BMJ Open Sport & Exercise Medicine

Preseason aerobic and anaerobic tests for prediction of alpine skiing performance: a molecular perspective

Arnold Koller,[1] Wolfgang Schobersberger[2]

To cite: Koller A, Schobersberger W. Preseason aerobic and anaerobic tests for prediction of alpine skiing performance: a molecular perspective. *BMJ Open Sport & Exercise Medicine* 2019;**5**:e000510. doi:10.1136/bmjsem-2019-000510

Accepted 24 February 2019

[1]Institute for Sports Medicine, Alpine Medicine and Health Tourism, Tirol Kliniken GmbH, Natters, Tirol, Austria
[2]Institute for Sports Medicine, Alpine Medicine and Health Tourism, University for Health Sciences, Medical Informatics and Technology (UMIT), Hall, Tirol, Austria

Correspondence to
Dr Arnold Koller;
arnold.koller@tirol-kliniken.at

Alpine ski racing is a demanding and multifaceted sport requiring high levels of physical and technical competence.[1] Because of the complexity of the sport, the selection of useful sport-specific tests of physiological capacities is challenging.[1] Aerobic capacity is commonly measured using incremental cycle ergometer exercise, while anaerobic capacity is commonly tested using metabolically highly demanding all-out Wingate cycling.[2] High aerobic/anaerobic capacity of an elite alpine skier is considered an important physiological determinant of competitive success.[2] However, new findings on the molecular basis for exercise-induced fatigue do not support this assertion.[3–6]

The contractile function of skeletal muscle declines during intense or prolonged physical exercise, that is, fatigue develops.[3] Within the muscle fibres, fatigue is generally related to increased energy demands, in which effective ATP resynthesis is needed to match the dramatically increased ATP consumption during contractions.[3] In contracting muscle fibres, ATP is mainly consumed by actomyosin cross-bridges and the sarcoplasmic reticulum Ca^{2+} pumps.[3] Adequate ATP delivery to the ATP-consuming proteins is essential for normal cell function and integrity.[3] Obviously, mechanisms to prevent these catastrophic consequences of ATP depletion exist within the muscle fibres.[3] These mechanisms involve, on the one hand, effective metabolic systems to resynthesise ATP and, on the other hand, a fatigue-induced decline in ATP consumption.[3] The latter fatigue mechanisms, which inhibit contraction-dependent ATP consumption, are a major focus of a recent review.[3] Examples of exercises in which different fatigue mechanisms might limit performance are given in table 1 of the review by Cheng *et al.*[3] Importantly, these different fatigue mechanisms might limit performance in metabolically demanding exercises.

For example, increased production of reactive oxygen/nitrogen species (ROS) and impaired cellular Ca^{2+} handling are implicated in prolonged force depression observed in skeletal muscle after metabolically highly demanding all-out Wingate cycling.[4] Moreover, muscle biopsies taken 24 hours after high-intensity cycling exercise show an extensive fragmentation of the sarcoplasmic reticulum Ca^{2+} channels, the ryanodine receptor 1 (RyR1).[4] Interestingly, elite endurance athletes develop a prolonged force depression after metabolically highly demanding all-out Wingate cycling, but no ROS-dependent RyR1 fragmentation.[4]

By contrast, prolonged force depression after mechanically demanding eccentric contractions (100 drop jumps from a height of 0.5 m) is largely independent of Ca^{2+} and ROS, and RyR1 fragmentation is observed in only some recreationally active elderly subjects.[5] However, the force depression was not more marked in these subjects.[5] Moreover, force depression after mechanically demanding eccentric contractions are similar in both recreationally active subjects and endurance trained athletes, despite the antioxidant capacity being higher in endurance trained muscles.[5] Thus, the mechanisms underlying prolonged force depression after mechanically demanding eccentric contractions are dissimilar to those after metabolically highly demanding all-out Wingate cycling.

