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Bachelor's project in Human Movement Science
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Kunnskap for en bedre verden

Abstract

Purpose: The first ventilatory threshold (VT1) and the lactate threshold (LT) are used to evaluate cardiorespiratory function and individually determine moderate and hard exercise intensities. Exercise tests to determine these thresholds are often done in thermoneutral conditions. Different ambient temperature puts different physiological strains on the organism during exercise. This literature review will therefore investigate the effect of cold (-30–13°C) and hot (25–40°C) ambient temperatures on VT1 and LT compared to thermoneutral temperatures (13–25°C). **Method:** The databases Pubmed, SPORTDiscuss and Google Scholar were searched for studies comparing VT1 and/or LT at different ambient temperatures. Studies included had to present cycling power, running velocity or oxygen uptake (VO₂) at VT1 and/or LT. **Results:** Ten studies were included. Four studies compared thermoneutral and cold conditions, and six studies compared thermoneutral and hot conditions. The main findings are that VT1 and LT are dependent on ambient temperature. Performance at VT1 and LT were improved in moderate cold (~0°C) in three studies and deteriorated in all studies containing hot conditions. **Conclusion:** Both VT1 and LT appear to occur at an increased performance level in the cold and at a lower performance level in hot conditions.

Abstrakt

Bakgrunn: Den første ventilasjons-terskelen (VT1) og laktattterskelen er brukt for å evaluere kardiorespiratorisk funksjon og individuelt bestemme moderate og harde treningsintensiteter. Fysiske tester for å bestemme disse tersklene blir ofte gjennomført i termonøytrale forhold. Ulike temperaturer i omgivelsene påvirker de fysiologiske systemene i kroppen ulikt. Derfor vil denne litteraturstudien undersøke effekten kalde(-30–13°C) og varme(25–40°C) temperaturer i omgivelsene har på VT1 og LT sammenlignet med termonøytrale temperaturer (13–25°C). **Metode:** Databasene Pubmed, SPORTDiscuss og Google Scholar ble brukt for å søke etter studier som sammenligner VT1 og/eller LT i ulike temperaturer. De inkluderte studiene måtte inneholde enten sykkeeffekt, løpefart eller oksygenopptak (VO₂) ved VT1 og/eller LT. **Resultat:** Ti studier ble inkludert. Fire studier sammenlignet termonøytrale og kalde forhold og seks studier sammenlignet termonøytrale og varme forhold. Hovedfunnene er at VT1 og LT forandres avhengig av temperatur. Prestasjon ved VT1 og/eller LT økte i moderat kulde (~0°C) i tre studier og ble forverret i varme i alle studiene som inneholdt varme forhold. **Konklusjon:** Det er tilsynelatende økt prestasjon ved både VT1 og LT i kalde forhold og forverret prestasjon ved VT1 og LT i varme forhold.

Introduction

Athletes compete in a variety of environmental conditions. Temperatures during the 2021 Tokyo Olympics may rise beyond 30°C, differences in temperature during mountain stages in road race cycling can be of 20°C, and competitions in cross country skiing can be held in temperatures down to -20°C. Different temperatures put different physiological strains on the athlete during exercise and endurance exercise capacity is impaired both in hot and cold conditions (1).

With increasing exercise intensity there is an increasing contribution of anaerobic metabolic pathways to the total metabolism (2,3). The increasing participation of anaerobic metabolic pathways together with an increasing total metabolism with increasing exercise intensity results in increasing levels of by-products from the metabolic pathways (2,3). Metabolic by-products like CO₂ and H⁺ trigger physiological responses by the organism (4). When exercise intensity is increased, there are points, often termed thresholds, after which the organism's response to a further increase in intensity is observed to change abruptly (2). These threshold concepts are used to evaluate cardiorespiratory capacity and individually determine exercise intensities with both invasive measurements of blood lactate concentration (bLa) or non-invasive measurements of expired CO₂ (VCO₂) and ventilation (V_e) (2). The different threshold concepts are usually determined for an individual during incremental exercise tests often done in laboratories with thermoneutral conditions. Thermoneutral testing ensures stable conditions between sessions and different laboratories, which enables better comparisons. Thermoneutral testing is often cheaper than testing done at more extreme temperatures in climate chambers. This is done even though exercise programs or/and competitions are done in cold or hot conditions. This may have implications for the validity of the measurement methods and may reduce the physiological transferability to conditions in training and competitions.

