following temporal order: (A) falling behind *antecedes* deterioration in core affect, (B) deterioration in core affect *antecedes* development of an action crisis, (C) action crisis *antecedes* non-adaptive endocrinological distress response, and (D) non-adaptive distress response *antecedes* performance decrement.

The extent to which the observed data fit the hypothesised cause-effect relationships will be assessed in chapter three.[224]

Confines and Suggestions

First, the TEA scale was used to assess the subjective awareness of the mental sense of effort, which has not been formally validated yet. Although it yields reliable findings coherent with the RPE scale phrasing “How hard are your trying?”, there are inconsistencies in its theoretical underpinnings and psychometric properties.[199] It is therefore suggested that future research should utilise established RPE scales phrasing “How difficult is it to swim/cycle/run at this pace?” using the descriptors ‘easy’ and ‘hard’ to assess perceived task difficulty and the mental effort *invested into / required to continue with* the task, and to instruct participants with illustrations provided by Smirmaul.[48]

Second, the focus was on the shift from an implemental to a deliberative mindset and thus the goal-disengagement component of the cognitive-evaluative dimension in goal-striving. However, the absence of an action crisis does not imply optimal experience or performance. The ‘winning factor’ can therefore only be indirectly inferred. Future investigations should therefore assess the temporal dynamics in mindsets on a continuum from a state of optimal experience or ‘flow’[225] to perceived action crisis. This will allow the investigation of facilitative cognitive-evaluative processes in goal-striving that are hypothesised to underpin performance enhancement.

CONCLUSION

The current findings provide preliminary supportive evidence for a three-dimensional framework of perceived fatigability. Perceived mental strain and perceived physical

strain, core affect with its underlying two dimensions valence and arousal, and the mindset-shift associated with an action crisis are: interdependent in a non-linear fashion with distinct temporal dynamics, context-dependent, constraint-based, distinguishable by well-trained cyclists, and interrelated with observed pacing behaviour, performance, and physiological disturbance. The proposed framework provides a more comprehensive alternative to the Gestalt concept of perceived exertion and more completely accounts for centrally regulated and goal-directed exercise behaviour.

CHAPTER THREE

MODELLING THE PROCESS OF FALLING BEHIND AND ITS PSYCHOPHYSIOLOGICAL CONSEQUENCES

ABSTRACT

A preceding article investigated the psychophysiological responses to falling behind a performance matched opponent. The following temporally linked cause-effect relationships were hypothesised: falling behind precedes deterioration in valence, deterioration in valence precedes development of an action crisis, experience of an action crisis precedes psycho-neuro-endocrinological distress response, and non-adaptive distress response reduces conduciveness to high performance, thereby preceding performance decrement.

In this article, structural equation modelling was applied to test the extent to which the observed data fit the hypothesised cause-effect relationships**.** A five-step procedure was applied to model the interrelationships between the major study variables in the hypothesised temporal order.

Significant linear relationships were found between all hypothesised predictor and outcome variable pairs ($p<0.024$). The dynamic change in valence was a significant mediator ($p=0.011$) as it explained 35% of the relationship between falling behind and action crisis. All hypothesised cause-effect relationships continued to be significant after controlling for performance, descriptor, training, and perceived strain variables. The observed data fitted the hypothesised structural model well with excellent model fit indices throughout.

The hypothesised debilitative psychophysiological processes that unfold in response to falling behind a performance matched opponent were applied, tested, and confirmed. The main findings were: deterioration in valence mediated the relationship between falling behind and action crisis, the mindset-shift associated with an action crisis predicted increased blood cortisol concentrations, and non-adaptive blood cortisol concentrations predicted performance decrement. The findings point towards the crucial role of affective and cognitive modifiers in centrally regulated and goal-directed exercise behaviour.

INTRODUCTION

A recent review highlighted cause-effect relationships between psychological factors and endurance performance that are both facilitative (e.g. a head-to-head competition) and debilitative (e.g. mental fatigue).[219] Since little is known about the psychological mechanisms underpinning the observed performance improvements and decrements, the authors encouraged researchers to: (A) explore additional psychological factors, other than motivation and effort, that could have an effect on endurance performance, specifically those that can have negative effects, (B) include psychological mediating (i.e. answering hypotheses of 'how' and 'why') and moderating (i.e. answering hypotheses of 'for whom' and 'when') variables, and (C) use psychological theories to determine which factors should be targeted and measured in experimental designs.

In chapter one,[199] a three-dimensional framework of centrally regulated and goal-directed exercise behaviour was developed based on established psychophysiological theories. The proposed framework offers a more sophisticated alternative to the traditional Gestalt RPE concept, comprising sensory-discriminatory, affective-motivational, and cognitive-evaluative dimensions. The delineated contributions of each dimension are determined by the respective lower-order constructs perceived physical and mental strain, valence (i.e. the intrinsic attractiveness and aversiveness of a stimulus) and arousal, and action crisis characterised by a shift from an implemental to deliberative mindset.

In chapter two,[226] the psychophysiological processes were investigated that unfold when cyclists respectively pull ahead or fall behind a performance matched opponent during a simulated 70-km head-to-head competition time trial. This intervention provided the ideal experimental test of the temporal dynamics of goal-striving and goal-disengagement processes, as it covers all aspects of goal-striving: setting a goal (i.e. winning), striving for the goal (i.e. competing), encountering barriers (e.g. falling behind a strong competitor), overcoming difficulties (e.g. exhaustion and frustration), using self-regulation strategies (i.e. coping), and finally goal-attainment or goal-failure.[162]

The dynamic changes in the proposed lower-order constructs (i.e. perceived physical strain, perceived mental strain, valence, arousal, and action crisis) and major study

outcome variables (i.e. gap to lead, metabolic and endocrinological response, and time difference to the cyclist's own individual time trial time) were assessed. The responses in affective and cognitive dimensions varied differentially between winners and losers and corresponded to variations in the endocrinological distress response as well as to alterations in performance.

In a theory-driven approach, the following temporally linked cause-effect relationships were hypothesised: (A) falling behind precedes deterioration in affective valence, (B) deterioration in affective valence precedes the development of an action crisis, (C) experience of an action crisis precedes non-adaptive psycho-neuro-endocrinological distress response, and (D) non-adaptive endocrinological distress response reduces conduciveness to high performance, thereby preceding performance decrement.

An accepted approach to lend credibility to the hypothesised cause-effect relationships is to statistically test the integrity of the hypothesised model structure in relation to observed data. Structural equation modelling (SEM) is a causal inference method that combines testable causal hypotheses based on scientific knowledge and empirical data as input, and produces quantitative causal claims, conditional on the input assumptions, as output.[227] The statistical inference is based on a simultaneous analysis of the entire system of variables determining the extent to which the observed data fit the hypothesised a priori specifications.[228] It thereby provides a higher-level perspective to the analysis and evaluation of entire models. Another key component to theory development is mediation analysis, which aims to clarify how cause-effect relationships come to be,[229] and which are therefore theoretical formulations for unidirectional relationships.[230] Thus, specifically when used in conjunction with an experimental design, the capabilities to formalise and implement causal inference makes SEM an indispensable tool in causal hypothesis testing and mediation analysis; its potential for theory development is even greater.[231]

However, SEM makes only simplified approximations to reality and does not imply that the tested model is actually true.[232] Thus, the theoretical underpinnings of the hypothesised model are of paramount importance so that the preceding articles should be critically reviewed for their internal logic and intuitive appeal.[199,226]

In this chapter, SEM was applied to test the theory-driven temporal relationships described above and to model the process of falling behind and its psychophysiological consequences.

METHODS

Data from chapter two,[226] involving 14 head-to-head 70-km cycling competitions, were used to test the model of causal relationships specified above using SEM. The methods of preliminary and experimental procedures as well as the outcome of conventional statistical analyses of the head-to-head competition time trial have been described in detail in the preceding chapter.[226]

A five-step procedure was applied to assess interrelationships between the major study variables in the hypothesised chain of events. Due to the relatively small sample size (n=28), the first four steps were used to ascertain that the theorised relationships were statistically robust enough for the structural modelling process.

In step one, conventional statistical analyses and visual inspection of the data took place. The dynamic changes in the major study variables that showed significant interaction effects between the competition outcome groups were best described as linear. Therefore, the trends of change in major study variables over time (i.e. their slopes) were calculated before proceeding with correlation and regression analyses.

In step two, zero-order correlations and linear regressions were computed. Zero-order correlations were calculated between the relationships in the dynamic change of all major study variables to determine the strength of association between each variable pair. However, correlation analyses do not allow the estimation of parameter values of variable y from variable x, and therefore do not allow predictions to be made. Thus, significant correlations between variable pairs were followed up with linear regression analyses.

In step three, mediation analyses were conducted when a predictor variable was correlated to the proposed direct outcome variable and another dependent variable. Mediation analysis provides the means to assess the significance of the indirect effect and the extent to which the mediator variable accounts for the relationship between the predictor and the outcome variable. The mediation analysis approach is particularly

appropriate when: (A) the predictor variable is experimentally manipulated and the mediator variable measured and (B) the dynamic change in the predictor variable precedes the dynamic change in the mediator variable; and both precede the outcome variable.[229,233]

In step four, multiple hierarchical regression analyses were conducted between the four pairs of study variables hypothesised to be linked in a temporal order: (A) gap to lead (i.e. falling behind) and valence, (B) valence and action crisis, (C) action crisis and blood cortisol concentration, and (D) blood cortisol concentration and time difference to $INDV_{TT}$ (i.e. performance decrement). Every variable pair was analysed by means of stepwise longitudinal regression analysis to ensure that the relationship between the direct predictor and the outcome variable in each pairwise comparison was independent of performance as well as descriptor, training, and perceived strain variables. Performance was considered indirectly controlled by the matching procedure and therefore not included again as a control variable. Thus, in stage one, the descriptive variables age and fat free mass were entered. Stage two controlled for training variables, i.e. training years and weekly training hours. In stage three, the dynamic changes in perceived physical and mental strain were entered. Lastly, in stage four, the direct predictor of the outcome variable of interest was entered.

In step five, the major study variables were fitted into one structural path model. Despite the small sample size, SEM was deemed appropriate as the variables included in the tested model: (A) were observed variables, (B) showed significant interaction effects in conventional statistical analyses, (C) showed significant zero-order correlations, (D) showed homogeneity in the responses within the groups, (E) showed differential and dynamic changes that were in full agreement with the theoretical predictions, and lastly (F) as a result of the relatively low complexity level of the tested model.[234]

Missing data points are a major problem for the estimation of structural equation models. An overall of 4.31% of all blood cortisol samples taken during the $COMP_{TT}$ were missing, due to blood clotting, insufficient sample, or analysis problems. Missing data were considered missing completely at random and thus replaced by means of multiple imputation procedure.[211,235]

Statistical Analysis

All conventional statistical analyses were conducted with IBM® SPSS® (Version 22.0, Chicago, IL, USA). The slope and intercept values were calculated using GRAPHPAD PRISM® (Version 6.00 for Windows, GraphPad Software, La Jolla California, USA). Mediation analysis and structural equation modelling was performed using IBM® SPSS® Amos (Version 22.0, Chicago, IL, USA). Model fits were estimated using the maximum likelihood method and results are represented as standardised estimates. Significance of the indirect effect within mediation analysis was estimated by means of bootstrap sampling and bias-corrected 95% confidence intervals based on 1.000 bootstrap samples.[236] Statistical significance was accepted at $p < 0.05$ (two-tailed).

RESULTS

Step 1: Visual Inspection and Conventional Statistical Analyses

The temporal dynamics of the major study variables showing significant interaction effects between the winners and the losers of the 70-km head-to-head competition time trial are shown in figure 6.

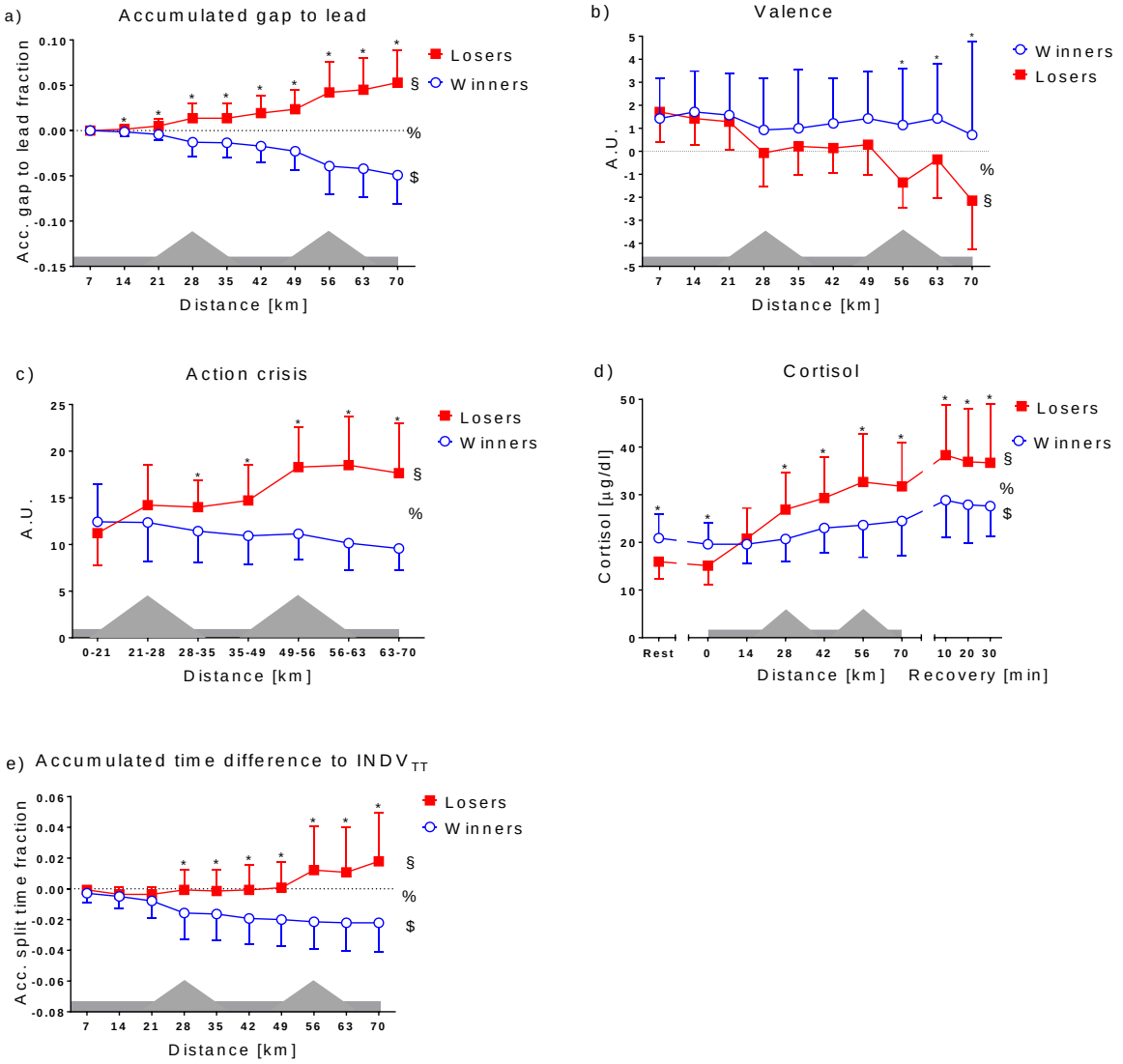

Figure 6: Dynamic changes in major study variables and differential responses between the winners and losers of a simulated 70-km head-to-head competition time trial over a virtual profiled 70-km time trial course.

Note the differences in the x-axes of action crisis and blood cortisol concentrations due to different sampling times.

Abbreviations: % = time x group interaction effect; $ = simple (main) time effect for winners; § = simple (main) time effect for losers; * = simple (main) group effect

Step 2: Correlation and Linear Regression

Zero-order correlations among the major study variables are provided in table 7.

Table 7: Zero order correlations between the dynamic change in major study variables and time difference to the individual time trial.

Variable	1	2	3	4	5	6	7
1. p-RPE slope	-						
2. TEA slope	.86**	-					
3. Felt arousal slope	.48**	.55**	-				
4. Gap to lead slope	-.25	-.13	-.15	-			
5. Valence slope	-.30	-.23	.08	-.48**	-		
6. Action crisis slope	.07	.14	-.01	.51**	-.54**	-	
7. Cortisol slope	-.06	-.07	-.15	.26	-.33	.43*	-
9. Difference to $INDV_{TT}$	-.10	-.06	.08	.58**	-.27	.36	.43*

Abbreviations: p-RPE = physical - Rating of perceived exertion scale used to approximate perceived physical strain; TEA = Task effort and awareness scale used to approximate perceived mental strain.
** Correlation significant at the 0.01 level (2-tailed). * Correlation significant at the 0.05 level (2-tailed).

Perceived physical strain, perceived mental strain, and felt arousal were significantly correlated to each other, but provided no differential information between the groups. These three variables further showed no significant relationships with any of the other perceptive, performance, or biochemical variables. Consequently, they were excluded as major study outcome variables from subsequent modelling steps, but perceived physical and mental strain were included as control variables in the multiple hierarchical regression analyses.

In contrast, significant linear relationships were found between gap to lead (i.e. falling behind) and valence (β = -0.48; p = 0.009), gap to lead and action crisis (β = 0.51; p = 0.006), and gap to lead and time difference to $INDV_{TT}$ (i.e. performance decrement) (β = 0.58; p = 0.001). The significant relationships between gap to lead, valence, and action crisis required the examination of a potential mediation effect of valence on the relationship between gap to lead and action crisis. Furthermore, there were significant relationships in the hypothesised temporal order between valence and action crisis (β = -0.54; p = 0.003), action crisis and cortisol (β = 0.43; p = 0.024), and cortisol and time difference to $INDV_{TT}$ (β = .43; p = 0.023).

Step3: Mediation Analysis

In full agreement with our theory driven approach, the dynamic change in valence was a significant mediator as it explained 35% of the relationship between gap to lead and action crisis. The significant total effect c (standardised estimate = 0.51**, p = 0.006) between gap to lead and action crisis was reduced to a non-significant direct effect c' (standardised estimate = 0.33, p = 0.060) after controlling for the significant indirect effect a x b (standardised estimate = 0.18, p = 0.011). Statistical details are provided in figure 7 and table 8.

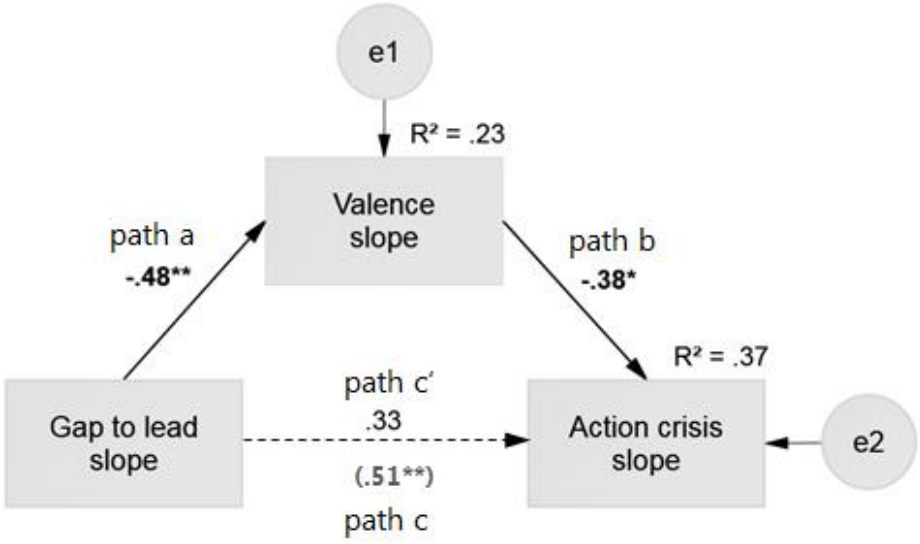

Figure 7: A mediation model of dynamic changes in gap to lead, valence, and action crisis during a 70-km head-to-head competition time trial. Dynamic change in valence was a significant mediating factor in the relationship between dynamic changes in gap to lead and action crisis.

Abbreviations: Squares represent observed variables; Single headed arrows represent regression paths; Bold regression paths are significant at * p < 0.05 and ** p < 0.01, respectively; Dashed regression paths are not significant; R^2 = total variance explained; e = residual error

Table 8: Mediation analysis testing whether the dynamic change in valence partly accounts for the association between gap to lead and action crisis.

Effect	Standardised estimate	CR	SE	95%CI	p
Path c (total effect)	.51	3.02			.006
Path a	-.48	-2.86			.004
Path b	-.38	-2.16			.031
a × b (indirect effect)	.18		.097	[.042, .437]	.011
Path c ' (direct effect)	.33	1.88			.060

Note: Maximum likelihood estimates are provided for paths c, a, b, and c'. For the standardised (a × b) indirect effect, bootstrap estimates with CIs are provided. Abbreviations: CR = Critical ratio; CI = Confidence interval; SE = Standard error.

Step 4: Multiple Hierarchical Regression Analyses

None of the control variables (i.e. age, fat free mass, training history, weekly training volume, perceived physical strain, and perceived mental strain) significantly predicted any of the major study outcome variables (i.e. valence, action crisis, cortisol, and time difference to $INDV_{TT}$). More importantly, all direct predictors (i.e. gap to lead, affective valence, action crisis, and cortisol) continued to significantly predict the respective outcome variable (i.e. valence, action crisis, cortisol, and time difference to $INDV_{TT}$) after controlling for descriptor, training, and perceived physical and mental strain variables. The results of the four stepwise regression analyses are summarised in table 9.

Table 9: Multiple hierarchical regression analyses predicting subsequent dynamic changes in valence, action crisis, cortisol, and time difference to individual time trial time in response to dynamic changes in the respective direct predictor variable.

	Gap to lead ⇨ Valence		Valence ⇨ Action Crisis		Action Crisis ⇨ Cortisol		Cortisol ⇨ Difference to ITT	
Predictor	ΔR^2	β	ΔR^2	β	ΔR^2	β	ΔR^2	β
Step1	.013		.001		.003		.039	
Age		-.114		.028		-.037		-.136
FFM		-.010		.017		.035		.127
Step 2	.107		.015		.011		.155	
Training years		.090		.123		.100		-.306
Training hours		.314		-.050		-.055		.322
Step 3	.048		.024		.010		.001	
p-RPE		-.254		-.192		-.034		-.031
TEA		.025		.286		-.073		.043
Step 4	.383**		.331**		.187*		.213*	
Direct predictor		-.694**		-.630**		.442*		.467*
Total R^2	.550*		.370		.211		.408	
n	28		28		28		28	

Performance was considered indirectly controlled by the matching procedure and therefore not included again as a control variable. p-RPE = physical-Rating of Perceived Exertion scale used to approximate perceived physical strain; TEA = Task effort and awareness scale used to approximate perceived mental strain; ** Correlation significant at the 0.01 level (2-tailed). * Correlation significant at the 0.05 level (2-tailed).

Step 5: Structural Equation Modelling

Details of the structural equation model are provided in figure 8. Besides the significant mediation effect of valence in the relationship between gap to lead and action crisis described previously, an increase in gap to lead significantly predicted a decrease in valence (standardised estimate = -0.48; $p < 0.01$). A decrease in valence significantly predicted an increase in perceived action crisis (standardised estimate = -0.38; $p < 0.05$). An increase in action crisis significantly predicted an increase in blood cortisol concentrations (standardised estimate = 0.43; $p < 0.05$), and an increase in blood cortisol concentrations significantly predicted an increase in time difference to $INDV_{TT}$ (standardised estimate = 0.30; $p < 0.05$). The strong correlation between gap to lead and time difference to $INDV_{TT}$ (standardised estimate = 0.51; $p < 0.01$) is considered a logical inference due to the study design. Consequently, gap to lead maintained much of the total effect (standardised estimate = 0.57; $p < 0.01$) on time difference to $INDV_{TT}$ as a direct effect (standardised estimate = 0.51; $p < 0.01$) within the full structural equation model shown below. The overall fit of the full structural model was excellent with excellent model fit indices throughout and a probability of 93% that the current data fit the theorised model ($\chi^2 = .865$, $p = .929$, $\chi^2/4 = .216$, NFI = 1.256, CFI = 1.000, RMSEA = .000 (95%CI = [.000, .089]; PCLOSE = .937), SRMR = .000).[237]

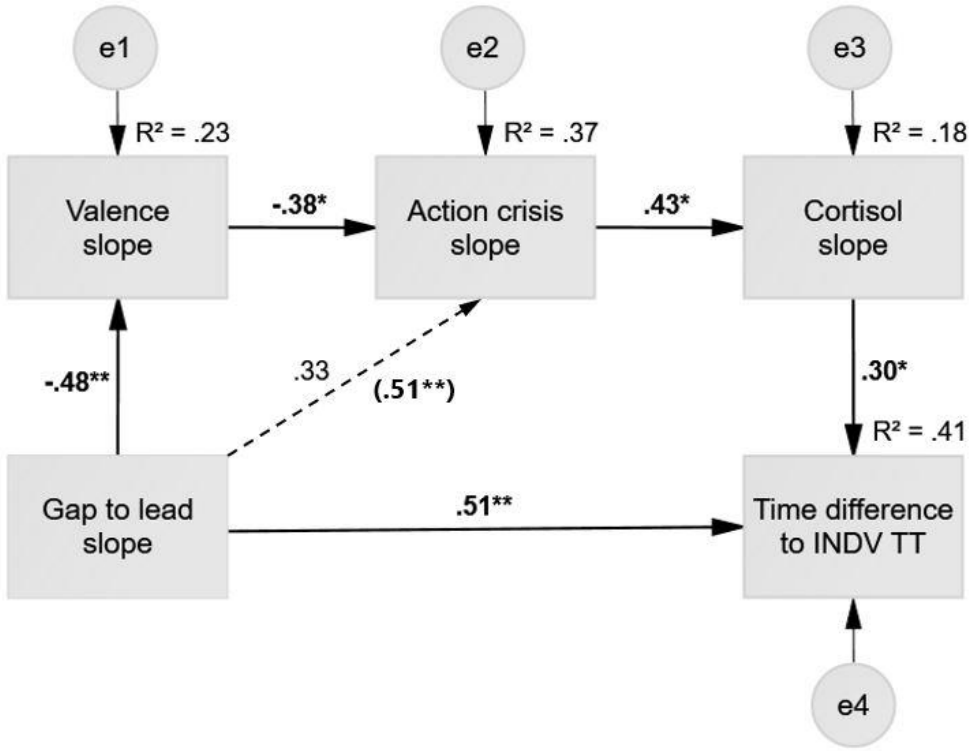

Figure 8: Structural equation modelling of dynamic changes in gap to lead, valence, action crisis, and blood cortisol concentrations during a 70-km head-to-head competition time trial.