In addition, eccentric muscle activity is a titin based and not an O_2 ATP coupled contraction form. Shortly, when the sarcomeres of a skeletal muscle are stretched, for example, by gravitational forces, the titin immunoglobulin (Ig) domain segments and the PEVK region (The PEVK region is a titin spring element, which is rich in proline (P), glutamate (E), valine (V), and lysine (K) residues and is considered to be an intrinsically disordered protein region) extend.[6] Titin domain folding against a force represents a potential source of work production in muscles, which presumably acts synchronously with the actomyosin contractile mechanism.[6] This way, titin is an active component in the sarcomere that helps to maximise work output without consuming ATP.[6] Titin as a force generating muscle protein is a major focus of a recent review.[6] Titin Ig domain refolding under force as a source of work production speaks against ATP depletion as a central factor underlying the impaired contractile function after mechanically demanding eccentric contractions.

Alpine skiing can arguably be characterised as the only sport in which well-coordinated eccentric muscle action is the decisive element.[7] Eccentric muscle contraction is essential for opposing the high centrifugal forces experienced by skiers in carved turns.[7] Carved turns of successful skiers are characterised by a short and distinct eccentric steering phase.[7] Well-coordinated eccentric muscle activation is thus a key feature for success in competitive alpine skiing.[7] Consequently, the mechanisms underlying force depression after ski racing are similar to those after mechanically demanding drop-jump exercise.[5]

Fatigue develops rapidly during physical activities requiring a rate of ATP production that exceeds the aerobic capacity of the muscle fibres.[3] This type of fatigue is closely related to the need for ATP production by anaerobic metabolism,[3] for example, metabolically highly demanding all-out Wingate cycling. Anaerobic metabolism leads to accumulation of lactate and hydrogen ions and increased blood lactate concentrations were measured after alpine ski racing without affecting competitive performance.[1 3] However, the high metabolic loads experienced by alpine skiers essentially stem from the simultaneous activation of a plethora of trunk muscles necessary to maintain core stability and balance.[7] Moreover, a recent study shows that recreational alpine skiing is associated with prolonged eccentric quadriceps and hamstring fatigue.[8] Interestingly, concentric types of endurance training (metabolically demanding exercises) (eg, biking) do not prevent fatigue during eccentric (ie, skiing) types of endurance exercise (mechanically demanding exercises).[8]

Based on these findings and in agreement with a recent study, we suggest that preseason aerobic and anaerobic tests are of limited use for prediction of alpine skiing performance.[1] A valid and reliable test battery that can predict performance in alpine skiing seems to be lacking. Therefore, future research directed towards screening for valid components of athletic performance is required.[1]

Contributors AK and WS wrote this editorial.

Funding The authors have not declared a specific grant for this research from any funding agency in the public, commercial or not-for-profit sectors.

Competing interests None declared.

Patient consent for publication Not required.

Provenance and peer review Not commissioned; externally peer reviewed.

REFERENCES

1. Nilsson R, Lindberg A-S, Theos A, *et al*. Aerobic variables for prediction of alpine skiing performance – a novel approach. *Sports Med Int Open* 2018;02:E105–12.
2. Ferland P-M, Comtois AS. Athletic profile of alpine Ski racers: a systematic review. *J Stregth Cond Res* 2018;32:3591–600.
3. Cheng AJ, Place N, Westerblad H. Molecular basis for exercise-induced fatigue: the importance of strictly controlled Cellular Ca^{2+} handling. *Cold Spring Harb Perspect Med* 2018;8.
4. Place N, Ivarsson N, Venckunas T, *et al*. Ryanodine receptor fragmentation and sarcoplasmic reticulum Ca^{2+} leak after one session of high-intensity interval exercise. *Proc Natl Acad Sci USA* 2015;112:15492–7.
5. Kamandulis S, de Souza Leite F, Hernández A, *et al*. Prolonged force depression after mechanically demanding contractions is largely independent of Ca^{2+} and reactive oxygen species. *Faseb J* 2017;31:4809–20.
6. Hoppeler H. *Eccentric exercise. Physiology and application in sport and rehabilitation*. London: Routledge Research in Sport and Exercise Science, 2014: 138–44.
7. Freundt JK, Linke WA. Titin as a force generating muscle protein under regulatory control. *J Appl Physiol*;104.
8. Koller A, Fuchs B, Leichtfried V, *et al*. Decrease in eccentric quadriceps and hamstring strength in recreational alpine skiiers after prolonged skiing. *BMJ Open Sport Exerc Med* 2015;0:e000028.