Research comparing thresholds within the transition from lower to higher contribution of anaerobic metabolic pathways between thermoneutral, cold, and hot conditions is limited. Therefore, a broad array of specific threshold definitions within two main threshold concepts will be used in this review. The two main threshold concepts in the review are the first ventilatory threshold (VT1) and the lactate threshold (LT). These threshold concepts are often used to differentiate between moderate and hard exercise intensity. VT1 can be defined as the exercise intensity at which the increase in ventilation becomes disproportional to the increase in power output or speed of locomotion during an incremental exercise test (5). Increased

VCO₂ can be seen with the disproportionate increase in ventilation at VT1. Wasserman et al proposed that this increased VCO₂ at VT1 was caused by bicarbonate buffering of H⁺ caused by elevated bLa (6). However, Perronet et al showed that concurrent bicarbonate buffering of H⁺ and hyperventilation caused the increased VCO₂ at VT1 (7). The hyperventilation reduces alveolar CO₂ concentration and increases diffusion, which results in an increased CO₂ output (7). The hyperventilation together with the relationship between lactate and acidosis discussed later, indicate that VT1 are not directly related to lactate (7).

LT can be defined as the intensity during an incremental exercise test at which bLa begins to rise above baseline levels(3). When there is no net accumulation of lactate in the blood there is an equilibrium between lactate production and removal (3). Lactate is mainly, but not exclusively produced by exercising muscles, and it is oxidized by both contracting and resting muscles, and other tissue (3). The lactate accumulation equilibrium is highly related to the metabolic rate, but not necessarily to oxygen availability (3). Lactate production is a means to reduce muscle acidosis from non-mitochondrial ATP turnover and production of cytosolic NAD⁺ to facilitate ATP regeneration from glycolysis (8). The lactate production, therefore, correlates with but is not the cause of metabolic acidosis (8). In the same matter is there no direct relationship between lactate accumulation and bicarbonate buffering (7,8). As a result of this relationship are the VT1 and LT not causally linked but correlate at best. Empirical evidence has indicated that VT1 and LT are not subserved by the same mechanism and that they occur at different times (9).

The body has a reduced ability to get rid of metabolic heat during exercise in hot conditions, because of a smaller temperature difference between air and skin. This is partly alleviated by increased sweating, increased cutaneous blood flow, and cutaneous blood pooling (10). Increased cutaneous blood flow and pooling impair the venous return to the heart which reduces stroke volume (10). An increased heart rate can compensate for the reduction in stroke volume during submaximal exercise in the heat (10). These cardiorespiratory responses to the heat impair the efficiency of oxygen transport to working muscles and other tissue, which in turn may impact the lactate production. The reductions in blood flow to visceral organs and non-working muscles impairs the ability to remove and oxidize lactate from the blood, as some of these organs are net consumers of lactate during exercise (3). Hyperthermia is accompanied by increased pulmonary ventilation both in rest and during exercise (11). The regulatory mechanism behind this is poorly understood but may be a means to increase heat loss (11). The increased ventilation has impacts on blood during

submaximal exercise as arterial CO₂ pressure is lowered, and pH is increased which may affect at which point the VT1 appears during an incremental exercise test (10).

The muscular temperature may decline during exposure to cold conditions, which results in diminishing contracting properties of the muscles (12). The reduction in muscular temperature in response to cold ambient temperatures can be attenuated by an increased metabolic rate, peripheral vasoconstriction, and clothing (13). An increased metabolic rate can be achieved through a higher work rate and/or shivering in resting muscles and the increased VO₂ during submaximal exercise has been partly attributed to shivering mechanisms (13). Peripheral vasoconstriction, clothing, and the reduced contracting properties of muscles in cold conditions may change patterns of motor unit recruitment and technique (12). The changes in motor unit recruitment and technique together with shivering may impair submaximal performance in cold conditions, which can result in earlier occurrence of VT1 and LT during an incremental exercise test.

Cold, thermoneutral, and hot conditions pose different physiological challenges on the organism during exercise. As the LT and VT1 have different regulatory mechanisms may different environmental conditions have differing influences on VT1 and LT. The goal for this review is therefore to determine how LT and VT1 change with different temperatures and if the change reflects changes in performance or the measure itself.

Methods

A literature search was carried out using the databases Google Scholar, SPORTDiscuss, and PubMed. Keywords used in the search were: Ventilatory threshold(s), Lactate threshold(s), and air temperature, ambient temperature, cold environment, cold air, hot air, heat, and hot environment.

Only peer-reviewed studies comparing VT1 and/or LT between thermoneutral and cold or hot conditions in healthy adults were included. Included studies had to be written in English and had to present cycling power, running velocity or oxygen uptake (VO₂) at VT1 and/or LT. Cycling power and running velocity were used as performance measures and VO₂ together with heart rate and bLa were used as physiological measures. Thermoneutral conditions were defined to an air temperature between 13°C and 25°C, as an expanded range of normal laboratory temperatures. Cold and hot conditions were subsequently defined to an air temperature between -30 °C and 13°C and 25°C and 40°C, respectively. Studies with bLa values at VT1 and LT above 4.0 mmol L⁻¹ were excluded to distinguish between other

threshold concepts like onset of blood lactate accumulation, maximal lactate steady state and the respiratory compensation threshold.

The original literature search generated 20 relevant studies based on title and abstract. The reference lists of the relevant studies brought about 6 new relevant studies. Inaccessible full text excluded 6 studies, 6 studies were excluded because of incomplete data, 3 studies were excluded by temperature definitions and 1 study was excluded because of LT and VT1 definitions. Determining a total of 10 included studies.