Abbreviations: Squares represent observed variables; Single headed arrows represent regression paths; Bold regression paths are significant at * $p < 0.05$ and ** $p < 0.01$, respectively; Dashed regression paths are not significant; R^2 = total variance explained; e = residual error; Standardised maximum likelihood measures are used. χ^2 = Chi-square; NFI = normed fit index; CFI = comparative fit index; RMSEA = root mean square error of approximation; PCLOSE = p of close fit; SRMR = standardised root mean square residual.

Model fit indices are: $\chi^2 = .865$, $p = .929$, $\chi^2/4 = .216$, NFI = 1.256, CFI = 1.000, RMSEA = .000 (95%CI = [.000, .089]; PCLOSE = .937), SRMR = .000.

DISCUSSION

The present research applied SEM to test the hypothesised psychophysiological responses to falling behind a performance matched opponent by combining causal (i.e. formalising and implementing testable causal hypotheses in an experimental design) and statistical (i.e. assessing the extent to which the experimental data fitted the hypothesised model) inference. Excellent model fit indices confirmed the credibility of the hypothesised cause-effect relationships. The main findings were: (A) deterioration in valence mediated the relationship between falling behind and action crisis, (B) the mindset-shift associated with an action crisis predicted increased

blood cortisol concentrations, and (C) non-adaptive blood cortisol concentrations predicted performance decrement relative to cyclists' individual performance capabilities.

First, falling behind a performance matched opponent can undoubtedly be conceived a negative affective event in goal-striving, which subsequently can induce changes in cognitive self-regulatory processes typical of an intra-psychic conflict between further goal-pursuit and goal-disengagement.[173] Thus, deterioration in non-reflective valence is hypothesised to be the consequence of setbacks in goal-progress and to stimulate increases in reflective cost-benefit thinking.[163,170] The current data provide empirical evidence for this hypothesised cause-effect relationship as deterioration in valence was a significant mediator explaining 35% of the relationship between falling behind and the development of an action crisis. The mediatory role of valence can therefore be interpreted as a possible link between heuristic (i.e. intuitive) and deliberative decision-making modes.[135] This further agrees with a recent proposal to apply dual-process theory in the investigation of pacing behaviour and provides convincing evidence against the conceptually flawed affect-cognition[238,239] and conscious-subconscious dichotomy.[32]

Second, the intrapsychic conflict between further goal-pursuit and goal-disengagement (i.e. an action crisis) is characterised by unbiased deliberation about the desirability and feasibility of the focal and alternative goals.[163] These affect laden self-regulatory processes are highly negative emotional and stressful events in goal-striving,[173] and the transactional psychobiological nature of these cognitive appraisals can alter the intensity of the ensuing psycho-neuro-endocrinological distress response.[185] The observed excessive endocrinological distress response despite performance decrement must be considered non-adaptive as it is above and beyond what is physiologically required, thereby providing a psychophysiological milieu that is not conducive to high performance.[222,223] The current findings support the notion that an action crisis exerts its effect on performance indirectly via the hypothalamo-pituitary-adrenal axis as dynamic increases in action crisis predicted aggravated blood cortisol concentrations, which subsequently predicted relative performance decrement to cyclists' individual performance capabilities.

Further empirical evidence for an indirect rather than a direct effect of action crisis on performance comes from a study by Brandstätter et al.,[164] who found that perceived action crisis in runners two weeks before a marathon predicted greater dynamic change in salivary cortisol concentrations and reduced race performance. The salivary cortisol slope was a significant mediator that explained 32% of the variance in the relationship between action crisis and marathon time. These findings corroborate to a large extent with the current data as there was a trend for a significant total effect of dynamic change in action crisis on time difference to $INDV_{TT}$ ($p = 0.058$). A mediation analysis was therefore conducted to ascertain that the total effect of an action crisis on performance is reduced by a non-trivial amount after controlling for the mediator blood cortisol concentration. Critically, the test of an indirect effect is also justified in the absence of a significant total effect between the independent and dependent variable.[240] The indirect effect approached significance ($p = 0.052$) indicating that endocrinological distress response is indeed be an important mediator as it explained 39% of the relationship between action crisis and performance decrement (for statistical details see figure 9 and table 10).

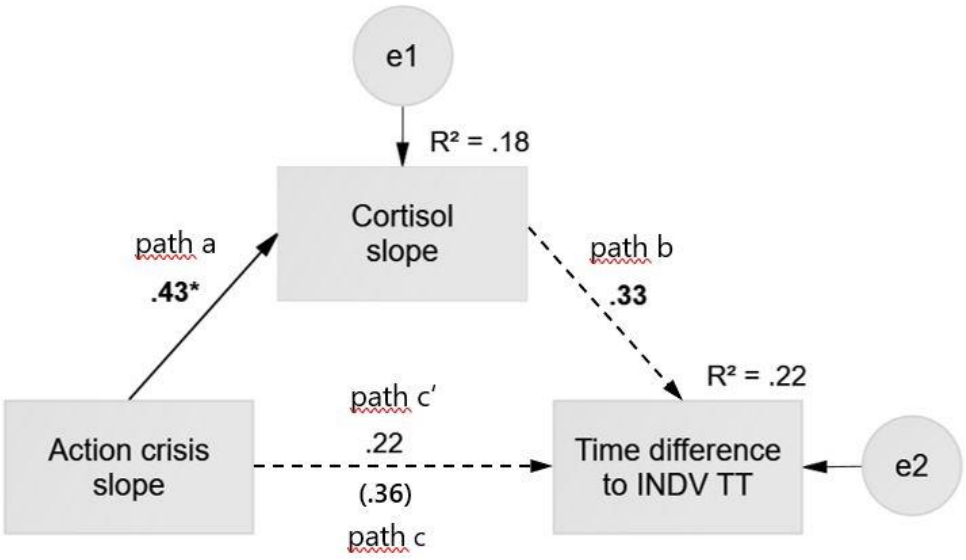

Figure 9: A mediation model of dynamic changes in action crisis, blood cortisol concentrations, and time difference to individual time trial during a 70-km head-to-head competition time trial.

Abbreviations: Squares represent observed variables; Single headed arrows represent regression paths; Bold regression paths are significant at * $p < 0.05$; Long-dashed regression paths show a trend at $p < 0.1$; Dashed regression paths are not significant; R^2 = total variance explained; e = residual error.

Table 10: Mediation analysis testing whether the dynamic change in cortisol partly accounts for the association between action crisis and time difference to individual time trial time.

Effect	Standardised estimate	CR	SE	95%CI	p
Path c (total effect)	.36	1.99			.058
Path a	.43	2.45			.014
Path b	.33	1.78			.076
a × b (indirect effect)	.14		.117	[-.001, .468]	.052
Path c ' (direct effect)	.22	1.18			.238

Note: Maximum likelihood estimates are provided for paths c, a, b, and c'. For the standardised (a × b) indirect effect, bootstrap estimates with CIs are provided. Abbreviations: CR = Critical ratio; CI = Confidence interval; SE = Standard error.

The marginal difference between the current data and those from Brandstätter et al.[164] can be explained by the small sample size in the current study and the respective use of relative time differences and absolute times as markers of performance.

Whilst this analysis provides support for the hypothesised model, it is also important to attempt to refute equally feasible and viable alternative hypotheses. To refute the hypothesis that perceived action crisis does exert a significant direct effect on performance decrement, the fit of a model was estimated with an additional path between dynamic change in action crisis and time difference to $INDV_{TT}$ (See figure 10).

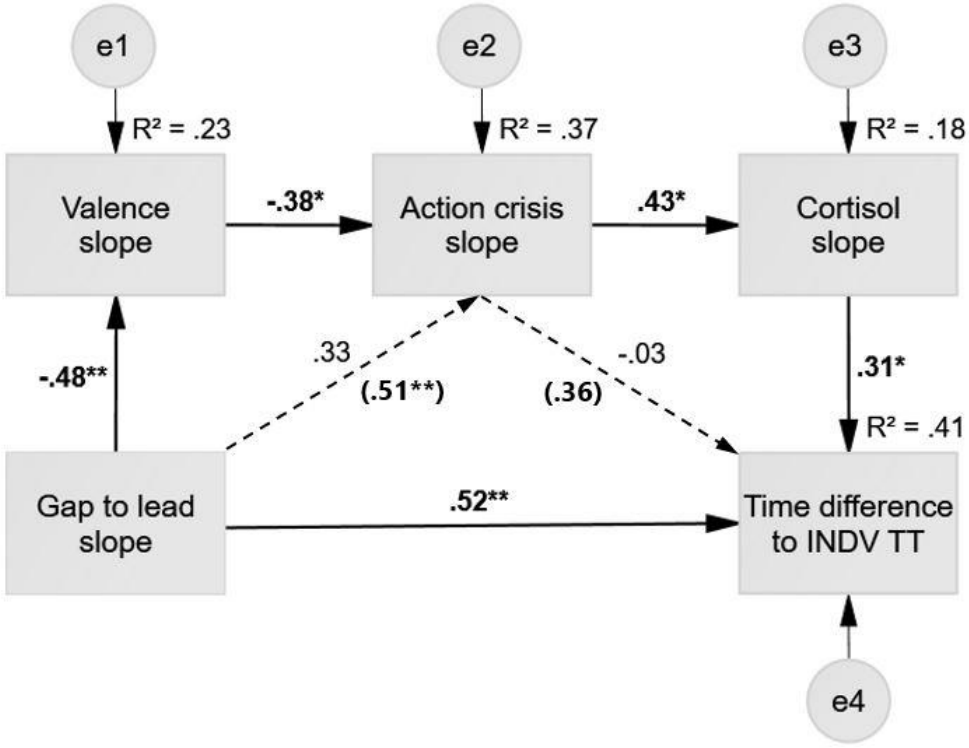

Figure 10: Structural equation modelling of dynamic changes in gap to lead, valence, action crisis, and blood cortisol concentrations during a 70-km head-to-head competition time trial. Exploring the hypothesis of a direct effect of dynamic change in action crisis on performance decrement.

Abbreviations: Squares represent observed variables; Single headed arrows represent regression paths; Bold regression paths are significant at *p < 0.05 and **p < 0.01, respectively; Dashed regression paths are not significant; R^2 = total variance explained; e = residual error; Standardised maximum likelihood measures are used. $\chi 2$ = Chi-square; NFI = normed fit index; CFI = comparative fit index; RMSEA = root mean square error of approximation; PCLOSE = p of close fit; SRMR = standardised root mean square residual.

Model fit indices are: χ^2 = .834, p = .841, $\chi^2/4$ = .278, NFI = 0.979, CFI = 1.000, RMSEA = .000 (95%CI = [.000, .184]; PCLOSE = .854), SRMR = .031.

Within the full structural model, the trend for a significant total effect between action crisis and performance decrement was reduced to a direct effect of almost zero (standardised estimate = -0.03) and is therefore negligible. Furthermore, the current data did not fit this alternative model as well as the hypothesised model, although the model fits were not significantly different ($\Delta\chi^2_{(1)}$ = 0.03, p = 0.86). Given that the hypothesised model is the more parsimonious model it is to be preferred based on the results of this model comparison.

CONCLUSION

The present work followed the proposal for greater eclecticism in the investigation of psychological factors that can have negative effects on endurance performance during head-to-head competitions. The hypothesised cause-effect relationships were applied, tested, and confirmed: (A) deterioration in valence mediated the relationship between falling behind and action crisis, (B) the mindset-shift associated with an action crisis predicted aggravated blood cortisol concentrations, and (C) non-adaptive blood cortisol concentrations predicted performance decrement. The findings clearly point towards the crucial role of affective and cognitive modifiers in centrally regulated and goal-directed exercise behaviour and provide novel insights into the psychophysiological mechanisms that unfold because of falling behind a performance matched opponent.

CHAPTER FOUR

THE PSYCHOPHYSIOLOGICAL REGULATION OF PACING BEHAVIOUR AND PERFORMANCE DURING LONG-DISTANCE RUNNING WITH LOCOMOTOR MUSCLE FATIGUE AND EXERCISE-INDUCED MUSCLE DAMAGE IN HIGHLY TRAINED FEMALE AND MALE RUNNERS

ABSTRACT

Locomotor muscle fatigue (LMMF) and exercise induced muscle damage (EIMD) are common conditions experienced during long-distance running due to the pooled effects of mechanical and metabolic strain on locomotor muscles. However, little is known about the instant effects of combined LMMF and EIMD on pacing behaviour and performance during the decisive final stages of 'real-world' long-distance endurance events.

Twenty-two highly-trained runners (11 females) completed two maximal self-paced 20-km treadmill time trials in a counterbalanced crossover design: (A) in a tapered condition and (B) with LMMF and EIMD. Indicators of muscle damage, muscle metabolic strain, and endocrinological stress were assessed to investigate the physiological effects; and a three-dimensional framework of perceived fatigability was applied to investigate the perceptual effects of running with LMMF and EIMD on performance fatigability.

LMMF and EIMD caused restrictions in work capacity and medium increases in blood leucocyte and neutrophil count, interleukin-6, and cortisol concentrations; collectively representing a physiological milieu likely not conducive to high-performance. LMMF and EIMD further caused large increase in perceived physical strain, large decrease in valence as well as large increase and decrease in action crisis and flow state, respectively. Thus, under the constraint of amplified physical duress, findings are suggestive of heuristic and rational antecedents in the goal-disengagement process.

Dynamic changes in physiological and perceptual effects of LMMF and EIMD are hypothesised to underpin the observed alterations in pacing behaviour and performance fatigability during long-distance running. The applied three-dimensional

framework provides a more comprehensive understanding of strain-perception-thinking-action coupling in centrally regulated and goal-directed exercise behaviour.

INTRODUCTION

Unaccustomed muscular exertion during training for, and competition in, prolonged endurance events can result in significant locomotor muscle fatigue (LMMF) and exercise induced muscle damage (EIMD).[241] Particularly, activities involving muscle lengthening contractions such as running are more likely to induce muscle damage due to high muscular strain coinciding with low neuromuscular recruitment. Thus, the underlying cause of EIMD is suggested to be largely mechanical in nature.[242] However, EIMD also occurs in activities predominantly involving muscle shortening contractions such as swimming and cycling. Metabolic deficiencies are therefore also suggested to play a significant role in muscle damage when metabolic strain outweighs mechanical strain.[243] Accordingly, EIMD is a commonly experienced condition in long-distance running events characterised by a pooled effect of mechanical and metabolic strain on skeletal muscle injury.[241,244–246]

So far, research has focussed on the physiological consequences of EIMD day(s) after inducing it and thus largely on delayed onset of muscular soreness (DOMS). For example, review articles discussed indicators of muscle damage, the repeat bout effect, and sex differences;[247] impact on neuromuscular function and excitation-contraction coupling, sarcomere insertion, and damage to contractile machinery and selective fibre types;[248] impaired metabolism;[243] inflammatory and cytokine response;[249,250] as well as molecular and cellular mechanisms of damage and recovery.[251]

More specifically, EIMD can negatively impact endurance performance by altering cardiorespiratory, metabolic, biomechanical, and thermoregulatory variables. This includes but is not limited to: tachycardia,[252,253] augmented ventilatory response and oxygen consumption,[254,255] attenuated peak oxygen uptake and ventilatory threshold,[256] increased intramuscular carbohydrate oxidation,[257] increased blood lactate concentrations,[258–260] compensatory modifications in running kinematics,[261] decreased running economy,[258,262] and increased core temperature.[263] Critically, differential responses in these variables are more likely to occur once ventilatory

threshold intensities are surpassed. In addition, the effect sizes of individual variables are only small to moderate, which might explain why experimental studies with usually small sample sizes of ≤10 participants do not consistently reach statistical significance, thereby erroneously suggesting conflicting findings or null effects.

Furthermore, these debilitating effects of increased physiological strain on endurance performance are accompanied by debilitating effects of increased perceived fatigability. This includes and is currently limited to: increased perception of effort and perceived exertion[252,253,255,264] and exercise induced pain[265,266] – two of several determinants in the central regulation of pacing behaviour and endurance performance.[68]

However, most of the hitherto mentioned studies investigated the impact of EIMD day(s) *after* inducing it and are therefore confounded by the pronounced systemic inflammatory response characterising DOMS. Accordingly, little is known about the instant effects of combined LMMF and EIMD on pacing behaviour and endurance performance as experienced *during* the decisive final stages of 'real-world' long-distance endurance events.

In contrast, two studies investigated the instant effects of LMMF and mild EIMD (average force loss of 19%) on high-intensity cycling exercise of ≈15 min in moderately-trained participants (performance level 2). Marcora et al.[253] used a time to exhaustion protocol at 80% of peak power output and observed significant decreases in endurance performance as well as significant increases in tachycardia, tachypnoea, and perception of effort during the fatigue trial. DeMorree et al.[267] used a 15-min time trial and observed large decreases in average power output (without differences in pacing behaviour), large decreases in vastus lateralis EMG activity over time, large reductions in blood lactate concentrations at the end, and large increases in perception of effort at the start of the intervention time trial. Both authors proposed a mediatory role of perception of effort in the relationship between LMMF and mild EIMD and endurance performance consistent with increased central motor command and reafferent

signalling.[c] However, the findings are of limited use in understanding the psychophysiological regulation of pacing behaviour and performance fatigability in high-performance long-distance runners, as they used cyclists rather than runners, moderately rather than highly-trained athletes, and short rather than prolonged time trials; and further solely assessed effort perception rather than using a more comprehensive multi-dimensional approach.

In chapter one,[199] a three-dimensional framework of centrally regulated and goal-directed exercise behaviour was proposed emphasising the dynamic and complex interplay of sensory, affective, and cognitive processes underpinning perceived fatigability. This framework more completely accounted for perception-thinking-action coupling in response to psychological distress (i.e. falling behind a performance matched competitor) than the traditional Gestalt concept of perceived exertion.[224] In this chapter, the proposed framework was applied under the constraint of physical distress investigating the psychophysiological determinants of pacing behaviour and endurance performance in highly-trained (performance level 4) long-distance runners during two self-paced maximal 20-km treadmill time trials: (A) running in a tapered condition *versus* (B) running with LMMF and EIMD. We hypothesised that the physiological and perceptual effects of amplified physical strain are associated with dynamic changes in performance fatigability and that strain-perception-thinking-action coupling of sensory, affective, and cognitive processes more completely accounts for the dynamic changes in observed pacing behaviour.

METHODS

Participants

Twenty-two runners, 11 females, estimated to fit performance level 4 criteria,[200] were recruited from local running clubs. Main inclusion criteria were a half-marathon personal best of less than 1h:40min and 1h:25min for female and male runners, respectively. The 15% absolute performance difference accurately reflects the average

[c] Reafference is the anticipated sensory stimulation elicited by voluntary movements and is linked to efferent motor command. It thereby allows distinction between self-initiated sensory stimulation from external stimuli, so called exafference. The computation of the error difference underpins the reafference principle.[360,361]

sex difference in the TOP-100 finishing times of an international half-marathon race event (www.twooceansmarathon.org.za) hosted locally. Performance level categorisation of each participant was subsequently verified according to criteria by DePauw and colleagues.[200,268] Sample size estimation using conventional methods and G*Power 3 software (G*Power, Version 3.1.9.2, Kiel, Germany) indicated overall 20 participants would be needed. All participants provided prior written informed consent to the procedures used in this study, which were approved by the institutional ethics committee and carried out in accordance with the Declaration of Helsinki**.**

Study Design

Participants completed in total four visits including three maximal self-paced 20-km treadmill time trials over a simulated profiled course. A 10-km time trial took place after preliminary testing and acted as a submaximal familiarisation time trial (FAM_{TT}). Before returning for their second visit participants were instructed to log their training and diet for 48 hours prior to the baseline time trial ($BASE_{TT}$) and to prepare in a way that resembled their routine before an important race. They were then advised to repeat this 'mini taper' for the following experimental time trials. Participants then completed the intervention time trial ($INTV_{TT}$) and control time trial ($CTRL_{TT}$) in a counterbalanced AB/BA crossover design. The $INTV_{TT}$ was preceded by a standardised drop-jump protocol (for details see Drop-Jump Protocol below), while the $CTRL_{TT}$ was preceded by a rest period of equal length. A wash-out period of four weeks took place before the crossover trial to (A) allow for complete recovery of muscle damage and (B) control for menstrual cycle in female runners (two were using hormonal contraception and two were amenorrhoeic), who were instructed to schedule experimental trials five to nine days after start of menses (early follicular phase).

Procedures

Preliminary Measurements

During the first laboratory attendance each participant had their age, stature, and body mass recorded. Body fat percentage was estimated using the seven skinfold method.[202]

Peak Treadmill Running Speed Test

A peak treadmill running speed (PTRS) test with peak oxygen consumption (VO_{2peak}) was performed on a motor-driven treadmill (Viasys LE500 CE, Hoechberg-Wuerzburg, Germany). The treadmill gradient was set to 1% simulating the energetic costs of outdoor running[269] and adjusting for treadmill characteristics.[270] After a 10-min self-paced warm-up, participants started the PTRS at 10 and 11 $km \cdot h^{-1}$ for female and male runners, respectively, after which speed increased stepwise by 0.5 $km \cdot h^{-1}$ every min. PTRS was calculated as the last completed stage added to the product of the speed increment and the completed fraction of the incomplete stage. During the progressive exercise test, expired ventilation volume (V_E), oxygen uptake (VO_2), and carbon dioxide production (VCO_2) was measured with an online breath-by-breath gas analyser and pneumotach (Cosmed Quark b^2, Rome, Italy). Calibration took place before each trial according to manufacturer's instructions using a 3-litre syringe (Hans Rudolph 5530, Inc., Kansas City, MO, USA) and gas mixture of known composition (5,05% CO_2; 15,97% N_2). Peak oxygen uptake was determined as the highest recorded VO_2 measurement averaged over 30 s**.** The first-ventilatory threshold and respiratory compensation point were determined according to methods described by Lucia et al.[271]

Maximum Isokinetic Power Output Test

Maximum isokinetic power output of the quadriceps and hamstrings muscles of the dominant leg were measured using the Biodex 3 Isokinetic dynamometer (Biodex Medical Systems, Shirley, NY, USA). Participants were familiarised with the procedure before the FAM_{TT} and $BASE_{TT}$. Participants were seated with their arms crossed over their chest, the hip flexed at 90°, and a knee angle of 90° from full leg extension. The lateral condyle of the femur was aligned with the rotational axis of the dynamometer and the ankle was secured to the lever arm of the dynamometer using a padded Velcro strap. Shoulder and waist straps were used to fixate joint positions during trials. Standardisation was enhanced by performing gravity correction. After a warm-up set of increasing intensity (50, 70, and 90%), participants performed six maximal isokinetic contractions at $120° \cdot s^{-1}$. Isokinetic measurements were chosen due to the repetitive cyclic nature of running specific muscle contractions and the above movement velocity was selected as it more closely resembles running specific contraction speeds as well as provides a trade-off between peak and explosive power output usually measured at

≈60°·s^{-1} and ≈180°·s^{-1}, respectively. Power output curves were displayed on a screen in front of the participant and verbal encouragement was given during maximal contractions.

During the experimental trials, maximum isokinetic power output was assessed twice: (1) after the 10-min self-paced warm-up and (2) within two min after completing the drop-jump protocol and control period, respectively.

Running Economy Test

Assessment of running economy took place before each of the three maximal 20-km time trials. After five min of running at a constant speed of 10 and 11 km·h^{-1} for female and male runners, respectively, participants ran for five min at a speed corresponding to Δ 1/3 between the first-ventilatory threshold and the respiratory compensation point determined during the PTRS. The average values of breath-by-breath VO_2 and VCO_2 during the final min were used to calculate the oxygen cost (OC) and energy cost (EC) of running. Updated non-protein respiratory quotient equations were used to estimate substrate use (g·min^{-1}) during the monitored period.[272] The mean energy content of the metabolised substrates was then calculated according to methods described by Jeukendrup and Wallis;[273] scaled to body mass (BM^{-1}) and expressed per kilometre (km^{-1}).[274]

Time Trial Procedure

Participants performed three maximal self-paced 20-km time trials over a simulated profiled course on the same treadmill. A customised course profile was written using h/p/cosmos para-graphics® software (Version 2.6.14, h/p/cosmos sports and medical GmbH, Nussdorf-Traunstein, Germany) simulating a 20-km long time trial with two uphill sections of 2-km length and a gradient of 7%, starting after 4 and 12 km, respectively. The uphill sections were immediately followed by two downhill sections of the same length and gradient. Participants were assisted with gradient-dependent alterations in running speed, but custom-made modifications of the treadmill enabled participants to self-select running speed at any time with a hand-held remote control in 0.1 km·h^{-1} increments. Only course profile, gradient, and distance covered were displayed to participants during each time trial. Participants were instructed to

complete the time trial in the shortest possible time, but no verbal encouragement was given during the time trial itself.

A fan was placed 1.5 m in front of the participants and the fan level was adjusted in accordance with the course profile to simulate speed-related alterations in peripheral cooling. During the uphill sections fans were set on level one, during the flat sections on level two, and during the downhill sections on level three respectively creating average wind speeds of 3.15, 3.80, and 4.25 $m \cdot s^{-1}$. Participants were permitted to consume water ad libitum and a commercially available carbohydrate drink (Enduren™ Endurance Energy Drink) was provided at 2-km intervals and upon request for an average rate of ≈60$g \cdot h^{-1}$. All experimental trials were conducted under stable climatic conditions (Temperature: 20.5 ± 0.7°C; Humidity: 58.3 ± 5.1%) and commenced at 8 am to control for diurnal variations.