Results

The ten included studies measured LT and/or VT1 during incremental exercise tests in at least two different environmental conditions. Subject characteristics, relevant methods, and results from the comparisons between the environmental conditions in each study are presented in table 1. Two studies included both LT and VT1, four included only LT, four only VT1. Six studies compared thermoneutral and hot conditions, and four studies compared thermoneutral and cold conditions. Eight studies compared two environmental conditions, one compared three, and one compared six. The mean number of subjects was 10.4, and the mean subject age ranged from 21 to 37 years. Only two of the ten studies contained female subjects. The mean temperature difference between the two most extreme conditions compared in each study was $21.31 \pm 9.7^\circ\text{C}$ and ranged from 6.6°C to 40.0°C . Total exposure to the environmental conditions ranged from 10 minutes to above one hour and mean incremental test duration ranged from 7 minutes to above 20 minutes.

Three studies measured performance at LT in moderate cold ($\sim 0^\circ\text{C}$) (14–16). They all found improvements in performance at LT in moderate cold compared to thermoneutral conditions. One of these studies, by Therminarias et al, found an increased performance level at VT1 as well (14). VO_2 at LT and VT1 was higher in the cold conditions in three studies, however Quiron et al only observed the increase in VO_2 in -20°C and not in 0°C , although the percentage of VO_2max ($\% \text{VO}_2\text{max}$) was increased in both -20°C and in 0°C (14,16,17).

Two studies measured performance at LT in hot conditions and observed a lower performance level at LT and a reduced VO_2 in the hot condition compared to the thermoneutral condition (18,19). Four studies measured performance at VT1 in hot conditions and they all found a reduced performance level at VT1 in the hot condition compared to thermoneutral conditions (20–23). This reduction in performance at VT1 in the hot condition was accompanied by an increased heart rate in three studies (21–23). VO_2 at VT1 was equal

or reduced in the hot condition compared to the thermoneutral condition in the three studies that measured VO_2 at VT1 in a hot condition (20,22,23).

Table 1: Each study's methods and results with mean and standard deviation by environmental condition.

Study	Method	Environmental conditions	Relevant findings		
First author, publication year.	Movement form, number of subjects, subject characteristics, mean subject age, mean VO ₂ max.	Protocol for incremental exercise tests, clothing used, definitions used to determine LT and VT1.	Ambient temperature, relative humidity, air velocity.	VT1 and/or LT Cycling power (W)/running velocity (km h ⁻¹), VO ₂ ((mL min ⁻¹ kg ⁻¹)/ (L min ⁻¹)), % VO ₂ max, heart rate (bpm), bLa (mmol L ⁻¹).	Max Cycling power (W)/Running velocity (km h ⁻¹), VO ₂ ((mL min ⁻¹ kg ⁻¹), bLa (mmol L ⁻¹).
Therminarias et al (14) 1989	Cycling n=8 Trained male football players at a regional-level 21 ± 2 years	Tests were performed 1 week apart. Subjects had 4-5 min rest before the test in the climate chamber. The starting workload was 0 W and was then increased by 15 W 2 min ⁻¹ and 30 W 2 min ⁻¹ after 30 W. Subjects were lightly dressed in shorts, shirts, and athletics shoes. LT was defined as the average of the two VO ₂ levels which immediately preceded and followed the nonlinear increase in bLa above the resting level. VT1 was defined as the average of the two VO ₂ levels which immediately preceded and followed the nonlinear rise in Ve.	24°C 10 m/s	VT1 156 ± 8 W 32.5 ± 1.4 mL min ⁻¹ kg ⁻¹ 65.0 ± 3.9 % 154 ± 7 bpm 3.29 ± 0.29 mmol L ⁻¹ LT 142 ± 7 W 29.4 ± 1.9 mL min ⁻¹ kg ⁻¹ 59.6 ± 1.9 % 147 ± 7 bpm 2.74 ± 0.31 mmol L ⁻¹	

Therminarias
et al (14)
1989

-2°C
10 m/s
VT1
165 ± 8 W
39.6 ± 1.7 mL min⁻¹ kg⁻¹
67.7 ± 2.5%
136 ± 4 bpm
2.67 ± 0.26 mmol L⁻¹

LT
180 ± 6 W
43.6 ± 2.0 mL min⁻¹ kg⁻¹
74.5 ± 2.3%
152 ± 6 bpm
3.04 ± 0.21 mmol L⁻¹

Quirion et al
(17)
1989
Cycling
n=8
Healthy males
25.1 ± 3.6
years
72.0 ± 5.4 mL
min⁻¹ kg⁻¹
Subjects had 5 min rest before the test in
the climate chamber.
The starting workload was 100 W and
was increased by 50 W 2 min⁻¹
Subjects were lightly dressed in shorts,
shirts, and athletic shoes in 20°C, cross-
country skiing suits in 0°C and -20°C.
LT and VT1 were determined by a non-
linear increase in bLa vs VO₂ and Ve vs
VO₂, respectively.

20°C
60 ± 1% RH
VT1
3.69 ± 0.39 L min⁻¹
71.5 ± 6.3%
LT
3.95 ± 0.45 L min⁻¹
76.4 ± 2.6%

Max
381.2 ± 25.9 W
5.2 ± 0.5 L min⁻¹
11.4 ± 3.1 mmol L⁻¹

0°C
60 ± 1% RH
VT1
3.74 ± 0.27 L min⁻¹
76.9 ± 2.9%
LT
3.95 ± 0.27 L min⁻¹
81.3 ± 4.3 %

Max
337.2 ± 35.3 W
4.9 ± 0.5 L min⁻¹
11.1 ± 2.2 mmol L⁻¹

-20°C
60 ± 1% RH
VT1
4.01 ± 0.32 L min⁻¹
81.4 ± 4.1%
LT
4.28 ± 0.20 L min⁻¹
83.6 ± 4.5%

Max
300.0 ± 37.8 W
4.9 ± 0.5 L min⁻¹
10.4 ± 1.8 mmol L⁻¹

Sandsund et al (15) 2012	Running n=9 Highly trained male endurance athletes 25 ± 3 years,	Test order was randomized. Subjects had a 10 min warm-up at 60% of VO ₂ max, then four 5-min stages with 1 km h ⁻¹ increments. They were dressed in wind boxer shorts, long-sleeved pullovers and long underpants, cross-country skiing suits, socks, headgear, and gloves. Headscarves and wool mittens were added for 1°C and lower temperatures. LT was defined as the intensity at which bLa reached 1.5 mmol L ⁻¹ above the average value derived from rest and warm-up values.	20°C 5 m/s	LT 9.7 ± 0.5 km h ⁻¹	Max 70.0 ± 4.7 mL min ⁻¹ kg ⁻¹			
			10°C 5 m/s	LT 9.7 ± 0.4 km h ⁻¹	Max 70.4 ± 5.7 mL min ⁻¹ kg ⁻¹			
			1°C 5 m/s	LT 10.0 ± 0.5 km h ⁻¹	Max 72.1 ± 4.3 mL min ⁻¹ kg ⁻¹			
			-4 °C 5 m/s	LT 10.1 ± 0.4 km h ⁻¹	Max 71.7 ± 4.1 mL min ⁻¹ kg ⁻¹			
			-9°C 5 m/s	LT 9.8 ± 0.4 km h ⁻¹	Max 69.8 ± 5.1 mL min ⁻¹ kg ⁻¹			
			-14 °C 5 m/s	LT 9.8 ± 0.5 km h ⁻¹	Max 70.0 ± 4.9 mL min ⁻¹ kg ⁻¹			
			Morrissey et al (16) 2019	Cycling n=7 Trained female cyclists and triathletes 37 ± 5 years 50.4 ± 3.0 mL min ⁻¹ kg ⁻¹	Test order was randomized. Subjects had 5 min rest before the test in the climate chamber. The starting workload was 2W per body mass (kg) and was increased by 25 W 4 min ⁻¹ . LT was defined as the exercise intensity prior to a 1.0 mmol L ⁻¹ or higher increase in bLa.	20°C	LT 182.1 ± 26.4 W 41.7 ± 3.2 mL min ⁻¹ kg ⁻¹ 161.0 ± 10.0 bpm 1.77 ± 0.65 mmol L ⁻¹	
						0°C	LT 200.0 ± 22.6 W 42.7 ± 4.1 mL min ⁻¹ kg ⁻¹ 164.0 ± 12.0 bpm 2.35 ± 0.58 mmol L ⁻¹	
Shou and Ishiko (19) 1994	Cycling n=8 Healthy males 19.8 ± 1.0 years 40.8 ± 3.3 mL min ⁻¹ kg ⁻¹	Subjects had 15 min rest before the test in the climate chamber. The starting workload was 30 W and was increased by 30 W · 3 min ⁻¹ . Subjects were dressed in sneakers, shorts, and socks. LT was defined as the VO ₂ at the time just prior to a continuous nonlinear increase in bLa.	25°C 30% RH 0.3 m/s	LT 115.7 ± 20.7 W 1.40 ± 0.12 L min ⁻¹ 55.9 ± 3.4 % 133 ± 8 bpm				
			40°C 30% RH 0.3 m/s	LT 111.4 ± 22.7 W 1.24 ± 0.22 L min ⁻¹ 47.1 ± 3.0 % 139 ± 14 bpm				