During each running economy test and time trial, speed, gradient, and distance covered were recorded and stored at one second intervals using h/p/cosmos para-graphics® software. Heart rate was recorded at two second intervals throughout each trial by telemetry (Suunto® T6, Suunto Oy, Vantaa, Finland). Performance and heart rate data were subsequently analysed with TrainingPeaks™ analysis software (WKO edition+, Version 3.0, Lafayette, CO, USA). All continuously captured data were averaged into 2-km bins before statistical analyses were performed.

Drop-Jump Protocol

During the intervention trial only, participants performed muscle lengthening contraction exercise consisting of 100 drop-jumps from a step of 45 cm height[275] known to induce force losses consistent with mild EIMD and without confounding effects of significant muscle metabolite accumulation (Blood lactate concentration [mmol/l]: CTRL = 1.5 ± 0.6 *vs* INTV = 1.4 ± 0.6) and cardiovascular demands (Heart rate [bpm]: CTRL= 65 ± 10 *vs* INTV = 100 ± 11).[267] Participants dropped to an approximate knee angle of 90° before jumping upward as high as possible. Drop-jumps were performed every 20 s for a total time of ≈33 min.

Vertical displacement of the centre of mass (COM) was calculated by means of a floor embedded force plate (AMTI, Watertown, MA, USA) and a customised program written

in MATLAB® (R2013a, The Mathworks Inc., Natick, MA, USA) determining vertical take-off velocity of COM using the following equation:[276]

Equation 1: Determining jump height from vertical take-off velocity.

$$\text{Jump-height} = TOV^2 \cdot 2g,$$

where TOV = vertical velocity of COM at take-off, g = 9.81 $m \cdot s^{-2}$.

Before and after the drop-jump protocol and rest period, perceived muscle discomfort and unpleasantness were assessed by means of 100 mm visual analog scales (VAS) ranging from 'no muscle discomfort / unpleasantness at all' to 'unbearable muscle discomfort / unpleasantness'. The extent to which participants experienced DOMS in the 84-hour recovery period after experimental trials was assessed using a 7-point Likert type scale.[277]

Measurements

Perceptual Measures

During the final min of the running economy test and at 2-km intervals during each time trial, participants provided ratings on the following four single-item scales presented in random order. All scales were anchored during the PTRS test.

The 15-point (6-20) Borg scale with the indicator terms 'light' and 'strong' was used to approximate perceived physical strain by phrasing "How strong are the physical sensations from your legs, lungs, and body?" Participants were instructed to include only the subjective perception of physical sensations *caused by* the task and to focus on location, quality, and intensity of physical sensations before returning an overall perceived physical strain score.

The 15-point (0-14) Borg scale with the indicator terms 'easy' and 'hard' was used to approximate perceived mental strain by phrasing "How difficult is it to run at this pace?" Participants were instructed to include only perceived task difficulty and mental effort *invested into / required to continue with* the task before returning a perceived mental strain score.

In line with recent recommendations,[145] chapter two outlined a detailed three-tiered justification process for measurement selection in the assessment of dynamic changes in core affective state during prolonged endurance exercise.[226] Ultimately, the 11-point (-5 'very bad' to 0 'neutral' to +5 'very good') Feeling Scale (FS) and 6-point (1 'low activation' to 6 'high activation') Felt Arousal Scale (FAS) were chosen to approximate dynamic changes in valence and felt activation, respectively.[38,208]

The Action Crisis Scale (ACRISS) and short Flow State Scale (FSS) were administered during the 30-min recovery period retrospectively measuring the extent to which participants experienced a shift from an implemental to a deliberative mindset. Both scales were administered for seven sections of the profiled TT course (3 flat, 2 uphill, and 2 downhill) and rated on 5-point Likert-type scales.

The ACRISS comprises six items: conflict, setbacks, implemental disorientation, rumination, disengagement impulses, and procrastination.[175] The internal consistency estimate of reliability was good with mean Cronbach´s alpha of 0.83 ± 0.08 and 0.89 ± 0.04 during the control and intervention time trial, respectively.

The FSS comprises nine items: challenge-skill balance, action-awareness merging, clear goals, unambiguous feedback, concentration on task at hand, sense of control, transformation of time, and autotelic experience.[278] The internal consistency estimate of reliability was good with mean Cronbach´s alpha of 0.81 ± 0.06 and 0.81 ± 0.07 during the control and intervention time trial, respectively.[d]

Haematological Measures

Venous blood samples from a superficial antecubital vein were taken at rest, after the drop-jump protocol, after the running economy test, half-way through and at the end of experimental time trials as well as after the 30-min recovery period. Blood samples

[d] Corrected total-item correlation of item eight (transformation of time) was consistently negative throughout both trials indicating that item eight loads negatively on the overall flow state construct. Removal of item eight would have been justified and significantly improved the internal consistency estimate of reliability on average from 0.81±0.06 to 0.91±0.04. However, given the small sample size and the nevertheless good Cronbach's alpha, item eight was kept, but it is recommended that critical revision of item eight of the flow state scale is required. For a recent critical review on flow in sport and exercise see Swann et al.[362]

were placed into four different pre-chilled Vacutainers, respectively containing: (A) serum clot activator for the analysis of cortisol, (B) potassium oxalate and sodium fluoride for the analysis of lactate concentrations, and (C) K_2-EDTA for the analysis of interleukin-6, differentiated white blood cell count, haemoglobin, and haematocrit. Where appropriate, samples were inverted five times, immediately centrifuged at 3000rpm at 4°C for 10 min, plasma/serum pipetted off, and kept on ice until stored at -80°C for subsequent analysis. Plasma lactate concentrations were determined using glucose oxidase method (YSI 2300 STAT PLUS, Ohio, USA). Serum cortisol was determined using an automated chemiluminescence system (Architect iSR, Abbott Diagnostics, IL, USA) with conventional reagent kits and calibrators. Interleukin-6 was analysed by means of enzyme linked immunosorbance assay (ELISA) (eBioscience, Bender MedSystems, Vienna, Austria). Differentiated white blood cell count, haematocrit, and haemoglobin were determined by Lancet laboratories using conventional methods. The degree of haemoconcentration was calculated and all blood samples were subsequently corrected for plasma volume changes.[209]

Performance Fatigability Measures

Besides the assessment of over time trial performance between experimental conditions, this study aimed the analyse the decline in objective measures of endurance performance over time. The 2nd half to 1st half split time quotient was calculated as a crude assessment of the degree of positive or negative pacing and repeated measures of 2-km split time intervals were used to locate onset and extent of performance fatigability. For a clearer graphical presentation, dynamics in perceived fatigability were further indicated by the percentage increase in split times during ITT compared to CTT and accordingly assessed via one-way repeated measures ANOVA.

Statistical Analysis

Data were tested for assumptions, normality, equality of variances, equality of co-variance matrices, and sphericity where appropriate. Independent samples t-tests were used to analyse inter-individual differences in parameters between the sexes, and paired samples t-tests were used for intra-individual comparisons in parameters between experimental trials. Between-group effect sizes were calculated using Cohen's d (trivial <0.20, small 0.21– 0.60, medium 0.61–1.20, large 1.21–2.00, very

large 2.01–4.00 and near perfect >4.00). A non-parametric Mann-Whitney-U test was performed when assumption of equality of variance was violated. Confidence intervals and magnitude based inferences for meaningful differences between time trial performances were derived from p-values in accordance with Hopkins.[212] Two-way repeated measures ANOVAs were used to compare changes in parameters over time between experimental trials. Significant interaction effects were followed-up with Bonferroni correction procedure. A Greenhouse-Geisser epsilon adjustment was made when sphericity was violated. Aligned rank transformation procedure (ARTool software 1.5.1, Washington, USA) was used to perform non-parametric factorial analysis on all perceptive data and when assumptions of parametric tests were violated.[213] All ANOVA effect sizes were calculated as partial eta squared (η_p^2). All data are presented as mean ± one standard deviation and an alpha level of <0.05 (two-tailed) was used to indicate statistical significance.

RESULTS

Participant Characteristics

Anthropometric characteristics and performance parameters of female (n=11) and male (n=11) participants are listed in table 11.

Table 11: Sex comparison of anthropometric, training, and performance data.

	Male (n=11)	Female (n=11)	p-value	Effect size
Descriptive data				
Age (years)	29 ± 7	28 ± 10	n.s.	-
Stature (cm)	173 ± 7	163 ± 5	0.001	large
Body mass (kg)*	64.7 ± 8.9	54.2 ± 5.2	0.016	large
Fat free mass (kg)*	60.9 ± 8.0	46.1 ± 3.8	<0.001	very large
Percentage body fat (%)	5.9 ± 1.7	14.8 ± 2.1	<0.001	near perfect
Training data				
Weekly volume ($km \cdot wk^{-1}$)	126 ± 61	61 ± 38	0.008	large
Other training ($h \cdot wk^{-1}$)*	3.1 ± 4.9	5.8 ± 4.2	0.021	small
Training history (years)	8.3 ± 4.4	4.2 ± 2.5	0.019	medium
Performance data				
PTRS ($km \cdot h^{-1}$)	20.1 ± 1.0	17.1 ± 0.7	<0.001	very large
Speed @ VT-1 ($km \cdot h^{-1}$)	15.6 ± 0.9	12.8 ± 0.7	<0.001	very large
Speed @ RCP ($km \cdot h^{-1}$)	17.6 ± 0.8	14.8 ± 0.9	<0.001	very large
abs VO_{2peak} ($ml \cdot min^{-1} \cdot kg^{-1}$)	4.49 ± 0.53	3.15 ± 0.43	<0.001	very large
rel VO_{2peak} ($ml \cdot min^{-1} \cdot kg^{-1}$)	69.8 ± 5.6	58.0 ± 4.6	<0.001	very large
$VO_2 \cdot VO_{2peak}^{-1}$ @ VT-1 (%)	78.8 ± 4.0	79.3 ± 2.7	n.s.	-
$VO_2 \cdot VO_{2peak}^{-1}$ @ RCP (%)	89.5 ± 3.0	90.4 ± 2.9	n.s.	-
$CTRL_{TT}$ time (h:min:s)	1:14:39 ± 0:04:31	1:31:43 ± 0:06:54	<0.001	very large

* = the assumption of equality of variances was violated and thus non-parametric Mann-Whitney-U test was performed. Abbreviations: PTRS = peak treadmill running speed; abs = absolute; rel = relative; VO_{2peak} = peak oxygen consumption; VT-1 = First ventilatory threshold; RCP = Respiratory compensation point; $CTRL_{TT}$ = Control time trial; Data represented as mean ± SD.

Despite the differences in anthropometric, training, and performance parameters between the sexes, there were neither significant differences in perceived fatigability and observed pacing behaviour during time trials nor in running performance after controlling for sex-dependent differences in body composition. Thus, psychophysiological responses to running in a tapered condition *versus* running with LMMF and EIMD are hereafter presented for female and male participants combined.

Drop-Jump Protocol

Jump-Height

There was a significant effect of time on jump-height during the drop-jump protocol ($F_{3.5,\ 72.5}$=9.2; p=0.000; η_p^2=0.31) with significantly lower jump-heights after 80 drop-jumps compared to the first 40 drop-jumps. (See figure 11 a)).

Delayed Onset of Muscular Soreness (DOMS)

There was a significant treatment × time interaction effect on DOMS ($F_{2.6,\ 55.4}$=4.4; p=0.010; η_p^2=0.17). The investigation of simple (main) treatment effects showed significantly greater DOMS at every 12-hour interval ($F_{1,\ 21}$>18.5; p<0.001; η_p^2>0.47) peaking 36-hours after the intervention ($F_{1,\ 21}$=198.1; p<0.001; η_p^2>0.90). DOMS changed significantly during the recovery from the intervention ($F_{3.4,\ 71.5}$=20.3; p=0.000; η_p^2=0.49) as well as the control time trial ($F_{2.8,\ 58.6}$=28.6; p=0.000; η_p^2=0.58). (See figure 11 b)).

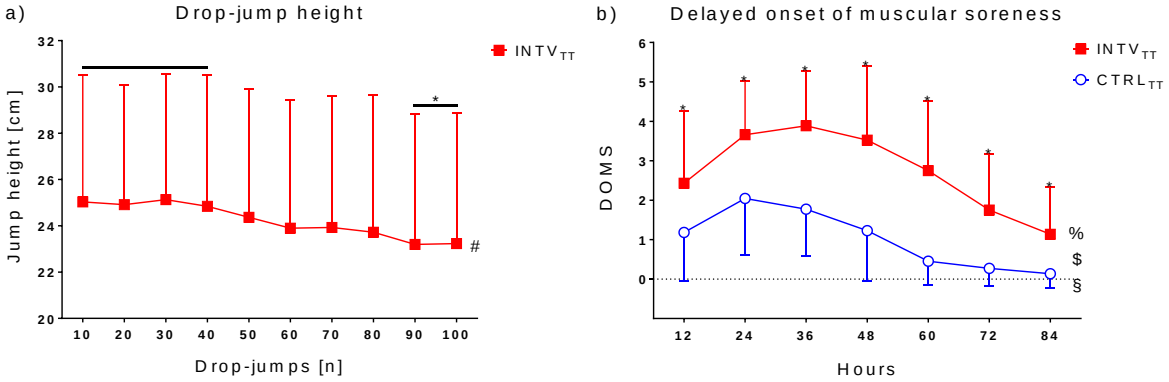

Figure 11: Decline in jump height over time during the drop-jump protocol and development of delayed onset of muscular soreness in the 84-hour recovery period.

Abbreviations: # = main time effect; & = main trial effect; % = time × group interaction effect; $ = simple (main) time effect for intervention trials; § = simple (main) time effect for control trials; * = simple (main) trial effect.

Psychophysiological Responses to Intervention and Running Economy Test

A summary of perceptual responses to the drop-jump protocol and its subsequent impact on isokinetic power output of knee extensors, psychophysiological responses during the running economy test, and time trial performance is provided in table 12.

Table 12: Summary of perceptual responses to the drop-jump protocol and its subsequent changes in isokinetic power output, running economy, and time trial performance.

	Control (n=22)	Intervention (n=22)	p-value	Effect size
Drop-jump protocol				
Perceptual responses				
Muscle discomfort pre*	1.0 ± 0.9	0.7 ± 0.6	n.s.	-
Muscle discomfort post*	0.8 ± 0.8	2.4 ± 1.7	<0.001	large
Unpleasantness pre*	0.8 ± 0.7	0.8 ± 0.8	n.s.	-
Unpleasantness post*	0.8 ± 0.7	2.3 ± 1.6	<0.001	large
Isokinetic power output test				
Knee extension pre* (W)	152 ± 40	150 ± 45	n.s.	-
Knee extension post* (W)	150 ± 40	133 ± 45	<0.001	large
Running economy test				
Oxygen cost ($ml \cdot kg^{-1} \cdot km^{-1}$)	199 ± 11	205 ± 14	0.005	small
Energy cost ($kcal \cdot kg^{-1} \cdot km^{-1}$)	0.99 ± 0.05	1.03 ± 0.07	0.002	medium
Carbohydrate usage ($g \cdot min^{-1}$)	2.74 ± 0.78	3.17 ± 0.96	0.013	small
Heart rate (bpm)	158 ± 11	164 ± 11	0.001	small
Perceptual responses				
Perceived physical strain	12.3 ± 1.9	13.5 ± 1.9	0.001	medium
Perceived mental strain	6.2 ± 1.5	7.7 ± 1.6	<0.001	medium
Valence	2.9 ± 1.3	1.5 ± 1.6	<0.001	medium
Felt activation	3.8 ± 1.0	4.0 ± 0.7	n.s.	-
Time trial times				
TT time (h:min:s)	1:23:11 ± 0:10:25	1:26:37 ± 0:11:36	<0.001	small
2^{nd} half / 1^{st} half split	0.1 ± 1.1	1.4 ± 2.2	0.003	medium

* = simple (main) trial effects (using partial eta squared as effect size measure) of two-way repeated measures ANOVA after significant treatment * time interaction effect. TT= Time trial. Data represented as mean ± SD.

Time Trial Times

Times taken to complete the $CTRL_{TT}$ and $INTV_{TT}$ were 1h:23min:11s ± 0h:10min:25s and 1h:26min:37s ± 0h:11min:36s, respectively. The average time difference was 3.96%. The mechanistic inference for this difference is most likely substantially negative (99.9% most likely substantially negative, 0.1% most unlikely trivial, and 0.0% most unlikely substantially positive).

Individual Responses to Intervention and Experimental Time Trials

For an individual distribution of responses to drop-jump protocol in knee extensor power output, 20-km time trial time, and 10-km time trial splits see figure 12.

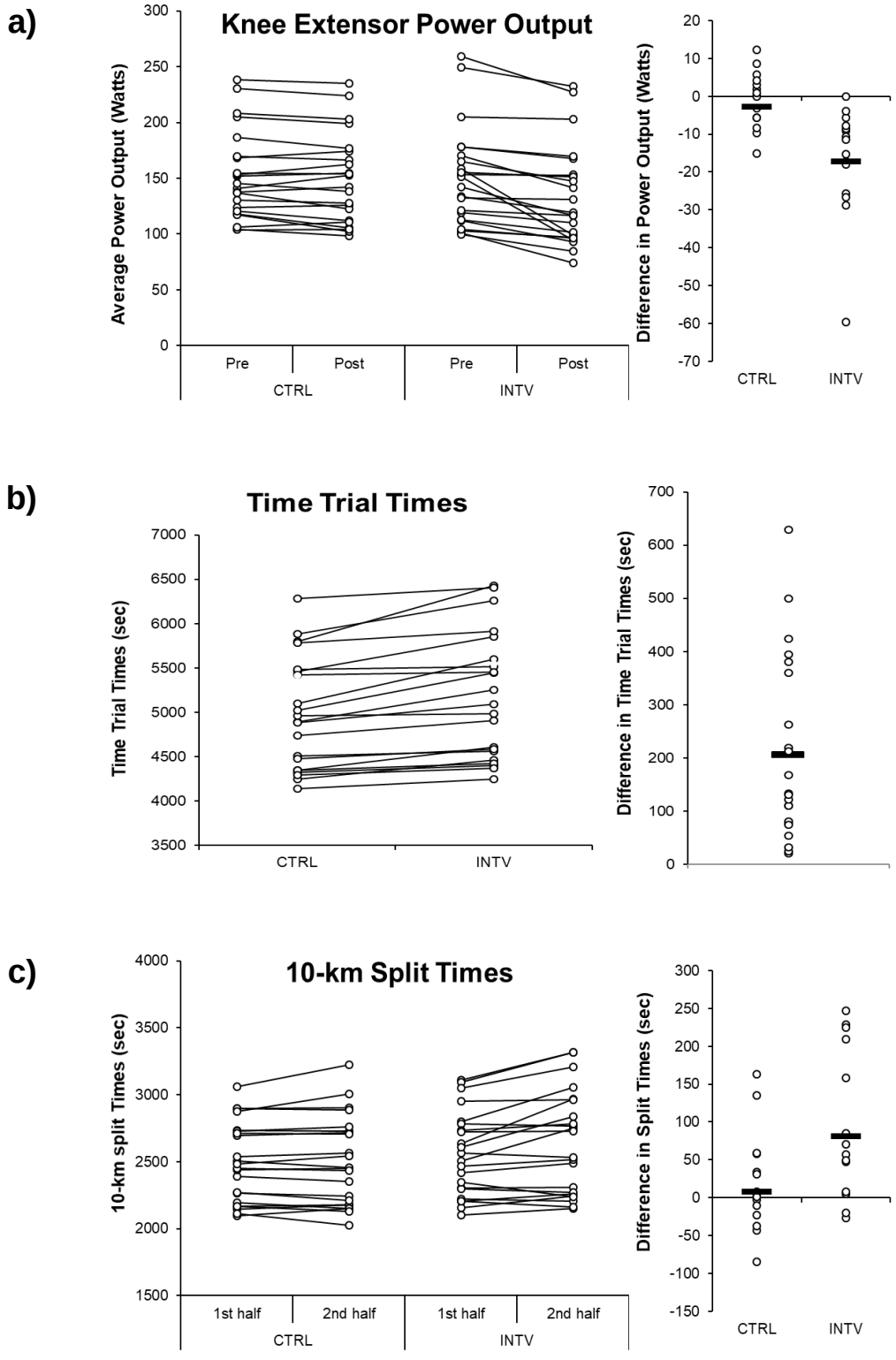

Figure 12: Distribution of individual responses to drop-jump protocol in knee extensor power output, 20-km time trial time, and 10-km time trial splits.

Abbreviations: CTRL = Control trial, INTV = Intervention trial; Sec = seconds.

Haematological Data

Leucocytes

There was a significant treatment × time interaction effect for blood leucocyte count ($F_{2.6, 55.2}$=6.5; p=0.001; η_p^2=0.24). The investigation of simple (main) treatment effects showed that runners had significantly greater blood leucocyte count after the drop-jump protocol ($F_{1, 21}$=16.2; p=0.001; η_p^2=0.44) and after 30 minutes of recovery (F1, 21=14.5; p=0.001; η_p^2=0.41) from the intervention time trial compared to the control time trial. Blood leucocyte concentrations increased significantly over time peaking after 30 min of recovery in both treatment groups with simple (main) time effects of ($F_{2.4, 50.0}$=53.4; p=0.000; η_p^2=0.72) in the control trial and ($F_{1.7, 35.1}$=37.9; p=0.000; η_p^2=0.64) in the intervention trial, respectively. (See figure 13 a)).

Neutrophils

There was a significant treatment × time interaction effect for blood neutrophil count ($F_{2.0, 43.0}$=7.9; p=0.001; η_p^2=0.28). The investigation of simple (main) treatment effects showed that runners had significantly greater blood neutrophil count after the drop-jump protocol ($F_{1, 21}$=12.1; p=0.002; η_p^2=0.37), at the finish of the 20-km time trial ($F_{1, 21}$=4.5; p=0.045; η_p^2=0.18) and after 30 minutes of recovery ($F_{1, 21}$=16.16; p=0.001; η_p^2=0.44) from the intervention time trial compared to the control time trial. Blood neutrophil count increased significantly over time peaking after 30 min of recovery in both treatment groups with simple (main) time effects of ($F_{2.3, 49.2}$=57.3; p=0.000; η_p^2=0.73) in the control trial and ($F_{1.6, 31.6}$=46.9; p=0.000; η_p^2=0.69) in the intervention trial, respectively. (See figure 13 b)).

Cortisol

There was a significant treatment × time interaction effect for blood cortisol concentrations ($F_{2.1, 43.8}$=7.1; p=0.002; η_p^2=0.25). The investigation of simple (main) treatment effects showed that runners had significantly lower blood cortisol concentrations after the drop-jump protocol ($F_{1, 21}$=12.5; p=0.002; η_p^2=0.37) and the

running economy assessment ($F_{1, 21}$=4.7; p=0.042; η_p^2=0.18), but significantly greater blood cortisol concentrations after 10 km ($F_{1, 21}$=4.4; p=0.048; η_p^2=0.17) and at the finish of the 20-km intervention time trial ($F_{1, 21}$=7.0; p=0.016; η_p^2=0.25) compared to the control time trial. Blood cortisol concentrations increased significantly over time peaking after 30 min of recovery in both treatment groups with simple (main) time effects of ($F_{2.3, 49.1}$=89.0; p=0.000; η_p^2=0.81) in the control trial and ($F_{2.0, 42.3}$=111.2; p=0.000; η_p^2=0.84) in the intervention trial, respectively. (See figure 13 c)).

Interleukin-6

The assumption of normal distribution in blood interleukin-6 concentrations was violated. Thus, non-parametric analysis of variance after aligned rank transformation was conducted. There was a significant treatment × time interaction effect for blood interleukin-6 concentrations (ART: $F_{2.1, 43.6}$=8.3; p=0.001; η_p^2=0.28). However, follow-up tests of simple (main) treatment effects did not reach statistical significance. Blood interleukin-6 concentrations increased significantly over time and peaked at the finish of the 20-km time trials respectively increasing by two-fold ($F_{1.8, 38.2}$=5.2; p=0.012; η_p^2 =0.20) and three-fold ($F_{3.0, 62.4}$=25.0; p=0.000; η_p^2=0.54) during the control and intervention trial. (See figure 13 d)).

Haematological Indicators of Muscle Damage and Physiological Strain

Graphical depictions of haematological markers of muscle damage, muscle metabolic strain, and endocrinological stress during experimental trials are summarised in figure 13.

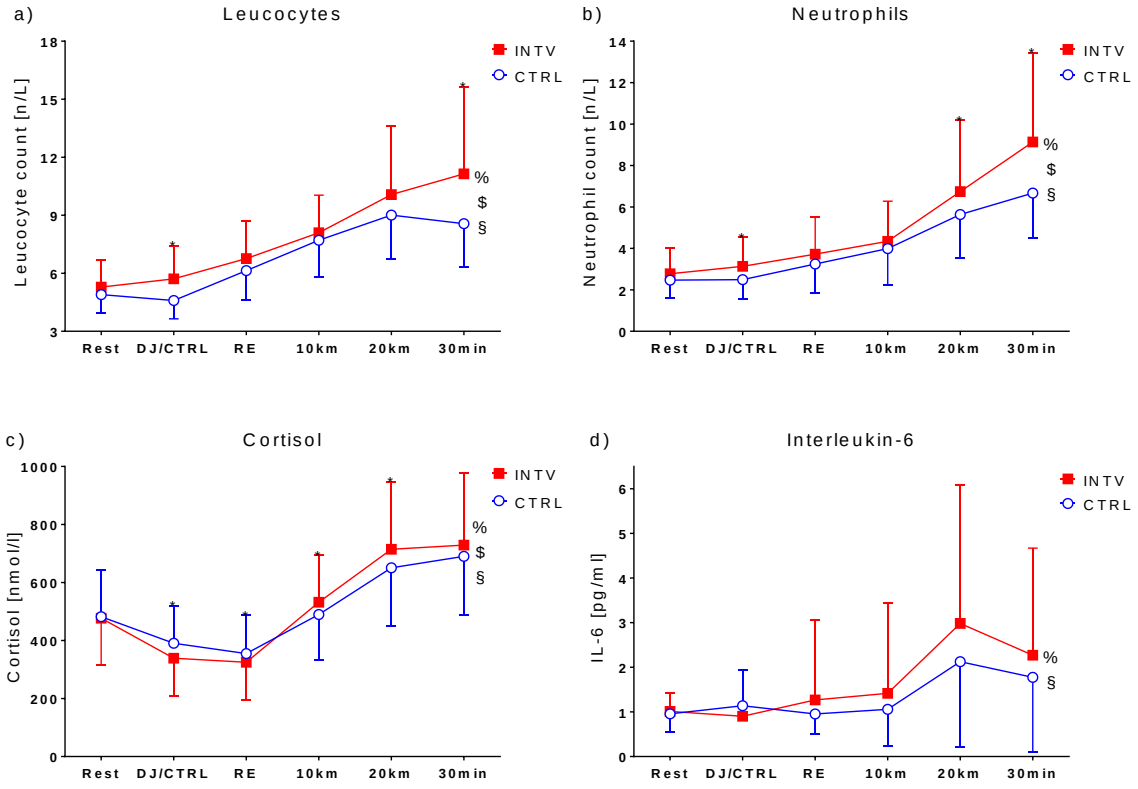

Figure 13: Differential responses in haematological markers of muscle damage, muscle metabolic strain, and endocrinological stress response during experimental trials.