James et al (18) 2017	Running n=17 16 males, 1 female Amateur club runners 32 ± 13 years, 61.0 ± 6.2 mL min ⁻¹ kg ⁻¹	Test order was not randomized. Subjects had 10 min rest and then 5 min warm up before the test in the climate chamber. The starting speed was individually determined based on running performance and a familiarization trial. Stages of 3 min + 1 min rest for blood sampling, separated by increments of 1km h ⁻¹ . Running velocity at LT was calculated by solving a polynomial regression equation for bLa versus speed at 2 mmol L ⁻¹ .	13°C 50% RH	LT 12.3 ± 1.9 km h ⁻¹	Max 16.1 km h ⁻¹ 61.0 mL min ⁻¹ kg ⁻¹
			32°C 60% RH	LT 11.7 km h ⁻¹	Max 15.8 km h ⁻¹ 56.3 mL min ⁻¹ kg ⁻¹
Hue et al (20) 2010	Cycling n=7 Acclimated high level male cyclists 23.2 ± 1.3 years	Test order was randomized. Subjects had 1 hour rest before the test in the climate chamber. They had a 3 min warm up at 50 W, before the workload was increased by 25 W · 1 min ⁻¹ . VT1 was determined using the Validated V-slope method.	19.2 ± 0.9 °C 51.7 ± 1.3% RH	VT1 291.7 ± 66.4 W 46.8 ± 13.4 mL min ⁻¹ kg ⁻¹ 156 ± 13 bpm	Max 62.7 ± 6.4 mL min ⁻¹ kg ⁻¹
			25.8 ± 1.1 °C 63.7 ± 2.3% RH	VT1 285.8 ± 39.4 W 45.7 ± 10.6 mL min ⁻¹ kg ⁻¹ 154 ± 16 bpm	Max 60.0 ± 7.3 mL min ⁻¹ kg ⁻¹
de Barros et al (21) 2011	Cycling n=8 Healthy young untrained males 23.9 ± 2.4 years 47.8 ± 4.9 mL min ⁻¹ kg ⁻¹	Test order was randomized. The starting workload was 60 W and was increased by 15 W 3 min ⁻¹ . VT1 was defined as the lowest work rate at which the increase in Ve /VO ₂ was accompanied by an increase of Ve/ VCO ₂ , according to Wasserman et al (6).	22°C 50% RH	VT1: 156 ± 9 W 145 ± 2 bpm 3.56 ± 0.38 mmol L ⁻¹	
			40°C 50% RH	VT1 128 ± 6 W 153 ± 4 bpm 2.97 ± 0.39 mmol L ⁻¹	

Edwards et al (22) 2016	Cycling n=10 Moderately fit men 27 ± 4 years	Test order was randomized. Subjects had a 3 min warm up at 100 W, before the workload was increased by 20 W 1 min ⁻¹ . VT1 was determined using the Validated V-slope method.	20°C 60% RH	VT1 204.2 ± 16.6 W 31.8 ± 2.5 mL min ⁻¹ kg ⁻¹ 74.4 ± 6.3% 150.4 ± 13.5 bpm	Max 42.8 ± 3.4 mL min ⁻¹ kg ⁻¹
			34.5°C 65% RH	VT1 181.2 ± 14.5 W 28.9 ± 2.3 mL min ⁻¹ kg ⁻¹ 68.3 ± 5.5 % 154.9 ± 13.9 bpm	Max 42.5 ± 3.2 mL min ⁻¹ kg ⁻¹
Perez- Quintero et al (23) 2021	Cycling n=22 Healthy males 22.1 ± 2.2 years	Subjects had a 5 min warm up at 50 W, before the workload was increased by 25 W 2 min ⁻¹ . VT1 was detected using a three-phase model.	22 ± 2°C	VT1 134.1 ± 21.2 W 20.9 ± 6.3 mL min ⁻¹ kg ⁻¹ 134.1 ± 18.5 bpm	Max 40.5 ± 5.5 mL min ⁻¹ kg ⁻¹
			42 ± 2°C	VT1 128.6 ± 21.3 W 20.6 ± 3.9 mL min ⁻¹ kg ⁻¹ 146.3 ± 17.8 bpm	Max 39.0 ± 6.9 mL min ⁻¹ kg ⁻¹

Note: LT=lactate threshold, VT1=first ventilatory threshold, n=number of subjects, RH=relative humidity, VO₂=oxygen consumption, VO₂max=maximal oxygen consumption, % VO₂max=percent of maximal oxygen consumption at VT1/LT, bLa=blood lactate concentration.

Discussion

The impact of cold conditions

LT and VT1 appear to occur at an increased performance level in cold conditions and the magnitude of increase is lower for VT1 than LT. This is contrary to our hypothesis of performance decrements at LT and VT1 in cold conditions, and other studies' reports of both reductions of submaximal and maximal performance in cold conditions (1,12). Therminarias et al reported higher power levels exhibited at both LT and VT1 in cold conditions and Morrissey et al reported higher power levels at LT (14,16). Their results differ both in magnitude of the power difference and relative workload at LT, with a larger impact of cold conditions for Therminarias et al (14,16). The temperatures studied and exercise time were approximately the same, but the air velocity in Therminarias et al study increased the conductive cooling effect (14,16). This increased cooling effect may have a greater impact on the relationship between LT and cold ambient temperatures. Thereby can Therminarias et al's results be interpreted as an appropriate reinforcement of the same tendency witnessed by Morrissey et al.