Note: Blood interleukin-6 concentrations violated the assumption of normal distribution and thus non-parametric ANOVA after aligned rank transformation was conducted. Despite a significant interaction effect, simple main treatment effects did not reach significance.

Abbreviations: # = main time effect; & = main trial effect; % = time × group interaction effect; $ = simple (main) time effect for intervention trials; § = simple (main) time effect for control trials; * = simple (main) trial effect.

Perceptual Data

All perceptive data were additionally analysed with a non-parametric factorial procedure (ART) using common analysis of variance after align rank transform. The results are provided in squared brackets after the parametric test results in parenthesis.

Perceived Physical Strain (6-20 Borg scale)

There was a trend for a treatment × time interaction effect in perceived physical strain during the intervention trial with higher scores during the start phase and increasingly

similar scores towards the end ($F_{5.4, 113.0}$=2.1; p=0.063; η_p^2=0.09) [ART: $F_{6.0, 126.8}$=1.8; p=0.104; η_p^2=0.08]. However, the main treatment effect was significant with greater perceived physical strain during the intervention trial compared to the control trial ($F_{1, 21}$=12.0; p=0.002; η_p^2=0.36) [ART: $F_{1, 21}$=9.9; p=0.005; η_p^2=0.32]. Perceived physical strain increased significantly over time and in accordance with course profile in both control and intervention trial ($F_{3.6, 75.5}$=55.8; p=0.000; η_p^2=0.73) [ART: $F_{3.9, 81.1}$=53.8; p=0.000; η_p^2=0.72]. (See figure 14 a)).

Perceived Mental Strain (0-14 Borg scale)

There was a trend for a treatment × time interaction effect in perceived mental strain during the intervention trial with higher scores during the start phase and increasingly similar scores towards the end ($F_{5.5, 115.4}$=1.9; p=0.089; η_p^2=0.08) [ART: $F_{6.1, 127.8}$=2.0; p=0.064; η_p^2=0.09]. Neither was there a significant main treatment effect between the intervention and control trial ($F_{1, 21}$=2.9; p=0.101; η_p^2=0.12) [ART: $F_{1, 21}$=2.2; p=0.154; η_p^2=0.09]. However, perceived mental strain increased significantly over time and in accordance with course profile in both control and intervention trial ($F_{3.7, 78.6}$=50.9; p=0.000; η_p^2=0.71) [ART: $F_{4.5, 94.4}$=54.9; p=0.000; η_p^2=0.72]. (See figure 14 b)).

Valence (FS)

There was a trend for a treatment × time interaction effect in valence during the intervention trial with lower scores during the start phase and increasingly similar scores towards the end ($F_{4.7, 99.0}$=2.0; p=0.084; η_p^2=0.09) [ART: $F_{4.6, 96.6}$=1.9; p=0.111; η_p^2=0.08]. However, the main treatment effect was significant with greater negative valence during the intervention trial compared to the control trial ($F_{1, 21}$=11.0; p=0.003; η_p^2=0.34) [ART: $F_{1, 21}$=8.5; p=0.008; η_p^2=0.26]. Valence deteriorated significantly over time and changed in accordance with course profile in both control and intervention trial ($F_{3.0, 63.7}$=30.0; p=0.000; η_p^2=0.59) [ART: $F_{3.6, 75.1}$=28.9; p=0.000; η_p^2=0.58]. (See figure 14 c)).

Felt Activation (FAS)

There was neither a significant treatment × time interaction effect ($F_{9, 189}$=1.5; p=0.166; η_p^2=0.07) [ART: $F_{5.5, 115.0}$=1.4; p=0.231; η_p^2=0.06], nor a main treatment effect for felt activation ($F_{1, 21}$=0.1; p=0.739; η_p^2=0.01) [ART: $F_{1, 21}$=0.3; p=0.574; η_p^2=0.01]. However, felt activation increased significantly over time and according to course profile in both control and intervention trial ($F_{2.6, 54.5}$=5.7; p=0.003; η_p^2=0.21) [ART: $F_{2.9, 75.1}$=11.8; p=0.000; η_p^2=0.36]. (See figure 14 d)).

Action Crisis (ACRISS)

There was no significant treatment × time interaction effect for perceived action crisis ($F_{3.2, 66.5}$=1.7; p=0.170; η_p^2=0.08) [ART: $F_{3.3, 69.3}$=2.3; p=0.075; η_p^2=0.10]. However, the main treatment effect was significant with greater perceived action crisis during the intervention trial compared to the control trial ($F_{1, 21}$=25.7; p=0.000; η_p^2=0.55) [ART: $F_{1, 21}$=27.6; p=0.000; η_p^2=0.57]. Perceived action crisis increased significantly over time and in accordance with course profile reaching peak values during the second climb in both control and intervention trial ($F_{2.2, 45.5}$=13.7; p=0.000; η_p^2=0.40) [ART: $F_{2.3, 47.7}$=13.3; p=0.000; η_p^2=0.39]. (See figure 14 e)).

Flow State (FSS)

There was no significant treatment × time interaction effect for perceived flow state ($F_{3.2, 66.6}$=1.1; p=0.363; η_p^2=0.05) [ART: $F_{3.6, 75.5}$=0.9; p=0.475; η_p^2=0.04]. However, the main treatment effect was significant with reduced perceived flow state during the intervention trial compared to the control trial ($F_{1, 21}$=13.4; p=0.001; η_p^2=0.39) [ART: $F_{1, 21}$=12.9; p=0.002; η_p^2=0.38]. Perceived flow state decreased significantly over time reaching minimum values during the second climb in both control and intervention trial ($F_{2.1, 44.5}$=11.8; p=0.000; η_p^2=0.36) [ART: $F_{2.3, 46.8}$=10.4; p=0.000; η_p^2=0.33]. (See figure 14 e)).

Sensory, Affective, and Cognitive Indicators of Perceived Fatigability

Graphical depictions of differential responses in sensory-discriminatory, affective-motivational, and cognitive-evaluative indicators of perceived fatigability during experimental trials are summarised in figure 14.

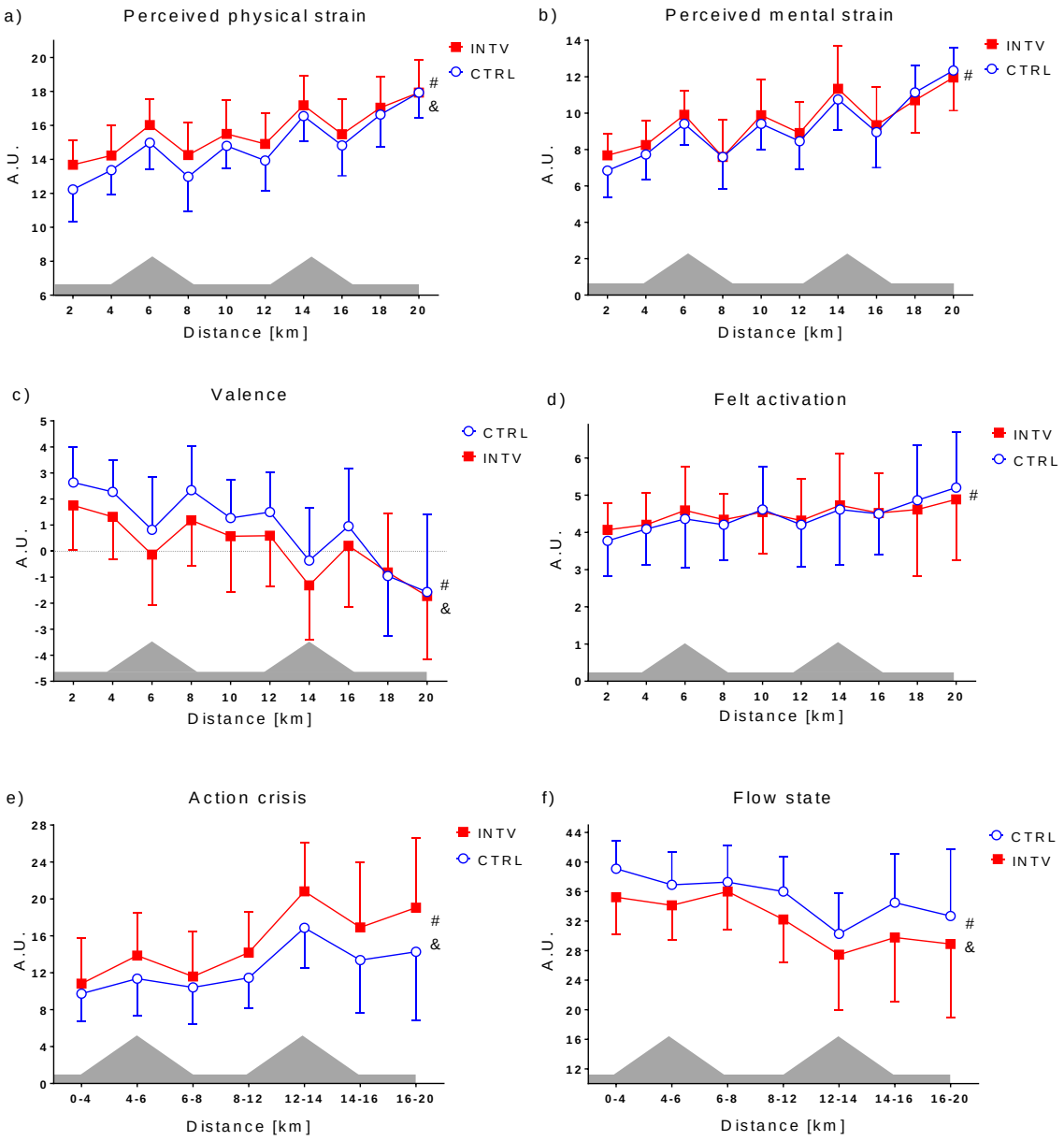

Figure 14: Differential responses in sensory, affective, and cognitive markers of perceived fatigability during experimental trials.

Note the differences in the x-axes of action crisis and flow state due to different sampling times.

Abbreviations: # = main time effect; & = main trial effect.

Performance Data

Split Time

There was a significant treatment × time interaction effect for 2-km split times ($F_{3.6, 76.4}$=4.9; p=0.002; η_p^2=0.19). The investigation of simple (main) treatment effects showed that runners took significantly more time during each 2-km section of the intervention trial. Except for the first downhill section ($F_{1, 21}$=5.2; p=0.033; η_p^2=0.20), all 2-km split times were highly significantly greater compared to the control trial ($F_{1, 21}$>13.9; p<0.001; η_p^2>0.40). Split times changed significantly in accordance with course profile in both treatment groups with simple (main) time effects of ($F_{1.7, 35.7}$=229.6; p=0.000; η_p^2=0.92) in the control ($F_{2.1, 44.1}$=161.4; p=0.000; η_p^2=0.89) and intervention trial, respectively. (See figure 15 a)).

Performance Fatigability

There was a significant main time effect for performance fatigability expressed as percentage increase in split time during the intervention time trial compared to the control time trial, which was assessed by means of one-way repeated measures analysis of variance ($F_{4.1, 83.5}$=4.0; p=0.005; η_p^2=0.16). The investigation of simple (main) time effects found no significant effects at 2-km intervals. (See figure 15 b)).

Heart Rate

There was a significant treatment × time interaction effect for average heart rate at 2-km intervals ($F_{3.3, 68.8}$=2.7; p=0.045; η_p^2=0.12). The investigation of simple (main) treatment effects showed a trend for significantly lower heart rates in runners during the intervention trial from the second climb onwards ($F_{1, 21}$=3.8; p=0.066; η_p^2=0.15) and this reached statistical significance during the final 2-km segment compared to the control trial ($F_{1, 21}$=9.5; p=0.006; η_p^2=0.31). Average heart rates increased significantly over time and changed according to course profile in both treatment groups with simple (main) time effects of ($F_{3.8, 80.3}$=71.4; p=0.000; η_p^2=0.77) in the control ($F_{3.5, 61.9}$=53.0; p=0.000; η_p^2=0.72) and intervention trial, respectively. (see figure 15 c)).

Lactate

There was a significant treatment × time interaction effect for blood lactate concentrations ($F_{3.6,\ 76.4}$=4.9; p=0.002; η_p^2=0.19). The investigation of simple (main) treatment effects showed that runners had significantly lower blood lactate concentrations at the finish of ($F_{1,\ 21}$=9.4, p=0.006; η_p^2=0.31) and after 30 minutes of recovery from ($F_{1,\ 21}$=11.7, p=0.003; η_p^2=0.36) the 20-km intervention time trial compared to the control time trial. Blood lactate concentrations increased significantly over time peaking at the finish of the 20-km time trial in both treatment groups with simple (main) time effects of ($F_{2.3,\ 48.5}$=53.1; p=0.000; η_p^2=0.72) and ($F_{2.9,\ 60.7}$=30.4; p=0.000; η_p^2=0.59) in the control and intervention trial, respectively. (See figure 15 d)).

Indicators of Performance Fatigability

Graphical depictions of differential responses in markers of performance fatigability are summarised in figure 15.

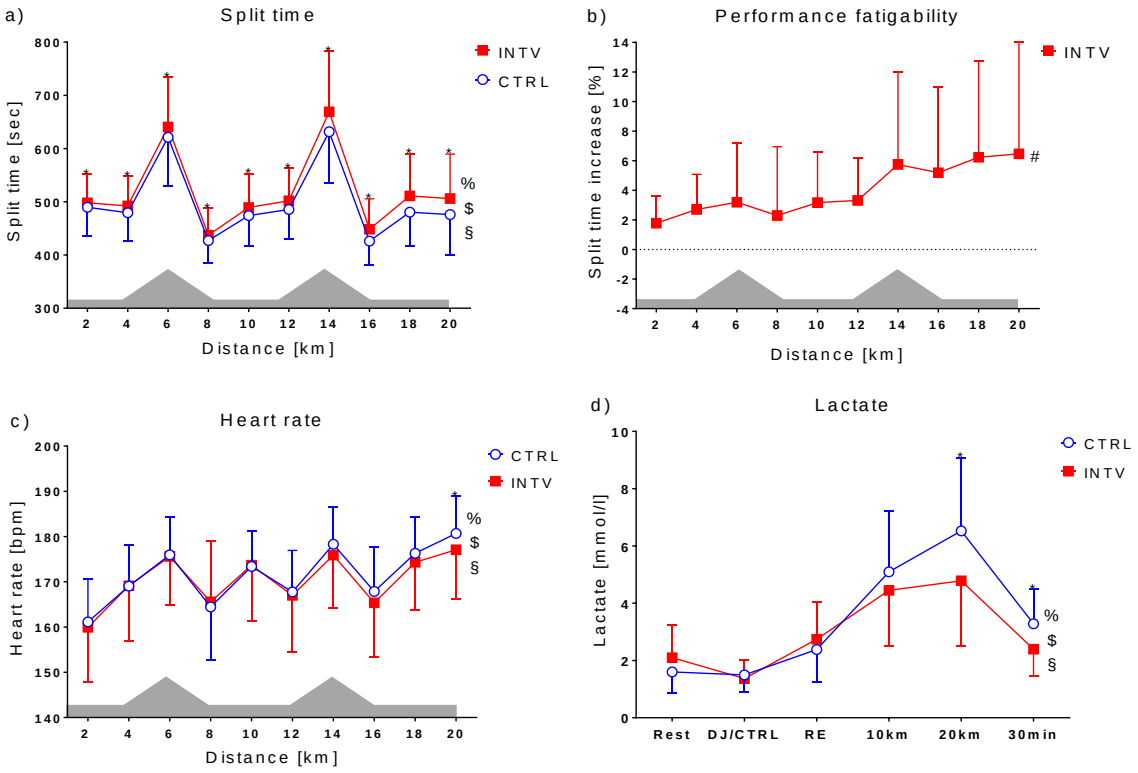

Figure 15: Differential responses in markers of performance fatigability during experimental trials.

Note the differences in the x-axes of blood lactate concentrations due to different sampling times. Performance fatigability is indicated by the percentage increase in split time during the intervention time trial compared to control time trial and assessed via one-way repeated measures ANOVA.

Abbreviations: # = main time effect; % = time x trial interaction effect; $ = simple (main) time effect for intervention trials; § = simple (main) time effect for control trials; * = simple (main) trial effect.

Performance, Perceptual, and Haematological Data

A summary of statistical findings in main performance, perceptive, and haematological variables is provided in table 13.

Detailed results of three-way (treatment × time × sex) repeated measures ANOVA are provided in supplementary material two.

Table 13: Summary of treatment, time, and interaction effects in main performance, perceptive, and haematological variables.

	Interaction Effect			Treatment Main Effect			Time Main Effect			Comment
	F	*p*	η_p^2	*F*	*p*	η_p^2	*F*	*p*	η_p^2	
Performance Data										
Split time	4.9	0.002	0.19		N/A			N/A		Increasingly greater split times over time during $INTV_{TT}$
Performance fatigability*		N/A			N/A		4.0	0.005	0.16	Greater performance fatigability over time during $INTV_{TT}$
Heart rate	2.7	0.045	0.12		N/A			N/A		Increasingly lower heart rates over time during $INTV_{TT}$
Lactate	4.9	0.002	0.19		N/A			N/A		Increasingly lower lactate levels over time during $INTV_{TT}$
Perceptual Data										
Perceived physical strain	2.1	*0.063*	0.09	12.0	0.002	0.36	55.8	<0.001	0.73	$INTV_{TT}$ > $CTRL_{TT}$; increase over time; change with profile
Perceived mental strain	1.9	*0.089*	0.08	2.9	0.101	0.12	50.9	<0.001	0.71	Increase over time; change with course profile
Valence	2.0	*0.084*	0.09	11.0	0.003	0.34	30.0	<0.001	0.59	$INTV_{TT}$ < $CTRL_{TT}$; decrease over time; change with profile
Felt activation	1.5	0.166	0.07	0.1	0.739	0.01	5.7	0.003	0.21	Increase over time; change with course profile
Perceived action crisis	1.7	0.170	0.08	25.7	<0.001	0.55	13.7	<0.001	0.40	$INTV_{TT}$ > $CTRL_{TT}$; increase over time; peak in 2nd uphill
Perceived flow state	1.1	0.363	0.05	13.4	0.001	0.39	11.8	<0.001	0.36	$INTV_{TT}$ < $CTRL_{TT}$; decrease over time; nadir in 2nd uphill
Haematological Data										
Leucocytes	6.5	0.001	0.24		N/A			N/A		Greater leucocyte count after DJ and TT during $INTV_{TT}$
Neutrophils	7.9	0.001	0.28		N/A			N/A		Greater neutrophil count after DJ and TT during $INTV_{TT}$
Interleukin-6(*)	8.3	0.001	0.28		N/A			N/A		Greater IL-6 levels after TT during $INTV_{TT}$
Cortisol	7.1	0.002	0.25		N/A			N/A		Greater cortisol levels during and after TT during $INTV_{TT}$

***** = Performance fatigability is indicated by the relative percentage increase in split times during ITT compared to CTT and assessed via one-way repeated measures ANOVA.
(*) = Blood interleukin-6 concentrations violated the assumption of normal distribution and thus non-parametric ANOVA after aligned rank transformation was conducted.
Abbreviations: TT=Time trial; $INTV_{TT}$=Intervention time trial; $CTRL_{TT}$=Control time trial; DJ=Drop-jump protocol

DISCUSSION

According to Enoka and Duchateau,[26] human fatigue is a psychophysiological symptom underpinned by interactions between (A) performance fatigability – the observed decline in an objective measure of endurance performance and (B) perceived fatigability – changes in the perceptions that regulate the integrity of the performer. The main findings of the current study provide supportive evidence for physiological and perceptual mechanisms underlying observed pacing behaviour and performance fatigability.

Physiological Effects of LMMF and mild EIMD on Performance Fatigability

First, the muscle lengthening contraction protocol induced muscle damage and the desired reduction in power output generating capacity of the knee extensors (see figure 11) without confounding effects of significant muscle metabolite accumulation and cardiovascular stress. The large decrease of 11% in isokinetic strength is consistent with mild EIMD (see table 12 and figure 12),[250] but only about half the decrease in isometric strength observed in other studies.[253,267] Although not directly comparable, the lesser strength loss is best explained by the high-performance calibre of our participants and the 'repeat-bout effect' rendering runners more resistant to muscle damage induced by muscle lengthening contractions than are moderately-trained cyclists.

Second, running with LMMF and mild EIMD stimulated amplified responses in cardiovascular, respiratory, and metabolic variables during the running economy test. A small increase in heart rate, oxygen consumption, and carbohydrate usage (including concomitant trend towards decreased fat usage) as well as a medium increase in energy cost of running collectively point towards increased physiological demands (see table 12). Thus, running with LMMF and mild EIMD at Δ 1/3 between the first-ventilatory threshold and respiratory compensation point (i.e. close to long-distance 'race-pace') increased absolute physiological demands and relative exercise intensity. Potential causes of augmented responses are manifold and, for example, could stem from inefficient alterations to running kinematics,[258,261,262] increased afferent feedback from group III and IV muscle afferent fibres resulting from structural disruption in extrafusal muscle fibres and local microvasculature,[279,280] and increased

compensatory efferent motor command to weakened locomotor muscles.[253,267] Both, afferent feedback and efferent feedforward components therefore seem to impact and modulate physiological responses and collectively support the notion of an underlying reafference principle in the allostatic regulation of homeostasis.[199]

Third, medium increases in blood leucocyte and neutrophil counts, interleukin-6, and cortisol concentrations were found during the $INTV_{TT}$ respectively indicating greater mobilisation and migration to damaged tissue (and thus indirectly EIMD), greater muscle metabolic strain,[281,282] and endocrinological stress (see figure 13 and table 13). The absolute values in these variables closely resemble those observed after 'real-world' half-marathon events.[283] Together, leucocytosis, neutrophilia, and pro-inflammatory immune response reflect the exacerbated physiological strain of running with LMMF and mild EIMD.[284,285] Additionally, endocrinological stress response was aggravated despite substantial performance decrement during the $INTV_{TT}$, thereby clearly indicating a non-adaptive distress response.[139,221] Collectively, the previously described responses constitute a physiological milieu that is likely not conducive to high performance as evidenced by the one-tailed detrimental effects of LMMF and mild EIMD on exercise performance (see figure 12).

Accordingly, we suggest that muscle damage and the amplified physiological responses to running with LMMF and mild EIMD *per se* have a debilitating effect on endurance performance due to increased absolute physiological demand and relative exercise intensity conveyed by peripheral and central fatigue mechanisms.[66,286] Time trial performance during $INTV_{TT}$ was constrained from the start indicating restricted work capacity and, potentially underpinned by accumulating EIMD, deteriorated further excessively from the second uphill onwards (see figure 15 a) and b)). Del Coso et al.[287] observed a similar differential pattern of pacing behaviour in marathon runners either maintaining (i.e. even split) or reducing (i.e. positive split) running speed and found significant correlations between increased performance fatigability and indirect markers of muscle damage. However, running with LMMF and mild EIMD also elicited potentially negative effects on performance fatigability via deterioration in sensory, affective, and cognitive processes hypothesised to underpin perceived fatigability.

Perceptual Effects of LMMF and mild EIMD on Performance Fatigability

First, a large increase in muscle discomfort was found in response to the muscle lengthening contractions protocol, although absolute scores are still relatively mild. Also, medium increases in perceived physical and mental strain were observed in response to running with LMMF and mild EIMD during the running economy test (see table 12). Perceived physical and mental strain showed a trend towards greater perceived strain scores during the start of the $INTV_{TT}$, but eventually approached similar end-points (see figure 14 a) and b)). This is coherent with findings from de Morree et al.[267] suggesting that both are primary regulatory variables of pacing behaviour.[226]

A significant scale × treatment interaction effect ($F_{1,21}$=10.1; p=0.005; η_p^2 = 0.32) was observed with a large main treatment effect for greater perceived physical strain only during the $INTV_{TT}$ (see table 13). This points towards the accuracy of perceived physical strain in the psychophysiological integration of homeostatic disturbance and the ability of athletes to differentiate between perceived physical strain and perceived mental strain.[64,214] Further empirical support was recently provided by Girard et al.,[288] who observed associations between increased physiological disturbance and subsequent augmented exercise-related physical sensations and performance fatigability during mental effort-clamped 'all-out' sprints in hypoxia compared to normoxia. This suggests that under conditions of intensified muscular, respiratory, and/or thermal strain, perceived physical strain can dissociate from perceived mental strain and may play a mediatory role in performance regulation.

Second, in support of a mediatory role, large decreases in valence in response to the muscle lengthening protocol and to running with LMMF and mild EIMD during the running economy test (see table 12) were associated with increases in perceived strain variables. More specifically, a large main treatment effect of attenuated valence during the $INTV_{TT}$ was associated with a large main treatment effect of augmented perceived physical strain (see figure 14 c)). This attachment of valence to interoceptive stimuli facilitates the awareness of homeostatic disturbance and confirms the paramount importance of valence in interoception, awareness, and computation of conflicting motivational drives.[137,138] Thus, valence is suggested to be the driving force by which attentional focus is shifted from goal-driven towards stimulus-driven processes. It

thereby functions as an attractor in attention allocation and decision-making; and ultimately performance regulation.[31,135]

Third, the decrease of valence during the $INTV_{TT}$ was associated with large increases and decreases in action crisis and flow state, respectively (see figure 14 e) and f)). An action crisis has been defined as an intra-psychic conflict between further goal-pursuit and goal-disengagement resulting from negatively valenced events in goal-striving.[163] In an action crisis athletes deliberate anew the desirability and feasibility of the pursued and alternative goals, thereby undermining effective goal-striving.[164] In contrast, flow has been described as a state of optimal experience, where athletes are totally immersed in goal-striving and goal-attainment.[225] Accordingly, their mirror-like dynamic responses suggest they form opposing ends of the mindset spectrum[161] and clearly identify a shift from an implemental mindset cognitively tuned towards the "how" of behaviour to a deliberative mindset cognitively tuned towards the "why" of behaviour.