LT was defined in the study by Morrissey et al as the exercise intensity prior to a $1.0 \text{ mmol}\cdot\text{L}^{-1}$ or higher increase in bLa (16). Therefore, should bLa at LT be roughly the same in both environmental conditions, as the baseline levels were similar. However, mean bLa in the study by Morrissey et al was 31.6% higher in the cold condition, which can indicate that LT was wrongly determined in the cold condition and there was no actual performance increase at LT (16). The same difference in bLa at LT was not seen in Therminarias et al's study where bLa at LT increased by only 10.9% (14). Therminarias et al defined LT as the average of the two VO_2 levels which immediately preceded and followed the nonlinear increase in blood lactate above the resting level (14). This nonlinear increase may be difficult to locate and may not take place or emerge for some subjects (24). The bLa function of intensity flattened in the cold condition, which increases the reliability problems with Therminarias et al LT definition (14). bLa is often measured by blood samples from fingers, especially during field testing, where more complex measurement methods are impractical. Morrissey et al sampled blood from fingers, whereas Therminarias et al sampled blood from the cubital vein (14,16). During cold exposure are peripheral vasoconstrictions used to conserve heat, which induces reductions in blood flow in the extremities (25). This may alter the blood samples validity to

measure whole body lactate accumulation. The difference in measuring location may have impacted the results, but which way is uncertain.

The higher relative workload at LT in cold conditions is evident also in Quirion et al's results (17). VO_2 at LT was the same in 0°C and 20°C but was higher in -20°C . $\% \text{VO}_{2\text{max}}$ was increased in 0°C and was even higher in -20°C (17). Quirion et al also observed lower maximal power levels and $\text{VO}_{2\text{max}}$ in the colder conditions (17). The higher performance levels at LT in cold conditions may therefore be ascribed to a displacement of LT, rather than actual higher performance in cold conditions. Indicating that an athlete using testing in cold conditions to determine LT, exercises at a higher relative workload than for LT determined in thermoneutral conditions.

Sandsund et al similarly to Therminarias et al and Morrissey et al reported higher performance levels at LT in cold conditions (14–16). They reported increased running speed at LT in moderate cold compared to thermoneutral, but the effect was not evident in the coldest conditions (14–16). Clothing may attenuate heat loss evident in cold conditions and therefore alter the effect of cold on LT. The amount of clothing in the study by Sandsund et al was larger than in both the study by Quirion et al and Therminarias et al (14,15,17). The extra amount of clothing may make the thermoregulatory strain in the thermoneutral condition more like the strain in hot conditions. The same impact may be seen on the moderate cold conditions, making them more like thermoneutral conditions in the other studies reviewed. Hot conditions may lower performance at LT as discussed later. Therefore, the performance increase at LT in moderate cold observed by Sandsund et al may be explained by the amount of clothing (15).

VT_1 in Therminarias et al's study occurred at a higher power level in cold, but the size of the increase was lower than for LT (14). This meant that LT preceded VT_1 in thermoneutral conditions, but VT_1 preceded LT in cold conditions (14). However, VT_1 preceded LT in all environmental conditions in Quirion et al's study even though $\% \text{VO}_{2\text{max}}$ at VT_1 increased in the cold conditions (17). This may indicate that VT_1 occurs before LT, but this conclusion is uncertain.

To summarize, both VT_1 and LT appear to occur at a higher performance level in cold conditions, but this increase in performance at VT_1 and LT may be ascribed to a displacement of the measures rather than actual performance increase, especially for LT.

The impact of hot conditions

The main findings are that both VT1 and LT appear to occur at a lower performance level in hot conditions compared to thermoneutral conditions. Both Shou and Ishiko, and James et al reported lower performance levels at LT in hot conditions compared to thermoneutral conditions (18,19). Shou and Ishiko reported significant ($p < 0.05$) decreases in power (3.7%) and VO_2 (11.4%) at LT in the hot condition (19). These decreases were accompanied with an increase in HR, which may indicate an increased strain on the cardiorespiratory system (19). James et al found a 4.9% decrease in running speed at LT and 7.7% decrease in VO_2max in the hot condition (18). This can be interpreted as the reduced running speed in heat is caused by an increased physiological strain and that LT occurs at approximately the same relative intensity in both conditions. The amount of decrement at LT in hot conditions may be exaggerated by the methodology by James et al (18). Air flow improves heat conduction if ambient temperatures are under body temperature. The absence of air flow, therefore, increases the heat strain in James et al study (18). The small air flow reported in Shou and Ishiko's study, may have negligible impact, but should increase the thermal strain in the hot condition as the ambient temperature was above body temperature (19). The lower performance levels at LT in the hot condition reported by both James et al and Shou and Ishiko may indicate that LT in hot conditions occurs at the same relative intensity and that performance at LT is impaired compared to thermoneutral conditions (18,19).