Critically, increased cost-benefit thinking of further goal-pursuit and goal-disengagement has been shown to vary independently from the linearly increasing physiological strain experienced during a marathon, thereby independently predicting performance decrement.[164,170] Within the sport domain, mediation analyses showed that an action crisis undermines performance by augmenting the endocrinological distress response,[164,224] while cross-lagged panel path analyses in the academic domain showed attenuation of goal-desirability and goal-attainability.[164,289] Thus, an action crisis can be understood as an antecedent in the goal-disengagement process by draining physiological and self-regulatory resources, eventually leading to the dissolution of the pursued goal.

Accordingly, during the second climb of the $INTV_{TT}$, the observed large deterioration and peak in action crisis indeed coincided with an excessive increment in performance fatigability, thereby suggesting that runners experienced a decisional conflict between further goal-pursuit and goal-disengagement just before they (had to) made the

decision to slow-down.[e] Supportive evidence (although only associative) for the notion that runners re-evaluated and partly disengaged from their focal stems from observations of increasingly lower heart rates and blood lactate concentrations towards the finish (see figure 15 c) and d)) as well as perceptual inflections towards more sustainable rates of change in valence and action crisis after the second climb (see figure 14).

CONCLUSION

The current findings demonstrate that running with LMMF and mild EIMD causes medium sized physiological and large sized perceptual effects that are associated with dynamic changes in performance fatigability and observed pacing behaviour; collectively closely resembling defining characteristics of the "hitting the wall" phenomenon.[290,291] It is further suggested that much of the variance in response to running with LMMF and mild EIMD can be explained by the dynamic and complex interactions between the investigated psychophysiological determinants of pacing behaviour and performance during prolonged endurance exercise. The applied three-dimensional framework of perceived fatigability provides a more comprehensive understanding of strain-perception-thinking-action coupling in centrally regulated and goal-directed exercise behaviour than the traditional Gestalt concept of perceived exertion.[292] In addition, both physiological and perceptual pathways are proposed to impact performance fatigability during running with LMMF and mild EIMD via (1) amplified physiological strain and non-adaptive endocrinological distress response and (2) increases in perceived fatigability. Specifically, it is hypothesised that an increase in perceived physical strain *antecedes* a decrease in valence, which in turn *antecedes* an increase in action crisis; eventually dissolving the initially aspired goal.

[e] A strength of this study was the use of a custom-made hand-held remote control in the investigation of maximal self-paced treadmill running behaviour. In contrast to cycling ergometer studies, where it is unclear whether participants intentionally or unintentionally change pace, runners in this study decisively had to use the remote control to adjust treadmill running speed. However, it remains unclear whether this decision was based on heuristic or rational computations, or a combination thereof.[32] The "(had to)" therefore refers to the hypothesised driving role of valence in an affect-heuristic triggered decision-making process.

The extent to which the observed data fit the hypothesised cause-effect relationships will be assessed in chapter five.[293]

CHAPTER FIVE

MODELLING PERCEPTION-ACTION COUPLING IN THE PHENOMENOLOGICAL EXPERIENCE OF “HITTING THE WALL” DURING LONG-DISTANCE RUNING WITH EXERCISE-INDUCED MUSCLE DAMAGE IN HIGHLY-TRAINED RUNNERS

ABSTRACT

“Hitting the wall” (HTW) can be understood as a psychophysiological stress process characterised by (A) discrete and poignant onset, (B) dynamic interplay between physiological, affective, motivational, cognitive, and behavioural systems, and (C) unintended alteration of pace and performance. A preceding article investigated the psychophysiological responses to 20-km self-paced treadmill time trials after producing exercise-induced muscle damage (EIMD) via a muscle-lengthening contraction protocol and observed all defining characteristics of HTW.

A five-step procedure was applied determining the extent to which the observed data fit the hypothesised cause-effect relationships. Running with EIMD negatively impacts performance fatigability via: (A) amplified physiological demands and a non-adaptive distress response and (B) deterioration in perceived fatigability: increase in perceived physical strain precedes decrease in valence, which in turn precedes increase in action crisis; eventually dissolving the initially aspired performance goal.

First, haematological indicators of EIMD predicted increased blood cortisol concentration, which in turn predicted increased performance fatigability. Second, perceived physical strain explained 44% of the relationship between haematological indicators of EIMD and valence; which in turn predicted increased action crisis; which in turn predicted increased performance fatigability. The observed data fitted the hypothesised dual-pathway model well with good model-fit indices throughout.

The hypothesised interrelationships between physiological strain, perception, and heuristic and deliberative decision-making processes in self-regulated and goal-directed exercise behaviour were applied, tested, and confirmed: amplified physiological strain and non-adaptive distress response as well as strain-perception-thinking-action coupling impact performance fatigability. The findings provide novel

insights into the psychophysiological processes that underpin the phenomenological experience of HTW and alteration in pacing behaviour and performance.

INTRODUCTION

The “hitting the wall” (HTW) phenomenon is a much talked about but poorly understood phenomenological experience that regularly occurs in prolonged endurance exercise. HTW can be understood as a psychophysiological stress process characterised by (A) discrete and poignant onset and duration, (B) dynamic and complex interplay between physiological, affective, motivational, cognitive, and behavioural systems, and (C) unintended alteration of pacing behaviour and performance deterioration.[290,291]

The sheer physicality of endurance sports, particularly long-distance running, ensures that HTW is tightly coupled to stimulus-driven processes resulting from extreme physical duress. The most popular physiological explanation for HTW is skeletal muscle glycogen depletion and hypoglycaemia.[170,294,295] However, a computational study by Rapoport[296] showed that glycogen storage capacity is only a performance limiting factor in runners with low and moderate aerobic capacities, but may not constrain performances of highly-trained long-distance runners. An alternative determinant of HTW is exercise-induced muscle damage (EIMD) caused by muscular exertion of unaccustomed exercise duration and/or intensity, especially when involving muscle lengthening contractions.[297] This notion is supported by length of longest training run predicting the likelihood of HTW[290,298] and correlations between deterioration in marathon running pace and indirect haematological markers of muscle damage.[287]

The performance level dependent nature of HTW[299,300] points toward the importance of salient primary appraisals of physiological sensations. However, the large context-dependent intra- and inter-individual variability in subsequent psychophysiological responses necessitates the integration of sentient secondary appraisals of psychophysiological perceptions into a more holistic multi-dimensional framework.[301]

Buman et al.[290] investigated the phenomenological characteristics of HTW in marathon runners and observed escalating generalised fatigue accompanied by unintentional slowing of running pace, increased desire to walk, and a shift from initially set performance goals to desire just finishing the race. Furthermore, runners experiencing

HTW felt more negatively impacted by these events and the authors interpreted the renegotiation of race aspirations as an adaptive behavioural response to exhaustion and performance deterioration as it did not *per se* increase the likelihood of race drop-out.[290]

In a subsequent study, Buman et al.[291] qualitatively explored the psychological and behavioural experiences of HTW. Raw data were thematised into five higher-order themes: (A) affective (e.g. frustration), (B) behavioural (e.g. pace disruption), (C) cognitive (e.g. changing goals), (D) motivational (e.g. desire to quit) and, (E) physiological (e.g. generalised and leg-related fatigue). As expected, physiological descriptors were reported most frequently,[71,302] but the authors concluded that the complex interactions of physiological, affective, motivational, cognitive, and behavioural perceptions truly underpin the goal-disengagement impulse associated with the HTW phenomenon.[291]

Goal-related cost-benefit thinking in goal-striving consistently peaks around the 32-km mark during a marathon before resolving closer to the finish line.[163,170,298] Thus, non-linear dynamics of a psychological crisis concord with linear dynamics of a physiological crisis during HTW, but can dissociate thereafter. More importantly, deliberating anew the desirability and feasibility of the initially aspired performance goal during a marathon independently predicted increased performance fatigability, thereby providing a psychobiological link between a mindset-shift in goal-striving and performance degradation.[164,170,224]

However, the above studies relied on correlational study designs, recreational runners, and low temporal resolution in perceptual and performance measures. These studies therefore provide limited information on pressing questions about (A) how hypothesised cause-effect relationships determining endurance performance come to be[219] and (B) how perception-action coupling underpins self-regulation and decision-making in pacing behaviour and endurance performance.[292]

The three-dimensional framework of centrally regulated and goal-directed exercise behaviour proposed in chapter one[199] was therefore applied to investigate alterations in pacing behaviour and endurance performance in highly-trained long-distance runners during a self-paced 20-km treadmill time trial after inducing EIMD by means of

a standardised drop-jump protocol.[297] The preceding study showed dynamic changes and interdependencies in physiological and perceptual effects on pacing behaviour and performance that are in full agreement with the defining characteristics of the phenomenological experience of HTW described previously.

In this chapter, structural equation modelling (SEM)[f] was used to determine the extent to which the observed data fit the following hypothesised cause-effect relationships of the dual-pathway model outlined below and which are described in detail in the preceding chapter.[297] Due to the limited sample size, a simplified version was tested that focused on the haematological indicators of EIMD and thus the muscle damage component rather than the combined effects of LMMF and EIMD. Differential responses in haematological indicators of EIMD are hypothesised to precede (1) amplified physiological and non-adaptive endocrinological distress response (indicated by blood cortisol concentrations) and (2) increase in perceived physical strain; which in turn mediates a decrease in valence; which in turn precedes an increase in action crisis; and both physiological and perceptual effects precede performance fatigability.

METHODS

Twenty-two (11 females) highly-trained (performance level 4) runners completed two maximal self-paced 20 km treadmill time trials over a simulated profiled course in a counterbalanced crossover design: (A) in a tapered condition and (B) with EIMD produced by a standardised drop-jump protocol. Indicators of muscle damage, muscle metabolic strain, and endocrinological stress were assessed to investigate the physiological effects; and a three-dimensional framework of perceived fatigability[199]

[f] "Structural equation modelling (SEM) is a causal inference method that combines testable causal hypotheses based on scientific knowledge and empirical data as input and produces quantitative causal claims, conditional on the input assumptions, as output. The statistical inference is based on a simultaneous analysis of the entire system of variables determining the extent to which the observed data fit the hypothesised a priori specifications. It thereby provides a higher-level perspective in the analysis and evaluation of entire models. Another key component to theory development is mediation analysis, which aims to clarify how cause–effect relationships come to be, and which are therefore theoretical formulations for unidirectional relationships. Thus, specifically when used in conjunction with an experimental design, the capabilities to formalise and implement causal inference makes SEM an indispensable tool in causal hypothesis testing and mediation analysis; its potential for theory development is even greater."[224]

emphasising the distinct contributions of sensory, affective, and cognitive processes was applied to investigate the perceptual effects of running with EIMD on performance fatigability. The methods of preliminary and experimental procedures as well as the outcome of conventional statistical analyses have been described in detail in the preceding chapter.[297] A five-step procedure was applied to ascertain that the theorised relationships were statistically robust enough for the structural modelling process.[224]

In step one, conventional statistical analyses and visual inspection of data took place. Main study variables that showed significant treatment × time interaction or main treatment effects were considered potential explanatory moderators and mediators of defended regulatory variables and observed trial-related differences in performance fatigability. To overcome discrepancies in temporal dynamics and sampling points as well as non-linearity in psychophysiological responses, trial-related differences in area under the curve[g] (Δ AUC) were calculated for each main study variable before proceeding with correlation and regression analyses.

In step two, zero-order correlations and linear regressions were computed. Zero-order correlations were calculated between Δ AUC in main study variables determining the strength of association between each variable pair. Significant correlations between variable pairs were followed-up with linear regression analyses, thereby allowing predictions to be made.

In step three, mediation analysis was conducted when a predictor variable was correlated to the proposed direct outcome variable and another dependent variable. Mediation analysis measures the significance of the indirect effect and the extent to which the mediator variable accounts for the relationship between the predictor and the outcome variable. Mediation analysis is particularly appropriate to use when: (A) the predictor variable is experimentally manipulated and the mediator variable measured and (B) the dynamic change in the predictor variable precedes the dynamic change in the mediator variable; and both precede the outcome variable.[229,233]

[g] To reflect the lag period in haematological indicators of EIMD and endocrinological strain, areas under the curve of haematological variables were integrated from pre-time trial to post-recovery period.

In step four, multiple hierarchical regression analyses were conducted between pairs of study variables hypothesised to be linked in a temporal order and separate for each pathway hypothesised to impact performance fatigability:[h] Path one: (A) blood leucocyte count and blood cortisol concentration, (B) blood neutrophil count and blood cortisol concentration, and (C) blood cortisol concentration and performance fatigability. Path two: (D) blood leucocyte count and perceived physical strain, (E) blood neutrophil count and perceived physical strain, (F) perceived physical strain and valence, (G) valence and action crisis, and (H) action crisis and performance fatigability. Every variable pair was analysed by means of stepwise longitudinal regression analysis to ensure that the relationship between the direct predictor and outcome variable in each pairwise comparison was independent of descriptor and training variables as well as conventional predictors of endurance performance. Age and weight were entered in step one. Weekly mileage and volume of other aerobic training were entered in step two. VO_{2peak} relative to body mass and running economy (i.e. energy cost of running at Δ 1/3 between the first-ventilatory threshold and the respiratory compensation point) were entered in step three. Lastly, the direct predictor of the outcome variable of interest was entered in step four.

In step five, main study variables were fitted into a singular structural path model. Despite the small sample size (n=22), SEM was deemed appropriate as the variables included in the tested model: (A) showed significant interaction or main treatment effects in conventional statistical analyses, (B) showed significant zero-order correlations, (C) showed inter-individual homogeneity in responses, (D) showed trial-dependent changes that were in full agreement with the theoretical predictions, and lastly (E) as a result of the relatively low complexity level of the tested model.[234]

Statistical Analysis

All conventional statistical analyses were conducted with IBM® SPSS® (Version 24.0, Chicago, IL, USA). Slopes and AUC were calculated using GRAPHPAD PRISM® (Version 6.00 for Windows, GraphPad Software, La Jolla California, USA). Mediation analysis and structural equation modelling was performed using IBM® SPSS® Amos

[h] The direct predictive effects of blood leucocyte and neutrophil count on blood cortisol concentration and perceived physical strain were assessed independently due to multi-collinearity.

(Version 24.0, Chicago, IL, USA). Model fits were estimated using the maximum likelihood method and results are represented as standardised estimates. Significance of the indirect effect within mediation analysis was estimated by means of bootstrap sampling and bias-corrected 95% confidence intervals based on 2.000 bootstrap samples.[236] Statistical significance was accepted at $p < 0.05$.

RESULTS

Step 1: Visual Inspection and Conventional Statistical Analyses

Trial-related differences in main study variables showing significant treatment × time interaction or main treatment effects are shown in figure 16.

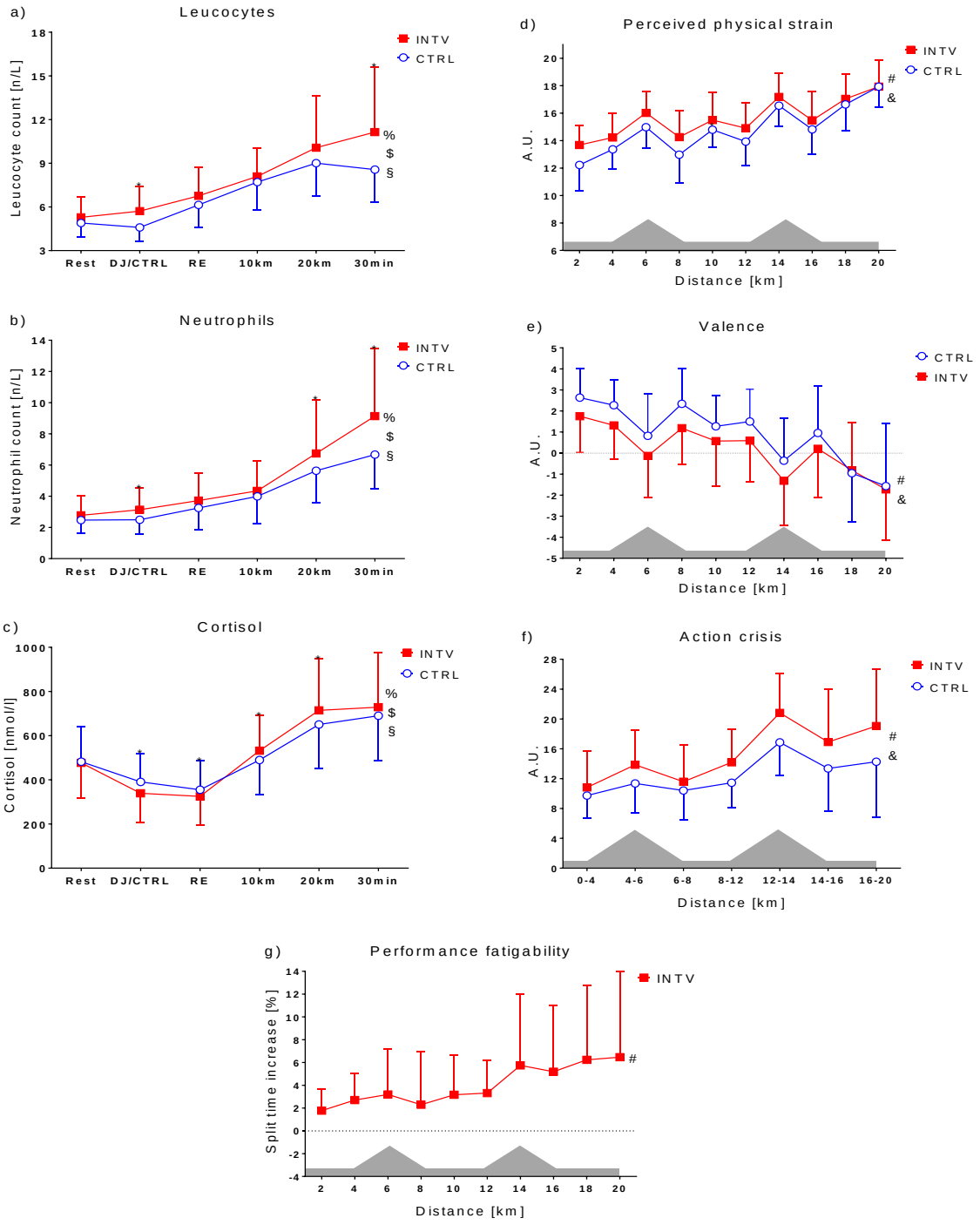

Figure 16: Trial-related differences in main study variables in response to running with mild exercise-induced muscle damage.

Note the differences in x-axes of action crisis and haematological variables due to different sampling times.

Abbreviations: % = treatment × time interaction effect; $ = simple (main) time effect for intervention trials; § = simple (main) time effect for control trials; * = simple (main) treatment effect; & = main treatment effect; # = main time effect; DJ/CTRL = Drop-jump protocol versus control; RE = Running economy test; CTRL = Control time trial; INTV = Intervention time trial.

Step 2: Correlation and Linear Regression

Zero-order correlations among the major study variables are provided in table 14.

Table 14: Zero-order correlations between trial-related differences in area under the curve of haematological indicators of exercise-induced muscle damage, blood cortisol concentration as well as sensory, affective, and cognitive indicators of perceived fatigability.

	1	2	3	4	5	6
1. Δ AUC - Leucocytes	-					
2. Δ AUC - Neutrophils	.98**	-				
3. Δ AUC - Cortisol	.56**	.59**	-			
4. Δ AUC - Perceived physical strain	.54*	.52*	.33	-		
5. Δ AUC - Valence	-.65**	-.69**	-.58**	-.78**	-	
6. Δ AUC - Action crisis	.45*	.47*	.30	.40	-.51*	-
7. Performance fatigability	.36	.39	.51*	.12	-.36	.57**

Abbreviations: Δ AUC=Trial-related difference in area under the curve. ** Correlation is significant at the 0.01 level (2-tailed). * Correlation is significant at the 0.05 level (2-tailed).

Significant linear relationships were found between haematological indicators of EIMD (i.e. Δ AUC in blood leucocyte and neutrophil count) and Δ AUC in blood cortisol concentration, which in turn showed a significant linear relationship with performance fatigability ($\beta = 0.51$; $p = 0.015$). However, Δ AUC in haematological indicators of EIMD also showed significant linear relationships with Δ AUC in perceived physical strain, valence, and action crisis (although only weakly with the latter). Furthermore, there were significant linear relationships in the hypothesised temporal order between the Δ AUC in perceived physical strain and valence ($\beta = -0.78$; $p = <0.001$), valence and action crisis ($\beta = -0.51$; $p = 0.015$), and action crisis and performance fatigability ($\beta = 0.57$; $p = 0.006$). The simultaneous significant linear relationships between Δ AUC in haematological indicators of EIMD, perceived physical strain, and valence required the examination of a potential indirect effect of perceived physical strain on the relationship between haematological indicators of EIMD and valence.

Step 3: Mediation Analysis

In full agreement with our theory-driven approach, Δ AUC in perceived physical strain was a significant mediator as it explained 44% of the relationship between Δ AUC in haematological indicators of EIMD and valence. The significant total effect c (standardised estimate = -0.68**, p < 0.001) between Δ AUC in haematological indicators of EIMD and valence was reduced by a non-trivial amount to a significant direct effect c' (standardised estimate = -0.38**, p = 0.001) after controlling for the significant indirect effect a × b (standardised estimate = -0.24, p = 0.025). Statistical details are provided in figure 17 and table 15.

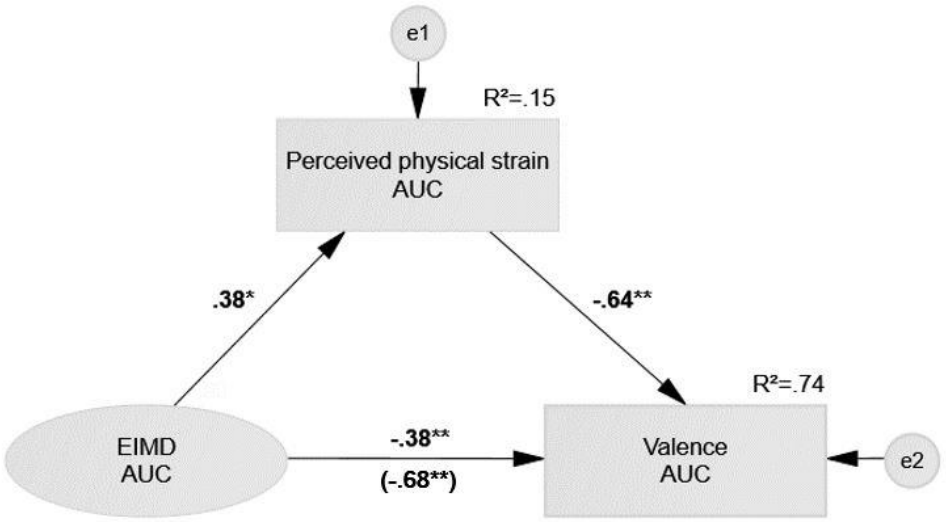

Figure 17: A mediation model of trial-related differences in area under the curve (Δ AUC) in haematological indicators of exercise-induced muscle damage (EIMD), perceived physical strain, and valence.

Note: Δ AUC of blood leucocyte and neutrophil count were used to indicate greater extent of EIMD during intervention trials. For graphical simplicity indicators and errors are not shown. Δ AUC of perceived physical strain was a significant mediating factor in the relationship between Δ AUC in haematological indicators of EIMD and valence

Abbreviations: Squares represent observed variables; Ovals represent latent variables; Single headed arrows represent regression paths; Bold regression paths are significant at * p < 0.05 and ** p < 0.01, respectively; EIMD = Exercise-induced muscle damage; AUC = Area under the curve; R^2 = total variance explained; e = residual error.

Table 15: Mediation analysis testing whether trial-related difference in area under the curve (∆ AUC) of perceived physical strain partly accounts for the association between ∆ AUC in haematological indicators of exercise-induced muscle damage and valence.

Effect	Standardised estimate	CR	SE	95%CI	p
Path c (total effect)	-.68	-4.16			<.001
Path a	.38	2.09			.036
Path b	-.64	-5.37			<.001
a × b (indirect effect)	-.24		.145	[-.022; -.563]	.025
Path c ' (direct effect)	-.38	-3.22			.001

Note: Maximum likelihood estimates are provided for paths c, a, b, and c'. For the standardised (a × b) indirect effect, bootstrap estimates with CIs are provided. Abbreviations: CR = Critical ratio; CI = Confidence interval; SE = Standard error.

Step 4: Multiple Hierarchical Regression Analyses

None of the control variables (i.e. age, weight, weekly mileage, other training, VO_{2peak}, and energy cost of running) significantly predicted any of the main study outcome variables (i.e. blood cortisol concentration, perceived physical strain, valence, action crisis, and performance fatigability). More importantly, all direct predictors (i.e. blood leucocyte count, blood neutrophil count, blood cortisol concentration, perceived physical strain, valence, and action crisis) continued to significantly predict the respective outcome variable (i.e. blood cortisol concentration, perceived physical strain, valence, action crisis, and performance fatigability) after controlling for anthropometric and training variables as well as conventional predictors of endurance performance. The results of the seven stepwise regression analyses are summarised in table 16.

Table 16: Multiple hierarchical regression analyses of trial-related differences in area under the curve (Δ AUC) of blood cortisol concentrations and performance fatigability as well as perceived physical strain, valence, action crisis, and performance fatigability in response to Δ AUC in the respective direct predictor variable.

	Leucocytes ⇨ Cortisol		Neutrophils ⇨ Cortisol		Cortisol ⇨ Performance fatigability		Leucocytes ⇨ Perceived physical strain		Neutrophils ⇨ Perceived physical strain		Perceived physical strain ⇨ Valence		Valence ⇨ Action crisis		Action crisis ⇨ Performance fatigability	
Predictor	ΔR^2	β	ΔR^2	β	ΔR^2	β	ΔR^2	β	ΔR^2	β	ΔR^2	β	ΔR^2	β	ΔR^2	β
Step 1	.056		.056		.002		.066		.066		.022		.081		.002	
Age		-.130		-.130		.002		-.255		-.255		.035		-.183		.002
Weight		.201		.201		.042		.036		.036		-.144		.211		.042
Step 2	.196		.196		.012		.011		.011		.005		.094		.012	
Weekly mileage		.172		.172		.150		.058		.058		.029		-.424		.150
Other training		.538		.538		.124		-.063		-.063		-.051		.161		.124
Step 3	.004		.004		.047		.220		.220		.148		.015		.047	
VO_{2peak}		-.069		-.069		-.182		.463		.463		-.175		.106		-.182
Economy		.067		.067		.244		-.514		-.514		.438		-.140		.244
Step 4	.372**		.395**		.291*		.307**		.276**		.528**		.210*		.461**	
Direct predictor		.708**		.726**		.626*		.643**		.608**		-.866**		-.504*		.755**

** Significant at the 0.01 level (2-tailed). * Significant at the 0.05 level (2-tailed).

Step 5: Structural Equation Modelling

Details of the full structural equation model are provided in figure 18. The dual-pathway model hypothesised to underpin increased performance fatigability in response to running with EIMD entails: (1) debilitative physiological effects via amplified physiological demand and non-adaptive endocrinological distress response and (2) debilitative perceptual effects via deterioration in sensory, affective, and cognitive processes hypothesised to underpin perceived fatigability.

First, an increase in Δ AUC in haematological indicators of EIMD significantly predicted non-adaptive endocrinological distress response indicated by an increase in Δ AUC in blood cortisol concentrations (standardised estimate = 0.69**; $p < 0.01$) despite reduced performance, which in turn significantly predicted increased performance fatigability (standardised estimate = 0.38*; $p < 0.05$).

Second, besides the significant mediatory role of perceived physical strain in the relationship between haematological indicators of EIMD and valence discussed previously, an increase in Δ AUC in haematological indicators of EIMD significantly predicted an increase in Δ AUC in perceived physical strain (standardised estimate = 0.52*; $p < 0.05$). An increase in Δ AUC in perceived physical strain significantly predicted a decrease in Δ AUC in valence (standardised estimate = -0.47*; $p < 0.05$). A decrease in Δ AUC in valence significantly predicted an increase in Δ AUC in perceived action crisis (standardised estimate = -0.51**; $p < 0.01$) and an increase in Δ AUC in perceived action crisis significantly predicted an increase in performance fatigability (standardised estimate = 0.46**; $p < 0.01$).

The observed data fitted the hypothesised dual-pathway model well with good model fit indices throughout and a 78% probability that the observed data fit the hypothesised cause-effect relationships: $\chi^2 = 7.277$, $p = .776$, $\chi^2/11 = .662$, NFI = .949, CFI = 1.000, RMSEA = .000 (95%CI = [.000, .156]; PCLOSE = .806), AIC = 55.277, SRMR = .068).[237]

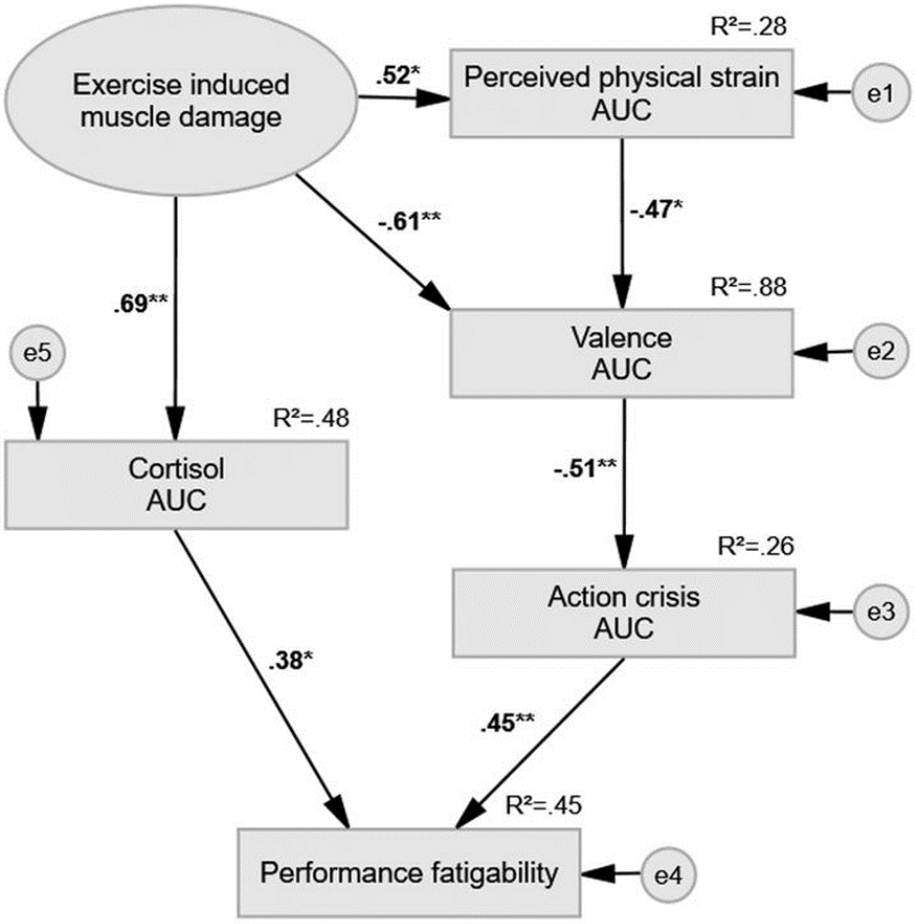

Figure 18: Structural equation model of physiological distress and perceived fatigability in response to running with mild exercise-induced muscle damage (EIMD) and the effects on performance fatigability.

Note: Trial-related differences in area under the curve of blood leucocyte and neutrophil count were used to indicate greater extent of EIMD during intervention trials. For graphical simplicity indicators and errors are not shown.

Abbreviations: Squares represent observed variables; Ovals are latent variables; Single headed arrows represent regression paths; Bold regression paths are significant at * $p < 0.05$ and ** $p < 0.01$, respectively; AUC = area under the curve; R^2 = total variance explained; e = residual error; Standardised maximum likelihood measures are used. χ^2 = Chi-square; NFI = normed fit index; CFI = comparative fit index; RMSEA = root mean square error of approximation; PCLOSE = p of close fit; AIC = Akaike information criterion; SRMR = standardised root mean square residual.

Model fit indices are: $\chi^2 = 7.277$, $p = .776$, $\chi^2/11 = .662$, NFI = .949, CFI = 1.000, RMSEA = .000 (95%CI = [.000, .156]; PCLOSE = .806), AIC = 55.277, SRMR = .068.

DISCUSSION

The present research applied SEM to measure the extent to which the observed data of the preceding chapter[297] fit the previously outlined psychophysiological cause-effect relationships hypothesised to shape the HTW phenomenon by combining causal (i.e. formalising and implementing testable causal hypotheses in an experimental design) and statistical (i.e. assessing the extent to which the experimental data fitted the hypothesised model) inference. Good model fit indices confirmed the credibility of the hypothesised model structure. The main findings were: (A) discrete and poignant event onset, (B) differential responses in haematological indicators of amplified muscular strain and non-adaptive endocrinological distress response, (C) sensory-discriminatory, affective-motivational, and cognitive-evaluative processes indicative of deterioration in perceived fatigability, and (D) unintended alteration of pacing behaviour and performance fatigability.

First, the muscle lengthening contraction protocol caused a large 11% decrease in power output generating capacity of the knee extensors, medium increases in haematological indicators of EIMD, and large increases in symptoms of delayed onset of muscular soreness in highly-trained runners; collectively confirming mild EIMD (for details see preceding chapter).[297] Still, strength loss was only about half of that observed in similar studies using moderately-trained cyclists.[253,267] Thus, given that highly-trained runners are arguably better adapted to mechanical strain through the repeat-bout effect, this might explain why the current study only observed trends towards significant zero-order correlations between haematological indicators of EIMD and performance fatigability.

However, the drop-jump protocol did induce small to medium increases in absolute cardiovascular, respiratory, and metabolic demands during steady-state running economy tests preceding the time trials, thereby indicating an increase in relative exercise intensity (for details see preceding chapter).[297] Running with mild EIMD therefore elicited a stressful physiological milieu that is not conducive to high-performance. In support, in the current study, differential responses in haematological indicators of EIMD predicted non-adaptive endocrinological distress response, which in turn predicted increased performance fatigability. Thus, the escalating leg-related and generalised fatigue experienced when HTW[291] are likely associated with greater

accumulation of EIMD, amplified physiological demands, and excessive endocrinological distress response during the second-half of marathons compared to half-marathons.[283] This notion is consistent with observational data showing correlations between greater decreases in marathon running pace after ≈25 km and haematological markers of muscle damage in moderately-trained runners who slowed down significantly more than those who were able to maintain their running speed.[287]

Second, the most prevalent cognitions described by runners in response to challenging exercise[71,302] are perceptions of salient physiological disturbance, and this particularly holds true under extreme physiological duress as experienced when HTW.[291] Another sentient characteristic of the phenomenological experience of HTW is that runners who HTW perceive these sensations to more negatively impact on their performance than those who do not, thereby becoming increasingly frustrated and discouraged.[290,291] This attachment of valence to homeostatic disturbance and perceived physical strain eventually undermines effective goal-striving by promoting an urge to disengage from further goal-pursuit.[136–138] In support, in the current study, differential responses in haematological indicators of EIMD predicted an increase in perceived physical strain as well as a decrease in valence. Importantly, perceived physical strain was a significant mediator explaining 44% of the relationship between haematological indicators of EIMD and valence. Thus, in full agreement with recent proposals of pacing behaviour and performance regulation,[32,135,199,303] stimulus-driven heuristic processes are hypothesised to motivate the desire to walk and quit,[i] thereby necessitating the opposing recruitment of goal-driven volitional processes in continued goal-striving.

Third, runners HTW have been found to renegotiate their initially set performance aspirations and change their goals to just finishing the race.[290,291] This closely resembles an intra-psychic conflict between further goal-pursuit and goal-disengagement resulting from negatively valenced events in goal-striving – the hallmark of an action crisis.[163,164] During an action crisis, the cognitive orientation shifts from an implemental mindset facilitating volitional tasks such as goal-striving in the

[i] Critically, the desire to walk and quit must be understood as a motivational drive as the vast majority of recreational runners HTW do not actually quit.[291] Similarly, the experience of an action crisis is not a binary event inevitably leading to self-regulatory failure, but constitutes an antecedent in the goal-disengagement process and does not necessarily imply that athletes let go of their goals entirely.[175]

face of adversity to a deliberative mindset facilitating motivational tasks such as deliberating anew the desirability and feasibility of the focal and alternative goals.[160,161] More specifically, the experience of such a mindset-shift during a marathon predicted and negatively impacted running performance independently from the detrimental effects of the linearly developing physiological crisis.[164,170] In support, in the current study, a decrease in valence predicted an increase in action crisis, which in turn predicted an increase in performance fatigability, thereby suggesting heuristic and deliberative antecedents in the goal-disengagement process.

In the academic domain, using cross-lagged panel path analysis, Brandstätter and colleagues[164,289] provided convincing evidence that an action crisis temporally antecedes devaluation of goal-desirability and deterioration in perceived goal-attainability. Similarly, recent neuroeconomic research found that in a fatigued state, and moderated by endurance capacity, anticipated costs of future efforts are escalated, while the attractiveness of anticipated rewards are discounted.[304] During psychophysiological crises, stimulus-driven reactive control therefore seems to undermine goal-driven proactive control and negatively impact on thinking-action coupling in the self-regulatory control of goal-striving.[165] However, according to Brick et al.,[196] endurance athletes can monitor and control their thoughts and actions in self-regulated endurance performance through metacognitive processes, albeit at the speculated cost of accruing mental fatigue. Thus, the HTW phenomenon can be understood as psychophysiological stress process draining on fatigable and temporally limited physiological and psychological resources, eventually leading to the dissolution of the initially aspired performance goal.

Last, intriguing neurofunctional evidence for the above outlined hypothesised dual-pathway model has been provided by Maier et al.,[305] who showed that acute stress biased decision-making and self-regulatory control via two parallel but distinguishable neuroanatomical pathways: (1) endocrinological distress (indicated by salivary cortisol concentration) increased connectivity with limbic system structures suggestive of augmented salient motivating stimuli (e.g. thereby facilitating the motivational desire to slow down) and (2) perceived stress levels decreased connectivity with prefrontal cortex structures suggestive of attenuated volitional goal-maintenance (e.g. thereby debilitating the volitional drive to attain the initially set performance goal).

Confines and Suggestions

First, a limitation of the current study is the sample size. The current findings therefore prevent the generalisation of findings beyond the specific context in which this study was conducted. However, the findings do lend credibility to the hypothesised cause-effect relationships and provide researchers with valuable information to drive further theory development and research design in strain-perception-thinking-action coupling during prolonged endurance exercise.

Second, to reduce data volume and simplify the modelling process, we focussed on action crisis to indicate the shift from an implemental to a deliberative mindset. However, the preceding chapter also showed mirror-like responses in flow state.[297] Thus, although model fit indices throughout were slightly better for action crisis compared to flow state (Δ AIC = 2.755), both measures seem valid indicators of a shift from an implemental mindset cognitively tuned towards the "how" of behaviour to a deliberative mindset cognitively tuned towards the "why" of behaviour (for statistical details see supplementary material three). This provides the opportunity to also investigate facilitative effects of cognitive-evaluative processes in the regulation of pacing behaviour and endurance performance.

Third, given the significant zero-order correlation between valence and blood cortisol concentration, the fit of a theoretically equally feasible model with an additional unidirectional path from valence to blood cortisol concentration was explored. The alternative and more restrictive model provided slightly worse model fit indices (not shown). Given the observed data, the hypothesised model is more parsimonious and thus it is to be preferred based on this model comparison.

CONCLUSION

By combining causal and statistical inference, the current chapter demonstrates that dynamic and complex interdependencies in physiological and perceptual effects determine the phenomenological experience of HTW and the behavioural outcome during long-distance running with EIMD. Specifically, it contributes to a better understanding of how relationships come to be and how strain-perception-thinking-action coupling underpins observed pacing behaviour and performance fatigability. Differential responses in haematological indicators of EIMD predicted (1) amplified

physiological strain and a non-adaptive endocrinological distress response and (2) increase in perceived physical strain, which in turn mediated a decrease in valence, which in turn predicted an increase in action crisis; and both physiological and perceptual effects predicted performance fatigability. The findings are in full agreement with recent theoretical developments in centrally regulated and goal-directed exercise behaviour and provide novel insights into the psychophysiological processes that (A) shape the phenomenological experience of HTW and (B) determine the poignant and unintended alterations in pacing behaviour and performance fatigability.

CHAPTER SIX

PERCEIVED FATIGABILITY: IMPETUS, FRAMEWORK, PRELIMINARY EVIDENCE, MEASUREMENT, AND INTERPRETATION

ABSTRACT

A three-dimensional framework of centrally regulated and goal-directed exercise behaviour proposed to differentiate between the distinct inputs of sensory-discriminatory, affective-motivational, and cognitive-evaluative processes that shape perceived fatigability during prolonged endurance exercise. The suggested non-linear dynamical systems approach enables addressing pressing questions about how hypothesised cause-effect relationships come to be and how perception-action coupling underpins observed pacing behaviour and performance.

Preliminary evidence supports the hypotheses that perceived strain plays a primary role in trajectory regulation of pacing behaviour, core affect plays a primary and mediatory role in behavioural performance regulation, and the mindset-shift associated with an action crisis plays a primary role in the intensity dependent volitional self-regulatory control of conflicting motivational drives. The constructs hypothesised to underpin perceived fatigability are interdependent in a non-linear fashion with distinct temporal dynamics, context-dependent, constraint-based, distinguishable, and interrelated with dynamic changes in observed pacing behaviour, performance, and physiological disturbance.

The most appropriate scales to measure the subordinate constructs perceived physical strain, perceived mental strain, valence, arousal, action crisis, and flow state are reviewed and discussed. Non-linear dynamical systems theory is introduced as an unbiased overarching theory of governing principles in the temporal evolution of complex systems, thereby providing an opportunity to discuss the bio-psycho-social fatigue phenomenon from a holistic perspective.

The proposed framework offers a sophisticated alternative to the traditional Gestalt concept of perceived exertion and more comprehensively accounts for the psychophysiological processes that determine pacing behaviour and performance. It

has the potential to enrich theory development and gain a deeper understanding of how goal-directed exercise behaviour is regulated.

INTRODUCTION

Initial Impetus

Current psychobiological models of exercise regulation[14,30,31,36,37,306–308] almost solely rely on the Gestalt phenomenon of perceived exertion to explain something as multifaceted as centrally regulated and goal-directed exercise behaviour. The reliance on the one-item rating of perceived exertion (RPE) scale clearly is an oversimplification of this complex psychophysiological construct,[38,39,41] ignores qualitatively distinct variations of perceived exertion, and therefore must limit our fuller understanding of the dynamic processes underpinning the fatigue phenomenon, observed pacing behaviour, and the limits of human endurance performance.[42]

In his seminal work, Borg conceptualised perceived exertion as a Gestalt phenomenon and proposed it to be the single best indicator of somatic stress and physical strain integrating information from peripheral (i.e. muscles and joints) and central (i.e. cardiovascular and respiratory) sites as well as from the central nervous system (i.e. affect, motivation, and cognitive variants such as memory and attention).[43,50,80,81] Thus, affective and cognitive performance modifiers were suggested to form integral parts of the Gestalt construct. Accordingly, Borg's aim was to create a global perceptual scale and he did neither intend to differentiate between exertion, exhaustion, and fatigue nor between the mental effort *invested into / required to continue with* the task and the perception of physical strain *caused by* the task. The genuinely unintended consequence of this approach however now creates confusion about what the RPE construct actually measures and what the neural origins of perceived exertion are.[309,310]

For example, proponents of the Central Governor Model suggest that the conscious perception of exertion[90,306] and fatigue[23,25] arise secondary to mechanistic inferences of mismatches between predictive coding and error differences using a Bayesian algorithm in the allostatic control of homeostasis. In contrast, proponents of the psychobiological model based on motivation intensity theory propose a narrower definition of the mental sense of effort independent from exercise-related

interoceptions suggesting that effort sensations are directly associated with corollary discharges.[45,47,60]

However, even in its most reduced form, the *perception of* effort (in contradistinction to effort) encompasses two interrelated perceptual components associated with the production and judgement of action: (A) the energy invested into the task and (B) task difficulty.[311] Critically, this implies that the mental sense of effort simultaneously mediates two conflicting behavioural processes respectively indicating utility (i.e. reward) and disutility (i.e. cost): (A) the incentive-performance relationship, which is largely a function of (but not equal to) motivation and (B) the effort-performance relationship, which is largely a function of (but not equal to) task difficulty.[312] The sole reliance on the perception of effort therefore must limit the interpretability and fuller understanding of cost-benefit analyses in decision-making and self-regulatory control.

Particularly under the constraints of fatigue and self-restraint, behavioural regulation is affected by at least three dynamically changing interrelated factors: (A) magnitude of the urge (i.e. disengagement impulse), (B) importance of self-regulatory success (i.e. volitional drive in further goal-pursuit), and (C) resistance ability within relevant performance systems (i.e. physical and/or mental resource capacities).[313] Hence, greater eclecticism in the choice of measurement tools and underlying theoretical concepts is needed to advance theory development in perceived fatigability research.

Enoka and Duchateau[26] define human fatigue as a psychophysiological symptom underpinned by interactions between (A) performance fatigability – the decline in an objective measure of performance over a discrete period of time and (B) perceived fatigability – changes in the sensations that regulate the integrity of the performer based on the maintenance of homeostasis and the psychological state of the individual. More specifically, the fatigue-performance relationship is proposed to be modulated by homeostatic, affective, cognitive, and motor functions.

The related[67,68,314,315] but independent[316–318] construct of pain and discomfort provides the well-established Gate Control Theory (GCT),[69] which comprehensively accounts for affective and cognitive modifiers of sensory perceptions in the phenomenological experience and behavioural regulation of pain.[70] Drawing on GCT, it was suggested to progress towards a three-dimensional framework of perceived fatigability that

differentiates between the qualitatively distinct inputs of sensory-discriminatory, affective-motivational, and cognitive-evaluative processes that shape the perceptual milieu during prolonged endurance exercise.[199] Moreover, by emphasising a non-linear dynamical systems approach, it was proposed that researchers will be better equipped to address pressing questions about (A) how hypothesised cause-effect relationships come to be[219] and (B) how perception-action coupling underpins decision-making and self-regulatory control of pacing behaviour and performance.[292]

A Three-Dimensional Framework of Perceived Fatigability and Its Proposed Subordinate Constructs

In order to address these questions and to more comprehensively explain the dynamic psychophysiological regulation of observed pacing behaviour and performance during prolonged endurance exercise, chapter one[199] extensively reviewed potential subordinate constructs of perceived fatigability based on: (A) Melzack's original descriptions of the three dimensions, (B) constructs that are imbedded in sound psychophysiological theories, (C) constructs that are interrelated but can be meaningfully delineated, (D) constructs that are relevant for exercise regulation, (E) constructs that can dynamically change and be repeatedly measured during exercise, and (F) modern neuroscientific evidence.

In short, chapter one[199] concluded with the following proposals and hypotheses: (A) perceived mental strain and perceived physical strain are primary regulators of trajectory pacing behaviour reflecting sensory-discriminatory processes necessary to align planned behaviour with current physiological state; (B) core affect plays a primary and mediatory role in the behavioural regulation of performance, and its underlying two dimensions valence and arousal reflect affective-motivational processes in the heuristic control of approach and avoidance behaviour; and (C) the mindset-shift associated with an action crisis plays a primary role in the intensity dependent volitional self-regulatory control of conflicting motivational drives, thereby reflecting cognitive-evaluative processes between further goal-pursuit and goal-disengagement.

Preliminary Supportive Evidence

Initial research aimed to capture, describe, and model the complex interdependencies and temporal dynamics in these processes as well as their interrelationships with

observed pacing behaviour, performance, and endocrinological distress response. More specifically, in part one of chapter two,[226] inter-individual differences in these dynamic processes were investigated during a maximal self-paced 70-km individual time trial ($INDV_{TT}$) over a simulated profiled course in 23 cyclists of distinct performance level (PL) categories (11 PL4 and 12 PL3). The main findings were:

- Perceived physical strain showed performance level dependent differences associated with observed pacing behaviour and was strongly correlated with endocrinological distress response, thereby pointing towards the accuracy of interoceptive feedback in the trajectory regulation of pace. However, perceived physical strain was not significantly different from perceived mental strain supporting the hypothesis that both contribute to the central regulation of pacing behaviour by attempting to minimise error differences between imperfect anticipatory feedforward[135] and time-lagged afferent feedback[48] control.
- PL3 cyclists showed beginning deterioration in core affect and its underlying two dimensions valence and arousal towards the finish suggesting that greater aerobic capacity facilitates better maintenance of arousal and valence. In contrast, PL4 cyclists showed a facilitative endocrinological stress response and greater tolerance of metabolic stress suggesting that positive affect influences the degree of physiological disruption that can be tolerated.[217] Thus, the observed performance level dependent differences in valence and arousal support the hypothesis of a primary role of core affect in the regulation of performance.

In part two of chapter two,[226] inter- and intra-individual differences in these processes were investigated under the psychologically stressful constraint of a head-to-head competition against a performance matched opponent over the same 70-km virtual course and in comparison to cyclists' $INDV_{TT}$ performance. Overall 14 head-to-head competitions were conducted. The main findings were:

- Despite substantial competition outcome dependent differences in performance and subsequent physiological demands, no differences were observed in perceived physical strain or perceived mental strain. Thus, the strain-performance relationship can be distorted,[319] thereby indirectly pointing towards

the crucial role of affective and cognitive performance modifiers within a given bio-psycho-social context.[14,23]

- Falling-behind was associated with deterioration in valence as well as the development of an action crisis, which by definition results from negatively valenced events related to setback and failures in goal-striving.[163] This supports the hypothesis of a mediatory role of valence in the regulation of performance by initiating a shift in attentional focus from deliberate goal-driven to heuristic stimulus-driven processes.[220]
- The mindset-shift associated with an action crisis represents a decisional conflict between further goal-pursuit and goal-disengagement[164] and was accompanied by an exacerbated endocrinological distress response despite relative performance decrement compared to losers' $INDV_{TT}$ performance. It thereby supports the hypothesis that volitional self-regulatory control of conflicting motivational drives is a non-adaptive stressful event in goal-striving and, by draining on limited psychological and physiological resources, not conducive to high performance.