Four studies investigated VT1 in hot compared with thermoneutral conditions (20–23). They all reported lower power values at VT1 in the hot condition, and all apart from Hue et al reported an increased heart rate at VT1 (20–23). The greatest decrease in power at VT1 (18.0%) was seen by de Barros et al, which had the slowest increments in exercise intensity of all reviewed studies, but the environmental exposure time did not differ from most of the other studies, since the starting intensity was relatively high compared to VT1 values (21). Perez-Quintero et al investigated approximately the same temperature difference as de Barros et al, but found only a 4.1% decrease in power at VT1 (21,23). They also found equal VO_2 values at VT1 in both conditions, this differs from Hue et al and Edwards et al which found a slight reduction (20–23). Perez-Quintero et al and Hue et al both observed a reduction in VO_2max in the hot condition, which can imply that aerobic performance was reduced and that this decrement in performance is responsible for the lower power values at VT1 in hot condition (20,23). De Barros et al observed a lower bLa level at VT1 in the hot condition, which can imply that the reduction in power at VT1 was caused by a change in the VT1-

measure rather than by a reduction in performance (21). On the other hand, did they observe lower bLa and power levels in the hot condition at the maximal lactate steady state as well, which suggests that the power decrease should nevertheless be attributed to a reduction in performance (21). The smaller power reduction at VT1 in the hot condition seen by Hue et al can be mainly attributed to the low temperature difference between the conditions and an ambient temperature in the hot condition which can almost be categorized as thermoneutral(20). From the lower performance reductions at smaller temperature differences can a relationship be proposed between increased ambient temperature and performance reductions at VT1 and should additionally be dependent on air velocity and humidity.

The subjects were acclimatized to the more extreme condition only in the study by Hue et al(20). This could explain the smaller reduction in power at VT1 in hot conditions as others have shown that acclimatization improves cycling performance in heat (26). A correlation study by Lorenzo et al investigated the effect of acclimation on both ventilation and blood-based lactate threshold methods and their ability to predict time trial performance in two different environmental conditions (27). They found that both ventilatory and blood-based methods were valid predictors, but both overestimated time trial performance in the hot condition before acclimation (27). Acclimation did however not change the predictive power of blood-based methods but exacerbated the ventilatory methods' predictive power (27). Perez-Quintero et al included a heat acclimation protocol after the initial test included in this review (23). The heat acclimation protocol consisted of passive intervallic exposure to high temperature (23). They observed an elevated performance level after the acclimation in both thermoneutral and hot conditions (23). Power and VO_2 at VT1 in the hot condition rose more than in the thermoneutral condition after acclimation, which diminished the difference between the hot and thermoneutral conditions after acclimation (23). This may be interpreted together with the results by Hue et al as acclimation and acclimatization can alleviate the reduction in performance at VT1 in hot conditions (20).

Limitations

Most of the included studies have a small number of subjects, eight studies have below 11 subjects. James et al and Perez-Quintero et al had the highest numbers of subjects, with 17 and 22 respectively, which are more respectable numbers of subjects for these kinds of studies (18,23). As most included studies have low subject numbers, the evidence value of each individual study is limited, but the trends the studies indicate meaningful practical differences for LT and VT1 between the reviewed environmental conditions. Eight of the ten studies only included males, which may limit the relevance for females. The exposure time to the

environmental conditions was lower than seen in most competitions and training sessions with the same exercise intensities proposed by VT1 and LT. Only Hue et al had an exposure time above 1 hour, but they also had the smallest temperature difference (20). The limited environmental exposures further limit the practical implications of the review and the effect of longer exposure times should be studied in new research.

Conclusion

The apparent increase in performance at VT1 and LT in cold conditions may be caused by a change to the threshold measures or that optimal temperatures for submaximal endurance performance are lower than anticipated. Performance at VT1 and LT in hot conditions was reduced, in compliance with earlier literature. The strength of these conclusions is limited, and more research is needed to confirm the practical implications and examine the explanatory mechanisms behind them.

References

1. Galloway SD, Maughan RJ. Effects of ambient temperature on the capacity to perform prolonged cycle exercise in man. *Med Sci Sports Exerc.* 1997 Sep;29(9):1240–9.
2. Anderson G, Rhodes E. A Review of Blood Lactate and Ventilatory Methods of Detecting Transition Thresholds. *Sports Med Auckl NZ.* 1989 Aug 1;8:43–55.
3. Faude O, Kindermann W, Meyer T. Lactate Threshold Concepts. *Sports Med.* 2009 Jun 1;39(6):469–90.
4. Ogoh S, Ainslie PN, Miyamoto T. Onset responses of ventilation and cerebral blood flow to hypercapnia in humans: rest and exercise. *J Appl Physiol.* 2009 Mar 1;106(3):880–6.
5. Svedahl K, Macintosh BR. Anaerobic Threshold: The Concept and Methods of Measurement. *Can J Appl Physiol.* 2003 Apr;28(2):299–323.
6. Wasserman K, Whipp BJ, Koyl SN, Beaver WL. Anaerobic threshold and respiratory gas exchange during exercise. *J Appl Physiol.* 1973 Aug;35(2):236–43.
7. Péronnet F, Aguilaniu B. Lactic acid buffering, nonmetabolic CO₂ and exercise hyperventilation: a critical reappraisal. *Respir Physiol Neurobiol.* 2006 Jan 25;150(1):4–18.
8. Robergs RA, Ghiasvand F, Parker D. Biochemistry of exercise-induced metabolic acidosis. *Am J Physiol-Regul Integr Comp Physiol.* 2004 Sep 1;287(3):R502–16.
9. Walsh ML, Banister EW. Possible Mechanisms of the Anaerobic Threshold. *Sports Med.* 1988 May 1;5(5):269–302.

10. Nybo L, Rasmussen P, Sawka M. Performance in the Heat-Physiological Factors of Importance for Hyperthermia-Induced Fatigue. *Compr Physiol*. 2014 Apr 1;4:657–89.
11. White MD. Components and mechanisms of thermal hyperpnea. *J Appl Physiol*. 2006 Aug 1;101(2):655–63.
12. Oksa J. Neuromuscular performance limitations in cold. *Int J Circumpolar Health*. 2002 Jun;61(2):154–62.
13. Doubt TJ. Physiology of Exercise in the Cold. *Sports Med*. 1991 Jun 1;11(6):367–81.
14. Therminarias A, Flore P, Oddou-Chirpaz MF, Pellerei E, Quirion A. Influence of Cold Exposure on Blood Lactate Response During Incremental Exercise. *Eur J Appl Physiol*. 1989 Jan 1;58(4):411–8.
15. Sandsund M, Saurasunet V, Wiggen Ø, Renberg J, Færevik H, van Beekvelt MCP. Effect of ambient temperature on endurance performance while wearing cross-country skiing clothing. *Eur J Appl Physiol*. 2012 Dec;112(12):3939–47.
16. Morrissey MC, Kisiolek JN, Ragland TJ, Willingham BD, Hunt RL, Hickner RC, et al. The effect of cold ambient temperature and preceding active warm-up on lactate kinetics in female cyclists and triathletes. *Appl Physiol Nutr Metab*. 2019 Oct;44(10):1043–51.
17. Quirion A, Laurencelle L, Paulin L, Therminarias A, Brisson GR, Audet A, et al. Metabolic and hormonal responses during exercise at 20°, 0° and -20°C. *Int J Biometeorol*. 1989;33(4):227–32.
18. James CA, Hayes M, Willmott AGB, Gibson OR, Flouris AD, Schlader ZJ, et al. Defining the determinants of endurance running performance in the heat. *Temp Multidiscip Biomed J*. 2017 May 25;4(3):314–29.
19. Shou G-C, Ishiko T. The Effect of Different Environmental Conditions on Blood Lactate Accumulation, Lt and Obla During Incremental Exercise. *体力科学*. 1994;43(1):58–65.
20. Hue O, Antoine-Jonville S, Galy O, Blanc S. Maximal oxygen uptake, ventilatory thresholds and mechanical power during cycling in Tropical climate in Guadeloupean elite cyclists. *J Sci Med Sport*. 2010;13(6):607–12.
21. de Barros CLM, Mendes TT, Mortimer LÁCF, Simões HG, Prado LS, Wisloff U, et al. Maximal Lactate Steady State is Altered in the Heat. *Int J Sports Med*. 2011 Oct;32(10):749–53.
22. Edwards A, Deakin G, Guy J. Brain and Cardiorespiratory Responses to Exercise in Hot and Thermoneutral Conditions. *Int J Sports Med*. 2016;37(10):779–84.
23. Perez-Quintero M, Siquier- Coll J, Bartolomé I, Robles-Gil MC, Muñoz D, Maynar-Mariño M. Three weeks of passive and intervallic heat at high temperatures (100±2 °C) in a sauna improve acclimation to external heat (42±2 °C) in untrained males. *J Therm Biol*. 2021 Feb 1;96:102837.

24. Hopker JG, Jobson SA, Pandit JJ. Controversies in the physiological basis of the 'anaerobic threshold' and their implications for clinical cardiopulmonary exercise testing. *Anaesthesia*. 2011;66(2):111–23.
25. Cheung SS. Responses of the hands and feet to cold exposure. *Temperature*. 2015 Mar 31;2(1):105–20.
26. Racinais S, Périard JD. Benefits of heat re-acclimation in the lead-up to the Tokyo Olympics. *Br J Sports Med*. 2020 Aug 1;54(16):945–6.
27. Lorenzo S, Minson CT, Babb TG, Halliwill JR. Lactate threshold predicting time-trial performance: impact of heat and acclimation. *J Appl Physiol*. 2011 Jul;111(1):221–7.