In chapter three,[224] a five-step structural equation modelling (SEM) procedure was applied to conduct formal tests of the direction and temporal precedence of hypothesised cause–effect relationships by testing the integrity of the hypothesised model structure. SEM combines causal (i.e. formalising and implementing testable causal hypotheses in an experimental design) and statistical (i.e. assessing the extent to which the experimental data fit the a priori specifications) inference based on a simultaneous analysis of the entire system of variables. It thus provides a higher-level perspective critical for theory development. The main hypotheses / findings were:

- In a theory-driven approach the following cause-effect relationships were hypothesised: (A) falling behind *antecedes* deterioration in core affect, (B) deterioration in core affect *antecedes* development of an action crisis, (C) action crisis *antecedes* non-adaptive endocrinological distress response, and (D) non-adaptive distress response *antecedes* performance decrement.
- Deterioration in valence was a significant mediator by explaining 35% of the relationship between falling behind and action crisis and thereby indicates how the relationship between falling behind and action crisis comes to be. It

therefore provides empirical evidence linking heuristic and deliberative decision-making modes as recently theorised by several researchers.[32,135,196,199]

- The mindset-shift associated with an action crisis predicted increased blood cortisol concentrations, which in turn predicted performance decrement relative to cyclists' performance capabilities. It thereby links perception-thinking coupling to action and provides empirical evidence for metacognitive processes in the self-regulatory control of pacing behaviour and endurance performance.[196]

In chapter four,[297] intra-individual differences in the psychophysiological regulation of pacing behaviour and performance were investigated under the physiologically stressful constraint of running with locomotor muscle fatigue (LMMF) and exercise-induced muscle damage (EIMD). Twenty-two (11 female) highly-trained (PL4) runners completed two maximal self-paced 20-km treadmill time trials in a counterbalanced crossover design: (A) in a tapered condition and (B) with LMMF and EIMD produced by a standardised drop-jump protocol. The main findings were:

- LMMF and EIMD caused large reductions in power output generating capacity of the knee extensors; small increases in heart rate, oxygen consumption, and carbohydrate usage as well as medium increases in energy cost of running; and medium increases in blood leucocyte and neutrophil counts, interleukin-6, and cortisol concentrations. Collectively, the increase in relative exercise intensity and subsequent amplified physiological demand provoked a non-adaptive endocrinological distress response that was associated with compromised endurance performance capacity.
- Moreover, LMMF and EIMD caused large exacerbations in perceived fatigability as indicated by large increases in perceived physical strain, large decreases in valence, and large mirror-like increases and decreases in action crisis and flow state, respectively. These perceptual cues too were associated with increased performance fatigability. It thus supports the hypothesis that perceived physical strain mediates the relationship between amplified physical strain and deterioration in valence, thereby constituting a psychobiological link initiating a

shift from volitional goal-driven to heuristic stimulus-driven processes in decision-making and self-regulatory control.

In chapter five,[293] SEM was applied to model the extent to which non-adaptive endocrinological distress response and strain-perception-thinking-action coupling accounted for the observed temporal dynamics in performance fatigability and the phenomenological experience of 'hitting the wall' (HTW) during long-distance running with EIMD. The main hypotheses / findings were:

- In a theory-driven approach, the following simplified[j] dual-pathway model was hypothesised: Running with EIMD impacts performance fatigability by (1) amplified physiological demands and non-adaptive endocrinological distress response and (2) deterioration in perceived fatigability: an increase in perceived physical strain *antecedes* a decrease in valence, which in turn *antecedes* an increase in action crisis; eventually dissolving the initially aspired goal.
- Differential response in haematological indicators of EIMD predicted (A) non-adaptive endocrinological distress response, which in turn predicted increased performance fatigability and (B) perceived physical strain was a significant mediator by explaining 44% of the relationship between haematological indicators of EIMD and deterioration in valence; which in turn predicted a mindset-shift indicated by mirror-like alterations in action crisis and flow state; which in turn predicted increased performance fatigability.
- Thus, the HTW phenomenon can be understood as psychophysiological stress process draining on fatigable and temporally limited physiological and psychological resources. It thereby supports the hypothesis that under the constraint of amplified psychophysiological duress stimulus-driven heuristic processes oppose and undermine goal-driven volitional processes; eventually leading to the dissolution of the initially aspired performance goal.

In conclusion, the proposed constructs are interdependent in a non-linear fashion with distinct temporal dynamics, context-dependent, constraint-based, distinguishable by

[j] Due to the limited sample size, a simplified version was tested that focused on the haematological indicators of EIMD and thus the muscle damage component rather than the combined effects of LMMF and EIM.

well-trained athletes, and interrelated with dynamic changes in observed pacing behaviour, performance, and physiological disturbance. Particularly, when combined with causal and statistical inference, the proposed three-dimensional framework of perceived fatigability provides a more holistic and comprehensive understanding of (A) how relationships come to be and (B) how strain-perception-thinking-action coupling underpins decision-making and self-regulation in centrally regulated and goal-directed exercise behaviour.

On the Measurement of Perceived Fatigability and Its Subordinate Constructs

The Sensory-Discriminatory Dimension

Various researchers recently emphasised the need to differentiate between subjective task difficulty and mental effort *invested into / required to continue with* the task at hand (i.e. perceived mental strain) and other exercise-related sensations, such as muscular, respiratory, and thermal strain *caused by* the task (i.e. perceived physical strain).[45,48,64,199,215]

Perceived Physical Strain

As mentioned previously, Borg conceptualised perceived exertion as the single best indicator of physical strain and originally constructed the 15-point (6 to 20) Borg scale to increase linearly with exercise intensity, heart rate, and oxygen consumption.[320] The ratings of perceived exertion are therefore easy to compare with objective measures of physiological strain during self-paced and time to exhaustion exercise. The equidistant interval scale further allows for extrapolations to be made, and these scalar properties of perceived exertion[321,322] are now a widely accepted key characteristic in the anticipatory regulation of pacing behaviour.

In contrast, the CR-10 and the finer grained CR-100 scales were constructed as category-ratio scales with a non-linear positively accelerating growth function as necessary to estimate the phenomenological experience of pain.[320] CR-10 ratings therefore improve scaling of the most extreme intensities of pain and related subjective somatic symptoms during incremental stress testing to voluntary task failure. However, exercise intensities and subsequent physiological demands in endurance events are by necessity always submaximal and the traditional 15-point (6 to 20) Borg scale is

therefore suggested to be the most appropriate scale to estimate perceived physical strain during prolonged endurance exercise.

Critically, the sensory-discriminatory dimension represents perceptual information on location, quality, and intensity.[70] The scale instructions should therefore not refer to pain and discomfort, which imply negative valence. Instead the question should be phrased in terms of intensity, which is affectively neutral. “How strong are the physical sensations from your legs, lungs, and body?” – using the anchors ‘light’ and ‘strong’. Depending on the experimental environment and research question at hand specifications can be made to rate perceived muscular, respiratory, or thermal strain.

Perceived Mental Strain

In the RPE scale instructions, Borg described perceived exertion as the feeling of how heavy and strenuous a physical task is.[320] Pageaux therefore recently proposed that the combined conscious sensations of perceived task difficulty and energy exerted suffice to operationalise the mental sense of effort, and that the addition of other confounding exercise-related sensation needs to be avoided.[45] This perceived mental strain has been shown to be sensitive to psychological and physiological manipulations, and thus too can be appropriately estimated with the 15-point Borg scale. However, the scale’s range should be modified (e.g. 0 to 14) using the anchors ‘easy’ and ‘hard’, thereby providing contrast and differentiating it from the traditional heart rate inspired (6 to 20) Borg scale used to estimate perceived physical strain.

An alternative scale assessing subjective task effort and awareness (TEA) independently from physical sensations of exercise has been developed by Swart et al.[57] Although research studies utilising the TEA scale provide reliable findings coherent with studies utilising the RPE scale phrasing “How hard are your trying?”, there are inconsistencies in its theoretical underpinnings and psychometric properties.[199] It has furthermore not yet been formally validated. In contrast, the well-established Borg scales have been shown to be valid and reliable,[320] and are therefore suggested to be the most appropriate scales to estimate perceived mental strain during prolonged endurance exercise.

As mentioned previously, the *perception of* effort encompasses two interrelated perceptual components associated with the production and judgement of action:

(A) energy invested into the task and (B) task difficulty.[311] However, according to Wright[313] and Brehm's Motivation Intensity Theory, the mental sense of effort is primarily determined and modulated by task difficulty, not energy mobilisation. Thus, the scale instruction should be phrased "How difficult is it to swim / cycle / run at this pace?", rather than "How hard are you trying or driving your locomotor muscles?"

Last, special emphasis should be placed on instructions and administration by including the following: standardised written instructions, explanations of differences between the constructs (e.g. using illustrations provided by Smirmaul[48]), anchoring of extreme ratings during incremental exercise testing to exhaustion, encouraging the use of decimals, and familiarisation with data collection during a baseline trial simulating experimental trial conditions.

The Affective-Motivational Dimension

A three-tiered justification process for measurement selection in affect research has been recommended by Ekkekakis.[144,145] First, the construct of core affect is suggested as it encompasses the global domain of affective states in a more holistic fashion than mood and emotion, and because distinct affective states during maximal self-paced prolonged endurance exercise cannot be predicted. Second, from the available conceptual models of core affect, the circumplex model of affect is suggested, as it allows the independent assessment of the two orthogonal and bipolar dimensions valence and arousal.[206] Third, the independence and bipolarity of the two dimensions have been psychometrically tested and confirmed.[207]

In addition, single-item dimensional measures have the advantage that they can be applied repeatedly over time, whilst interfering as little as possible with the participant's focal state. They are therefore preferred in the measurement of temporal dynamics in affective states during prolonged endurance exercise. In contrast, multi-item dimensional measures of affect are less prone to random measurement error, but only suitable to assess pre-to-post trial changes and chronic effects of exercise interventions.

Valence

The single-item dimensional Feeling Scale (FS) was constructed and validated as a bipolar 11-point (-5 to +5) scale measuring the range of core affective responses from pleasure to displeasure during exercise.[38] Despite its widespread use and easy to understand scale format, the verbal anchors and spacing between the anchors were derived non-empirically. More recently, Lishner et al.[323] addressed this issue by developing the Empirical Valence Scale (EVS), which may provide more sensitive quantitative and qualitative information. The EVS should therefore be considered a valuable alternative to the non-empirical FS in future research.

The 20-item dimensional Positive Affect (later redefined Positive Activation) Negative Affect (later redefined Negative Activation) Schedule (PANAS) is one of the most widely used measures of affect.[324] However, PANAS contains a mixture of emotion, mood, and core affect statements inconsistent with recent conceptual developments in affect research. Another issue pertains the sole reliance on high arousal states.[325] It therefore prevents the measurement and interpretation of low arousal states such as fatigue, which are undoubtedly of great interest in exercise behavioural research.

Given the aim to capture, describe, and model the temporal dynamics and interrelationships of valence, the above described single-item dimensional measures are suggested to be most appropriate to estimate valence during prolonged endurance exercise.

Arousal

The single-item dimensional 6-point (1 to 6) Felt Arousal Scale (FAS) was originally developed as part of telic (i.e. arousal avoiding) and paratelic (i.e. arousal seeking) meta-motivational states in reversal theory, and contrasted felt arousal with preferred arousal in goal-directed behaviour.[208] However, a common challenge with the FAS is to convey to participants that arousal is a cognitive, behavioural, *and* physiological construct.[326] Thus, additional terms such as ‘felt activation’ and ‘engagement’ should be used to clarify the arousal concept and instruct participants.

Other limitations pertain the limited range of possible responses necessitating the encouragement to use decimals and the overly succinct instructions of the FAS.

Ekkekakis[145] therefore suggested to utilise the more detailed instructions of the Affect Grid (AG),[327] which is a 9 by 9 grid with valence and perceived activation respectively representing the horizontal and vertical axes as well as additional anchors at the four corners. Although the AG was designed for repeated measurements, separate single-item scales are arguably easier to understand and to administer practically during prolonged endurance exercise.

Like the PANAS, the 20-item dimensional Activation Deactivation Adjective Check List (AD-ACL) provides a 45° rotational variant of the circumplex model of affect constructed around the two bipolar dimensions of energetic arousal and tense arousal.[328] Although, the AD-ACL includes low activation states and therefore more completely accounts for the circumplex space than the PANAS,[329] it still leaves some sectors uncovered.[325,330] An improved factor structure has been shown for the conceptually similar state version of the 20-item Four Dimension Mood Scale (4-DMS) comprising positive energy, tiredness, negative arousal, and relaxation.[331] Moreover, the 4-DMS's tiredness scale more directly refers to fatigue rather than sleepiness, which may make it more sensitive to physical exercise effects.[332]

Still, given that special attention is paid to careful instruction of participants, the single-item dimensional FAS is suggested to be most appropriate to estimate the temporal dynamics in perceived activation during prolonged endurance exercise.

The Cognitive-Evaluative Dimension

Fatigue-induced alterations in observed pacing behaviour and performance during prolonged endurance exercise are preceded by self-regulatory and decision-making processes between goal-oriented volitional drives urging further goal-pursuit and stimulus-oriented motivational drives urging goal-disengagement.[195] The measurement of this dynamic and interactive process therefore requires the differentiation between the volitional task of goal-striving and the motivational task of goal-setting as well as their distinct underlying mindsets (i.e. implemental *versus* deliberative) optimal for the specific task at hand.[199]

Action Crisis

The Action Crisis Scale (ACRISS) was developed to measure the extent to which participants experience a decisional conflict between further goal-pursuit and goal-disengagement resulting from negatively valenced events in goal-striving.[163] The ACRISS comprises six items: conflict, setbacks, implemental disorientation, rumination, disengagement impulses, and procrastination rated on a 5-point Likert type scale ranging from 1 ‘no agreement’ to 5 ‘very much agreement’. It has shown good internal consistency, reliability, and concurrent validity with increased cost-benefit thinking, implemental to deliberative mindset-shift, and temporally dynamic goal-disengagement processes.[163,164,289]

A related concept is task-specific self-efficacy (defined as the individual’s perceived capability to accomplish a certain performance level), a core construct in social cognitive theory (SCT),[333] which influences what actions are pursued, how much effort is invested in goal-pursuit, and what level of persistence is demonstrated in the face of setbacks and difficulties.[334] Even though self-efficacy is considered the active agent in SCT, it should nevertheless be measured in conjunction with other core determinants of SCT such as outcome expectations and goals, which are required to determine the perceived costs and benefits of goal-striving. When combined with the need to provide hierarchical measurements of task-specific self-efficacy,[335] this construct seems to be more suited for the assessment of pre-to-post trial changes and interventions over longer time frames.

Given the dedicated development of ACRISS to measure the underlying mindset-shift of antecedents in the goal-disengagement process, the ACRISS is suggested to be most appropriate to estimate the temporal dynamics of debilitative cognitive-evaluative processes in goal-striving during prolonged endurance exercise.

Flow State

The short Flow State Scale (FSS) was developed to measure the state level and thus the extent of flow – or optimal experience - during an event or activity.[225] The short FSS comprises nine items: challenge-skill balance, action-awareness merging, clear goals, unambiguous feedback, concentration on task at hand, sense of control, transformation of time, and autotelic experience rated on a 5-point Likert type scale

ranging from 1 ‘no agreement’ to 5 ‘very much agreement’. It has been validated through confirmatory factor analysis and has shown good psychometric properties.[336] Critically, defining characteristics of flow such as total focus, involvement, and absorption are all facilitated by an underlying implemental mindset during action phases.[160]

A related concept to flow is psychological momentum (PM), which refers to the individual’s perception of goal-progress. The experience of PM itself is influenced by situational and personal factors and hypothesised to have facilitative effects on performance, which are moderated by contextual and personal factors.[337] However, scientific evidence for the elusive momentum-performance relationship is scarce and positive inhibition and negative facilitation are believed to form vital components.[338] Using the five-item Perception of Psychological Momentum Scale during a bogus bicycle competition, Perreault et al. showed that both negative (i.e. falling-behind) and positive (i.e. coming from behind) momentum correlated with improved performance.[339] Thus, the systematic understanding of psychological momentum is limited at this stage and may benefit from qualitative research to inform clear conceptualisation and quantification.[340]

In contrast, the scientifically sound development of the FSS was based on Csikszentmihalyi’s established (albeit recently critically reviewed by Swann et al.[362]) theory of flow as applied to the sport setting[278] and is thus suggested to be most appropriate to estimate the temporal dynamics of facilitative cognitive-evaluative processes in goal-striving during prolonged endurance exercise.

However, the ACRISS and the short-FSS comprise six and nine items, respectively, which necessitates the retrospective assessment of these scales during the recovery period. This carries the limitation of potential recall bias. Thus, opportunities must be taken to enhance recall, for example by using simulated profiled courses instead of overly simple laboratory protocols.

A summary of the proposed three-dimensional framework assessing the dynamic processes underlying perceived fatigability during prolonged endurance exercise, its proposed underlying constructs, and their suggested measurement is provided in figure 19.

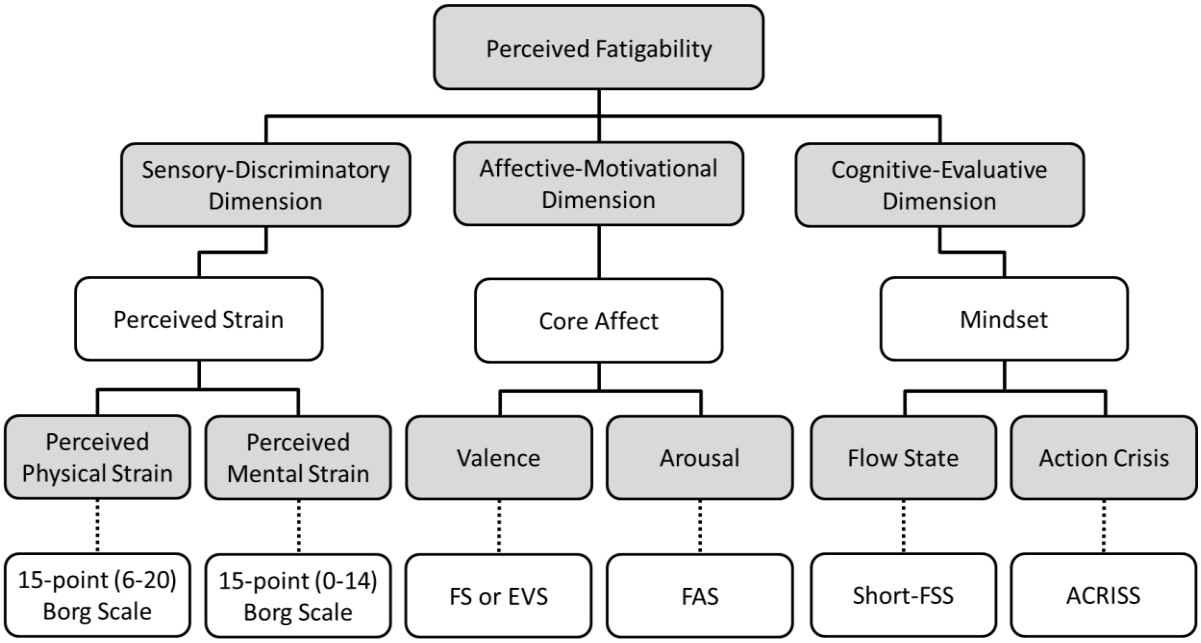

Figure 19: A summary of the proposed three-dimensional framework assessing the dynamic processes underlying perceived fatigability during prolonged endurance exercise, its proposed underlying constructs, and their suggested measurements.

Unidimensional versus Multidimensional Approaches

The first main objective of the current line of research was to progress towards a three-dimensional framework of centrally regulated and goal-directed exercise behaviour, thereby providing a more sophisticated alternative to the Gestalt concept of perceived exertion. The initial research studies utilising this three-dimensional approach provided preliminary support for its utility and a more comprehensive understanding of the psychophysiological mechanisms that underpin the observed dynamics in pacing behaviour and performance during prolonged endurance exercise. It is further suggested that the hypothesised interdependencies also underpin short-term exercise. However, while some researchers have pushed the frequency of scale application,[341,342] it is currently recommended to explore the multidimensionality of exercise behaviour during longer time frames due to the necessity to administer multiple perceptual scales during exercise. Thus, the Gestalt concept of perceived exertion[320] and unidimensional constructs of fatigue[343] remain valid and important tools in exploratory analyses and during short-term exercise, where repetition bias and disruption of the athlete's natural focal state prevent the meaningful application of more comprehensive multidimensional approaches.

Dynamic Processes versus Long-Term Adaptions

The second main objective of the current line of research was to focus on the dynamic processes within each dimension as well as on the dynamic interplay between the dimensions and their underlying constructs during prolonged endurance exercise. The initial research studies emphasising the temporal dynamics of sensory, affective, and cognitive perceptions provided preliminary support for its utility and a more comprehensive understanding of the temporal precedence of events, thereby informing how hypothesised cause-effect relationships come to be. Thus, the proposed framework more completely accounts for non-linear and dynamic interactions that underpin the observed alterations in pacing behaviour and performance. However, the three-dimensional framework is also suggested to inform theory development in adaptational processes of general exercise behaviour over longer time frames. For example, the affective construct of mood and the cognitive construct of task-specific self-efficacy remain valid and important tools in longitudinal research studies such as on the effects of exercise on depression or chronic adaptations of self-efficacy in response to healthy-weight training interventions. Measuring these more stable constructs pre-to-post over longer time frames will allow the application of cross-lagged panel path analyses and thus assessment of direction and temporal precedence in hypothesised cause-effect relationships.

Interpretation

To make sense of research findings, observed data need to be interpreted in relation to a theoretical framework. However, progressive specialisation and multidisciplinary approaches in the exercise sciences have led to fragmentation as well as reductionism and dichotomies in models and methodologies.[188] Accordingly, there is a plethora of scientific disciplines, theories, and variables that need to be integrated to inform theory-development in interdisciplinary phenomena such as exercise-related fatigue (e.g. biomechanics, biochemistry, physiology, psychology, and sociology). This conflicts with the implicit goal of all sciences to synthesise the maximum number of phenomena with a minimum number of general principles.[189] Thus, “opportunities must be taken to discuss fatigue from a holistic perspective so that we can integrate different components of fatigue into a modern understanding” (p.66).[186]

Non-Linear Dynamical Systems Theory

Non-linear dynamical systems theory (NDST) is a set of interconnected general principles and concepts and therefore provides an opportunity to discuss the bio-psycho-social fatigue phenomenon from a level- and model-independent perspective.[189,190] NDST acknowledges the context-dependent, dynamic, non-linear, and circular bottom-up (i.e. feedback) and top-down (i.e. feedforward) effects between molecular, organismic, and social levels.[189] It has therefore been suggested to be particularly suited to investigate the psychophysiological regulation of exercise behaviour and performance under changing constraints imposed by fatigue.[191] The chief task of the NDST approach is to capture, describe, and model the complexity of dynamic processes in observed systems, rather than reducing it. It therefore provides an unbiased underpinning theory in the understanding of governing principles in the temporal evolution of systems, thereby allowing for theoretical pluralism.[189]

Exercise scientists and clinicians readily agree that fatigue is a multifactorial, interactive, and complex phenomenon.[187] However, mechanistic and reductionistic approaches and methodologies continue to prevail, despite exercising humans naturally embodying dynamical systems. The interactions of elements within a dynamical system are too complex to be characterised by cause-effect relationships between isolated components.[344,345] Instead, the dynamic systems approach postulates that observed behaviour emerges or self-organises from the complexity of interactions, dependencies, and relationships between the elements that form the system.[346] Other typical properties of dynamical systems are: non-stationarity (i.e. phase changes over time), history-dependence (i.e. adaptation), and non-linearity.[344] Thus, NDST can easily explain why, depending on the current state of the system, perturbations may cause an exponential effect, a proportional effect, or no effect at all; something hard to reconcile with current psychobiological models of exercise regulation[14,30,31,36,37,306–308] without reverting to *ad-hoc* explanations.

Accordingly, following successful application in other research fields such as biology and sociology, exercise scientists have begun utilising NDST to understand the general principles that underpin sport-related phenomena such as coordination,[346] attention focus,[192,198] decision-making,[347] fatigue-induced goal-disengagement,[197,348,349] perceived exertion,[350,351] and athlete-environment

interactions.[352] NDST furthermore lends itself to investigate collective behaviour in team sports and provides a promising avenue to understand heuristic (i.e. herd-principle) and rational tactical behaviours in mass-start competitions.[353,354]

More specifically, another two typical properties of NDST are discussed below along with exemplary findings of the present work, which are essential for a better understanding of centrally regulated and goal-directed exercise behaviour: (1) attractors and (2) meta-stability.

Drawing from Vallacher et al.[355] and Balague et al.,[192] key findings of the present work can be interpreted using the gravity well metaphor, which underpins complex and dynamical systems theory by emphasising the spontaneous emergence of stable behaviours from the complex and dynamic interdependencies between its constituents and attractors. “An attractor refers to a subset of potential states or patterns of change to which a system’s behaviour converges over time” (p.264).[355] It therefore represents a state of energy minimum resistant to perturbing influences consistent with the notion of homeostasis. Various factors can disturb the state of the system, but eventually it will spontaneously stabilise on a state that minimises incompatibilities and error differences.

The gravity well metaphor, attractor concept, and phase shifts between stable and metastable (i.e. spontaneous alternation between possible states) states are depicted in figure 20. The balls represent possible states of the system and the valleys represent the attractors of the system. The attractors are characterised by the width and depth of the gravity well. The wider the gravity well the wider the variety of elements that are attracted. The deeper the gravity well the harder it is to overcome the attractor. The system is in permanent fluctuation (indicated by the black arrows), but ultimately aims to attain stable states or energy minima (indicated by the black balls). Positive and negative attractors direct behaviours towards the attainment of the focal goal, while resisting other possibilities, thereby determining the probability of attaining a specific observed behaviour. Alternative states are possible (indicated by the grey balls) but are likely to change to more stable states over time due to weaker attractors or to alternate between equally feasible states (indicated by the grey arrows).

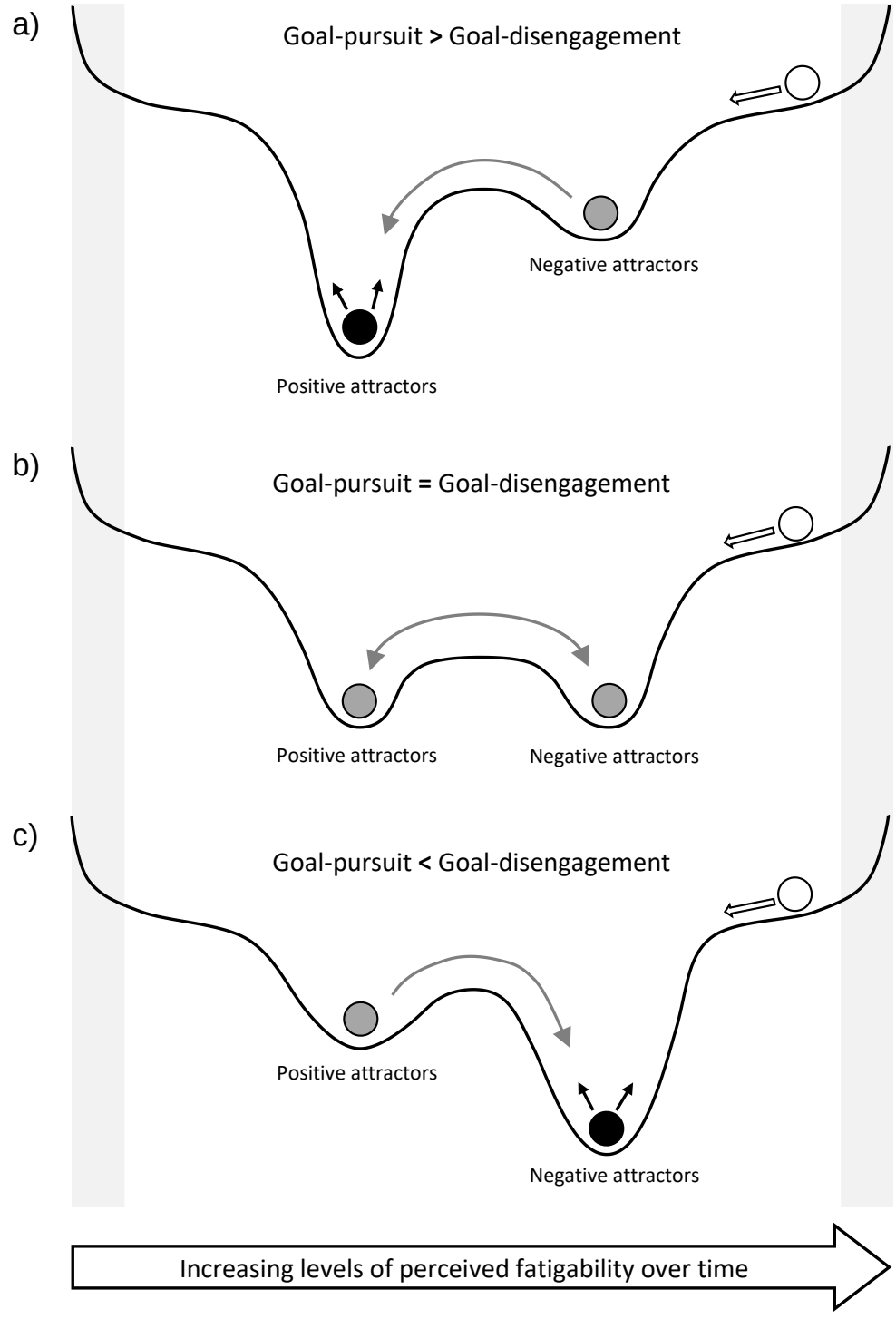

Figure 20: The gravity well metaphor, attractor concept, and phase shifts between stable and metastable (i.e. spontaneous alternation between possible states) thought states under the constraint of increasing levels of perceived fatigability. Dynamic changes in the influence of positive and negative attractors on implemental and deliberative mindsets underlying thoughts about further goal-pursuit and goal-disengagement. See text for description. Adapted from Vallacher et al.[355] and Balague et al.[192]

Overcoming and resisting the preferred state and approaching psychophysiological limits is temporarily possible (indicated by the white balls), but ultimately futile as perpetual energy investment and volitional drive are required (indicated by the white arrows). Last, the range of possible states of a system is bounded by extreme unstable equilibria, which are referred to as repellors (indicated by the grey bars).

The preliminary supportive findings of the present work are fully compatible with the above descriptions. The goal of winning or achieving a personal best performance steers goal-directed behaviour towards energising goal-pursuit and thereby functions as a positive attractor. In the early stages of a race, perceived fatigability is still mild and negatively valenced attachments to perceived strain urging to disengage from the aspired goal emerge only intermittently and are easy to overcome (see figure 20 a)). Thus, a stable implemental mindset persists, thereby facilitating goal-striving and further goal-pursuit.

Over time, perceived fatigability increases, and this process can be intensified by situational (e.g. falling behind a performance matched opponent) or stimulus-driven (e.g. running with exercise-induced muscle damage and amplified physiological strain) factors. As shown in chapters two to five, the experiences of psychological and physiological distress act as repellors and their common denominator negative valence increasingly attracts thoughts about partial goal-disengagement (e.g. slowing down), thereby opposing the initially set performance goal. This suggests that the dynamic change in core affect acts as a negative attractor and the driving force by which the attentional focus and cognitive mindset is shifted. During an action crisis, athletes are caught in a decisional conflict between further goal-pursuit and goal-disengagement. As positive and negative attractors are equally strong, no preferred state exists and the barrier between states is reduced (see figure 20 b)). This leads to spontaneous alternation between implemental and deliberative mindsets and subsequent behavioural fluctuations between further goal-pursuit and goal-disengagement (i.e. cognitive metastability).

The combined continued rise of psychophysiological strain and drainage of psychophysiological resources amplifies the strength of the negative attractors over time. This will have to be compensated for with ever increasing volitional drive, which itself is fatigable and therefore temporally limited. An action crisis furthermore evokes

devaluation of goal-desirability and deterioration in perceived goal-attainability by deliberating anew the desirability and feasibility of the pursued and alternative goals, thereby simultaneously undermining the strength of positive attractors and further goal-pursuit (see figure 20 c)). Thus, stable deliberative thought states, and ultimately a new implemental mindset, about partial goal-disengagement (i.e. settling for a lesser alternative goal) will emerge, thereby dissolving the initially aspired performance goal and determining the changes in observed pacing behaviour and performance.

LIMITATIONS AND FUTURE DIRECTIONS

The present work emphasised the complex and dynamic processes that underpin observed pacing behaviour and performance during prolonged endurance exercise. Specifically, the proposed three-dimensional framework differentiates between the distinct inputs of sensory-discriminatory, affective-motivational, and cognitive-evaluative processes that shape perceived fatigability. The utility of this holistic approach lies in its ability to more completely account for dynamic alterations on perceived fatigability and performance fatigability; and ultimately observed exercise behaviour. However, it is also noted that, while the proposed three-dimensional framework may assist researchers to gain a deeper understanding of the underlying psychophysiological interactions and mechanisms, perceived fatigability must continue to be understood as an organised whole that is more than the sum of its parts. The proposed framework therefore remains an approximation of the Gestalt phenomenon that is perceived fatigability.

In addition, investigations focussed on the dynamic changes under the constraints of psychological and physical duress, thereby concentrating on the goal-disengagement component in goal-striving. A limitation of this approach was that facilitative processes underpinning goal-pursuit and performance enhancement (i.e. the ‘winning factors’) can only be indirectly inferred. However, the mirror-like responses in action crisis and flow state observed in chapters four and five suggest that opposing effects of mindsets underpin self-regulatory control and decision-making in exercise behaviour. Future research should therefore test the veracity of this prediction by testing the temporal dynamics in implemental and deliberative mindsets under conditions of experimentally induced positive momentum.

Another limitation was the focus on mediatory processes between the psychophysiological determinants of exercise behaviour, and thus on questions of 'how' and 'why' hypothesised cause-effect relationships come to be. Thus, the effect of moderating factors (other than performance level and sex) were not yet included, thereby limiting the ability to predict for 'whom' and 'when' the observed interdependencies apply. Future research should therefore investigate the role of dispositional moderators such as action versus state orientation,[218,356] which have been shown to respectively protect from the development of an action crisis or aggravate the perception of the same.[174] This has the potential to implement screening procedures for individuals that are most likely to benefit from psychological skills training.

From a practical application point of view, mental contrasting and implementation intentions have been shown to successfully ease the consequences of a deliberative mindset during an action crisis in a favourable manner.[357,358] Thus, volitional self-regulatory abilities can be improved through training interventions and their effects on performance can be tested. Future research studies should therefore assess the extent to which such interventions can affect and improve self-regulation under constraints of psychophysiological duress as experienced during the decisive final stages of endurance competitions.

A psychometrical limitation pertains the measurement of action crisis and flow state with multi-item questionnaires. This necessitates the retrospective assessment of cognitive-evaluative processes during the recovery period, thereby carrying the limitation of potential recall bias. Thus, opportunities must be taken to enhance recall, for example by using simulated profiled courses instead of overly simple laboratory protocols. However, currently there are no measures available to assess 'online' the shift from an implemental to a deliberative mindset during graded exercise to voluntary exhaustion and time to exhaustion trials to task failure. Thus, the development of single-item action crisis and flow state scales is required to approximate the temporal dynamics and critical boundary patterns in cognitive-evaluative processes during such protocols.

In extension of the above, the current line of research focussed on the dynamic processes within and between the subordinate constructs hypothesised to underpin

perceived fatigability and self-paced exercise behaviour. However, these psychophysiological variables themselves are defended regulatory parameters, thereby preventing the assessment subtle differences in response to imposed constraints. Time to exhaustion tests therefore continue to be valid and important research tools in the investigation of strain-perception-thinking-action coupling that precedes task failure. Thus, ultimately closed- and open-loop exercise trials are required to gain a comprehensive understanding of centrally regulated and goal-directed exercise behaviour.[359]

Lastly, a statistical limitation pertains the limited sample size available in chapters three and five, which prevents the generalisation of SEM findings beyond the specific context in which these studies were conducted. An attempt was made to address this limitation by applying a strict five-step modelling procedure to ensure that the current data were robust enough for the structural modelling process. Thus, the findings do lend credibility to the hypothesised cause-effect relationships. Nevertheless, future studies are warranted to replicate the current research findings with larger sample sizes and under different internal and external constraints.

CONCLUSIONS

The proposed three-dimensional framework of perceived fatigability emphasises the complex and dynamic interplay between the sensory, affective, and cognitive processes that shape the perceptual milieu during prolonged endurance exercise and underpin the regulation of goal-directed exercise behaviour. It thereby provides a more sophisticated alternative to the traditional Gestalt concept of perceived exertion. Particularly when combined with a non-linear dynamical systems approach, it more comprehensively accounts for the psychophysiological processes that determine the observed alterations in pacing behaviour and performance under changing constraints of psychophysiological duress. Moreover, it can deal with non-linearity and explain the spontaneous emergence of deliberative thoughts and behaviours from underpinning homeostatic and heuristic processes and interdependencies. The utility of the proposed approach has been tested under psychological and physiological constraints and was confirmed with causal hypothesis testing. Based on the findings of the present thesis, the principled three-dimensional framework offers a holistic perspective in fatigue research by integrating different components into a modern understanding,

thereby improving the ability to address pressing questions about how hypothesised cause-effect relationships come to be and how strain-perception-thinking-action coupling underpins perceived fatigability. Thus, it has the potential to enrich theory development in centrally regulated and goal-directed exercise behaviour by providing a practical, theoretically sound, and unbiased outline in the investigation of the psychophysiological determinants of pacing behaviour and performance during prolonged endurance exercise.

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APPENDIX

SUPPLEMENTARY MATERIAL ONE (CHAPTER TWO)

Three-Way (time × trial × competition outcome) RM ANOVA

Performance Data

There were no significant time × trial × group interaction effects for absolute power output, power to weight ratio, or power to VT-1 ratio, but significant time × trial interaction effects with highly significantly greater values at the start of the $COMP_{TT}$ for absolute power ($F_{3.3\ 86.6}$=10.7; p<0.001; η_p^2=0.289), power to weight ratio ($F_{4.0,\ 102.7}$=8.3; p<0.001; η_p^2=0.242), and power to VT-1 ratio ($F_{3.4,\ 89.5}$=10.4; p<0.001; η_p^2=0.286). The three variables further showed significant trial × group interaction effects of ($F_{1,\ 26}$=16.7; p<0.001; η_p^2=0.391), ($F_{1\ 26}$=8.2; p=0.008; η_p^2=0.240), and ($F_{1,\ 26}$=15.4; p=0.001; η_p^2=0.372), respectively.

Three-way ANOVA for split time revealed a significant time × trial × group interaction effect ($F_{2.8,\ 72.6}$=3.5; p=0.022; η_p^2=0.119) with losers only having significant simple two-way time x trial interaction effect ($F_{2.6,\ 33.8}$=6.6; p=0.002; η_p^2=0.388). Simple, simple (main) group effects in losers found significantly faster split time over the first 7 km ($F_{2.6,\ 33.8}$=7.7; p=0.016; η_p^2=0.371) and significantly slower split times for the second climb and the final 7 km ($F_{2.6,\ 33.8}$>10.3; p<0.007; η_p^2>0.442).

There were no differential responses in cadence, but a highly significant time x trial interaction effect for heart rate with higher heart rate rates recorded in the first half of the $COMP_{TT}$ ($F_{2.7,\ 70.1}$=9.9; p<0.001; η_p^2=0.275).

Perceptual Data

No significant time x trial x group interaction effects were found for perceived physical and mental strain, hedonicity, and felt arousal. However, there were significant simple time × trail interaction effects for p-RPE ($F_{5.0,\ 131.2}$=6.8; p<0.001; η_p^2=0.198), TEA ($F_{5.1,\ 133.3}$=3.0; p=0.013; η_p^2=0.102), and FAS ($F_{5.8,\ 151.6}$=2.4; p=0.034; η_p^2=0.089)

scales, respectively, with higher scores in the first half of the $COMP_{TT}$ when compared to $INDV_{TT}$.

In contrast, there were no significant simple time x trial effect for hedonicity, but there was a highly significant simple trial x group interaction effect ($F_{2.7, 70.1}$=9.9; p<0.001; η_p^2 =0.275) with winners and losers equally experiencing greater pleasure and displeasure, respectively, when compared to their $INDV_{TT}$.

A highly significant time x trial x group interaction effect was found for action crisis ($F_{3.9, 102.4}$=8.5; p<0.001; η_p^2 =0.247). Simple time × trail interaction effect was highly significant for losers only ($F_{3.2, 41.4}$=9.0; p<0.001; η_p^2 =0.409). Simple, simple (main) group effects in losers became significantly different from the $INDV_{TT}$ from the first climb onwards ($F_{1, 26}$=5.3; p=0.038; η_p^2 =0.290) and rose continuously becoming highly significant at the finish line ($F_{1, 26}$=39.2; p<0.001; η_p^2 =0.751).

Haematological Data

No significant time × trial × group, but significant time × trial interaction effects were observed for blood glucose and lactate concentrations with higher values for glucose ($F_{4, 84}$=5.9; p<0.001; η_p^2 =0.186) and lactate ($F_{2.8, 73.5}$=14.6; p=0.024; η_p^2 =0.359) at the start of the $COMP_{TT}$.

There was a trend for a three-way interaction effect for blood cortisol concentrations, but only simple time × trial ($F_{2.5, 64.9}$=3.8; p=0.019; η_p^2 =0.129) and trial × group ($F_{1, 26}$=5.4; p=0.029; η_p^2 =0.171) interaction effects reached significance with greater increases during the $COMP_{TT}$ compared to $INDV_{TT}$, and greater increases in losers compared to winners.

Lastly, there was no significant three-way interaction effect for blood norepinephrine concentrations, but the trial × group interaction effect reached significance ($F_{3.2, 41.4}$=4.7; p=0.040; η_p^2 =0.152) with losers only showing higher blood norepinephrine concentrations during the $COMP_{TT}$.

SUPPLEMENTARY MATERIAL TWO (CHAPTER FOUR)

Three-Way (treatment × time × sex) RM ANOVA

Haematological Data

There were no significant treatment × time × sex interaction effects for blood concentrations in lactate ($F_{2.8,\ 55.9}$=0.4; p=0.771; η_p^2=0.02), cortisol ($F_{2.0,\ 39.9}$=1.2; p=0.306; η_p^2=0.06) and interleukin-6 (ART: $F_{2.0,\ 40.3}$=1.6; p=0.218; η_p^2=0.07), but there were trends towards greater blood cell count for female runners during the intervention trial in leucocytes ($F_{2.9,\ 57.3}$=2.5; p=0.067; η_p^2=0.11) and neutrophils ($F_{2.2,\ 44.0}$=2.6; p=0.077; η_p^2=0.11).

However, significant main sex effects with greater blood concentration in female runners were observed in cortisol ($F_{1,\ 20}$=7.5; p=0.013; η_p^2=0.27) as well as greater leucocyte ($F_{1,\ 20}$=4.7; p=0.042; η_p^2=0.19) neutrophil counts ($F_{1,\ 20}$=3.9; p=0.063; η_p^2=0.16). These effects disappeared after controlling for sex dependent differences in weekly mileage for cortisol ($F_{1,\ 19}$=1.1; p=0.313; η_p^2=0.05), leucocytes (ANCOVA: $F_{1,\ 19}$=0.7; p=0.426; η_p^2=0.03) and neutrophils (ANCOVA: $F_{1,\ 19}$=0.3; p=0.589; η_p^2=0.02), respectively.

Perceptual Data

All perceptive data were additionally analysed with a non-parametric factorial procedure (ART) using common analysis of variance after align rank transform. The results are provided in squared brackets after the parametric test results in parenthesis.

There were no significant treatment × time × sex interaction effects for perceived physical strain ($F_{5.3,\ 105.1}$=1.4; p=0.248; η_p^2=0.06) [ART: $F_{9,\ 180}$=1.4; p=0.206; η_p^2=0.06], perceived mental strain ($F_{5.4,\ 108.3}$=0.9; p=0.475; η_p^2=0.04) [$F_{9,\ 180}$=1.3; p=0.233; η_p^2=0.06], valence ($F_{4.5,\ 90.8}$=0.8; p=0.570; η_p^2=0.04) [ART: $F_{4.5,\ 89.5}$=0.9; p=0.487; η_p^2=0.04], felt activation ($F_{9,\ 180}$=0.8; p=0.585; η_p^2=0.04) [ART: $F_{5.4,\ 108.5}$=0.5; p=0.761; η_p^2=0.03], perceived action crisis ($F_{3.3,\ 65.5}$=1.9; p=0.125; η_p^2=0.09)

[ART: $F_{3.5, 69.2}$=1.7; p=0.160; η_p^2=0.08] and perceived flow state ($F_{3.2, 64.6}$=1.1; p=0.378; η_p^2=0.05) [ART: $F_{3.6, 71.4}$=1.1; p=0.402; η_p^2=0.05].

There were also no main sex effects for perceived physical strain ($F_{1, 20}$=0.3; p=0.591; η_p^2=0.02) [ART: $F_{1, 20}$=0.2; p=0.625; η_p^2=0.01], perceived mental strain ($F_{1, 20}$=0.9; p=0.349; η_p^2=0.04) [ART: $F_{1, 20}$=0.8; p=0.383; η_p^2=0.04], valence ($F_{1, 20}$=0.6; p=0.447; η_p^2=0.03) [ART: $F_{1, 20}$=0.5; p=0.486; η_p^2=0.03], felt activation ($F_{1, 20}$<0.0; p=0.961; η_p^2 <0.00) [ART: $F_{1, 20}$<0.0; p=0.979; η_p^2<0.00], perceived action crisis ($F_{1, 20}$<0.0; p=0.917; η_p^2<0.00) [ART: $F_{1, 20}$<0.0; p=0.845; η_p^2<0.00] or perceived flow state ($F_{1, 20}$=0.6; p=0.430; η_p^2=0.03) [ART: $F_{1, 20}$=0.1; p=0.728; η_p^2=0.01].

Performance Data

There were no significant treatment x time x sex interaction effects for split time ($F_{3.6, 72.3}$=0.8; p=0.531; η_p^2=0.04), performance fatigability ($F_{3.8, 76.2}$=0.9; p=0.479; η_p^2 =0.04) or heart rate ($F_{3.1, 61.9}$=0.8; p=0.490; η_p^2=0.04).

However, significant main sex effects were observed for split time ($F_{1, 20}$=51.6; p=0.000; η_p^2=0.72). These effects disappeared after controlling for sex dependent differences in body fat percentage ($F_{1, 19}$=0.6; p=0.432; η_p^2=0.03).

SUPPLEMENTARY MATERIAL THREE (CHAPTER FIVE)

Table 17: Zero-order correlations between trial-related differences in area under the curve of haematological indicators of exercise-induced muscle damage, blood cortisol concentration as well as sensory, affective, and cognitive indicators of perceived fatigability and performance fatigability.

	1	2	3	4	5	6	7
1. Δ AUC - Leucocytes	-						
2. Δ AUC - Neutrophils	.98**	-					
3. Δ AUC - Cortisol	.56**	.59**	-				
4. Δ AUC - Perceived physical strain	.54*	.52*	.33	-			
5. Δ AUC - Valence	-.65**	-.69**	-.58**	-.78**	-		
6. Δ AUC - Action crisis	.45*	.47*	.30	.40	-.51*	-	
7. Δ AUC - Flow state	-.41	-.42*	-.23	-.43*	.44*	-.74**	-
8. Performance fatigability	.36	.39	.51*	.12	-.36	.57**	-.58**

Abbreviations: Δ AUC=Trial-related difference in area under the curve. ** Correlation is significant at the 0.01 level (2-tailed). * Correlation is significant at the 0.05 level (2-tailed).

Table 18: Multiple hierarchical regression analyses of trial-related differences in area under the curve (Δ AUC) of blood cortisol concentrations and performance fatigability as well as perceived physical strain, valence, flow state and performance fatigability in response to Δ AUC in the respective direct predictor variable.

	Leucocytes ⇨ Cortisol		Neutrophils ⇨ Cortisol		Cortisol ⇨ Performance fatigability		Leucocytes ⇨ Perceived physical strain		Neutrophils ⇨ Perceived physical strain		Perceived physical strain ⇨ Valence		Valence ⇨ Flow state		Flow state ⇨ Performance fatigability	
Predictor	ΔR^2	β	ΔR^2	β	ΔR^2	β	ΔR^2	β	ΔR^2	β	ΔR^2	β	ΔR^2	β	ΔR^2	β
Step 1	.056		.056		.002		.066		.066		.022		.018		.002	
Age		-.130		-.130		.002		-.255		-.255		.035		.111		.002
Weight		.201		.201		.042		.036		.036		-.144		-.077		.042
Step 2	.196		.196		.012		.011		.011		.005		.020		.012	
Weekly mileage		.172		.172		.150		.058		.058		.029		-.148		.150
Other training		.538		.538		.124		-.063		-.063		-.051		.009		.124
Step 3	.004		.004		.047		.220		.220		.148		.003		.047	
VO_2peak		-.069		-.069		-.182		.463		.463		-.175		.028		-.182
Economy		.067		.067		.244		-.514		-.514		.438		.050		.244
Step 4	.372**		.395**		.291*		.307**		.276**		.528**		.209^		.346*	
Direct predictor		.708**		.726**		.626*		.643**		.608**		-.866**		.503^		-.601*

** Significant at the 0.01 level (2-tailed). * Significant at the 0.05 level (2-tailed). ^ Significant level = 0.068 (2-tailed).

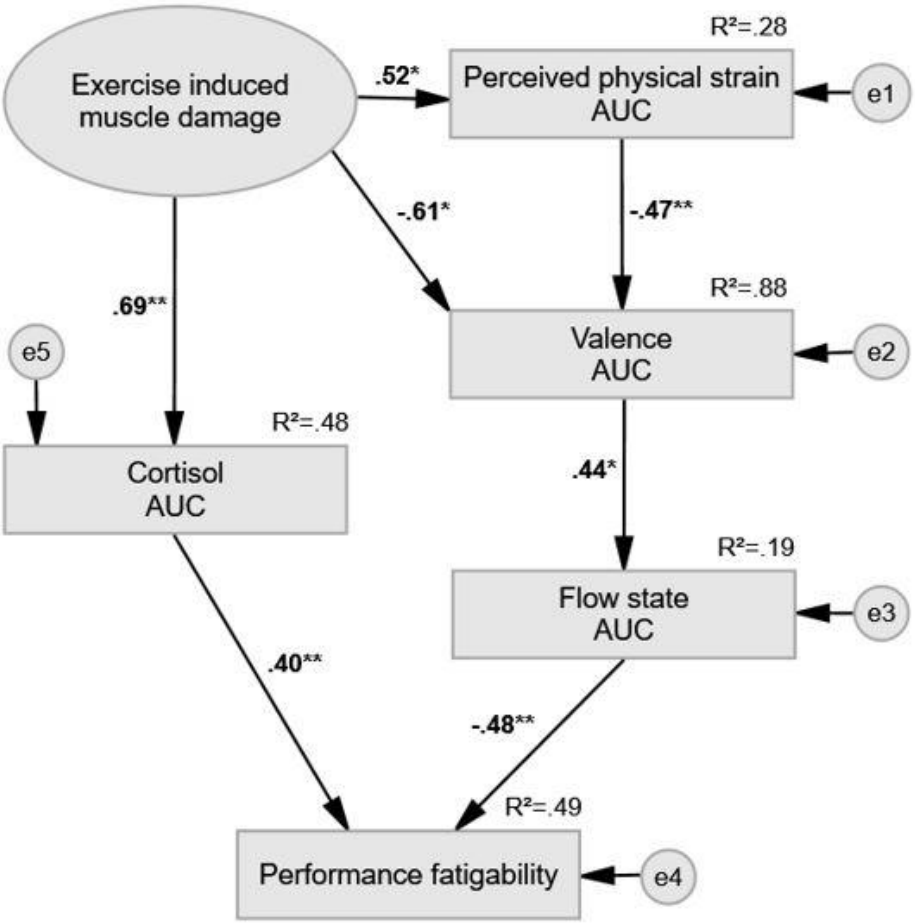

Figure 21: Structural equation modelling of physiological distress and perceived fatigability in response to running with exercise induced muscle damage and their effect on performance fatigability.

Note: Difference in area under the curve (AUC) of leucocytes and neutrophils between intervention ($INTV_{TT}$) and control time trial ($CTRL_{TT}$) were used to indicate extent of exercise induced muscle damage (for reasons of graphical simplicity not shown). All variables are expressed as differences between $INTV_{TT}$ and $CTRL_{TT}$.

Abbreviations: Squares represent observed variables; Ovals are latent variables; Single headed arrows represent regression paths; Bold regression paths are significant at * $p < 0.05$ and ** $p < 0.01$, respectively; AUC = area under the curve; R^2 = total variance explained; e = residual error; Standardised maximum likelihood measures are used. χ^2 = Chi-square; NFI = normed fit index; CFI = comparative fit index; RMSEA = root mean square error of approximation; PCLOSE = p of close fit; AIC = Akaike information criterion; SRMR = standardised root mean square residual.

Model fit indices are: $\chi^2 = 10.032$, $p = .528$, $\chi^2/11 = .912$, NFI = .931, CFI = 1.000, RMSEA = .000 (95%CI = [.000, .213]; PCLOSE = .573), AIC = 58.032, SRMR = .069.