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EDITORIAL

Improving $\dot{V}O_2$max in already trained athletes is a challenging but feasible task: some cues from ischaemic preconditioning

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$\dot{V}O_2$max is the most important factor influencing endurance performance. Olympic medallists in events lasting from a few minutes to several hours generally have excellent $\dot{V}O_2$max values (i.e., above 80 or 68 ml·kg^{-1}·min^{-1} for males and females, respectively). $\dot{V}O_2$max is so vital that increasing it in athletes, even without training, simply by enhancing oxygen delivery through methods like hyperoxic breathing or blood transfusion, is usually linked to a rightward shift in the lactate-intensity relationship and improved endurance performance. However, endurance capacity may also be enhanced without expanding $\dot{V}O_2$max via metabolic and neural adaptations. Additional mechanisms that allocate a larger proportion of $\dot{V}O_2$ to active muscles could also help improve muscle $\dot{V}O_2$peak while whole-body $\dot{V}O_2$ remains similar, for example, by reducing the cost of breathing and redirecting a significant share of the oxygen delivery to the active muscles.

Untrained healthy adults can increase $\dot{V}O_2$max by ~15–20%, reaching 50–90% of their potential after 12–16 weeks, and likely approaching 95% of their potential for improvement within 9–12 months of training (Scharhag-Rosenberger et al. 2009). Therefore, the challenges are twofold: first, to accelerate the increase in $\dot{V}O_2$max with training; and second, to overcome barriers to further adaptation when the potential for enhancing $\dot{V}O_2$max appears to be exhausted.

In top-level endurance athletes, the main factor limiting $\dot{V}O_2$max is oxygen delivery, because endurance exercise training shifts the limitation from the capacity to utilise $\dot{V}O_2$ to the capacity to transport $\dot{V}O_2$ (Broxterman et al. 2024). Although, in theory, $\dot{V}O_2$max can also be increased by enhancing O_2 extraction, highly trained male and female athletes already extract 90–95% of the O_2 delivered to their exercising legs, implying that there is barely room for further enhancement through training (Skattebo et al. 2025).

It is well established that sprint interval training (SIT) is more effective than high-intensity interval training (HIIT) or continuous endurance training for enhancing $\dot{V}O_2$max within a short period (i.e., 2–4 weeks) (Molmen et al. 2024). However, the mechanisms behind the short-term superiority of SIT remain unclear, as answering this question requires a large study population and the use of invasive procedures capable of detecting subtle adaptive differences. Sprint interval training has been shown to induce cardiac hypertrophy, increase blood volume, and improve cardiac output within 6 weeks of training (Eriksson et al. 2024). Given that training at supramaximal intensities may enhance or accelerate hemodynamic adaptations, it is not surprising that studies combining the usual training with speed-endurance training (i.e., training at intensities that elicit O_2 demands above $\dot{V}O_2$max) enhance performance in already trained athletes (Bangsbo et al. 2025). Under this rationale of merging stimuli to boost the adaptive response to training, some success has been reported by combining the usual training with blood flow restriction (Thompson et al. 2024), irrespective of changes in blood volume. Since hypervolemia may facilitate cardiac filling and cardiac output during maximal exercise, some strategies have focused on interventions that increase blood volume, such as altitude training, heat-stress training, or combining both (Nybo et al. 2024).

In this pursuit of more efficient training methods to improve $\dot{V}O_2$max, Loukas et al. (2026) reported a clear advantage of a training program that included ischaemic preconditioning before the training sessions. The training

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involved two HIIT sessions at 90–100% $\dot{V}O_2$max and three continuous sessions (70–80% $\dot{V}O_2$max) per week, for eight weeks. Before the training sessions, eight participants underwent 3 × 5 min of total blood flow occlusion of the leg's circulation, while the other eight underwent a similar procedure without pressure applied around the thigh. $\dot{V}O_2$max increased by 7.6 and 2.7% in the groups training with and without ischaemic preconditioning, respectively. As expected, the improvement in $\dot{V}O_2$max was accompanied by faster running times, with greater improvement in the group trained with ischaemic preconditioning. The changes in $\dot{V}O_2$max were accompanied by increases in blood and plasma volumes, which were more pronounced in the group training with pre-exercise ischaemia. Thus, Loukas et al. (2026) nicely demonstrate that adding ischaemic preconditioning heightens the haematological adaptations elicited by endurance training. How is this superiority of ischaemic preconditioning explained? It was found that the ischaemic group achieved a 3.3% higher average training velocity than controls during HIIT sessions and, consequently, completed a greater total HIIT training volume. A deeper examination of the potential mechanisms by which ischaemic preconditioning may have contributed to enhance $\dot{V}O_2$max and performance may inspire future research in this field. Increased $\dot{V}O_2$max implies that either O_2 delivery and/or O_2 extraction has been improved. The reported haematological adaptations are consistent with increased oxygen delivery, likely mediated by increased cardiac output. Besides, the authors provide indirect evidence that a larger fraction of the cardiac output is distributed to the locomotory muscles, since the increased blood pressure response to maximal exercise on the cycle ergometer after ischaemic preconditioning training could be explained by a higher systemic vascular resistance. Furthermore, repeated ischaemia at rest may enhance exercise chemoreflex sensitivity, as shown during exercise in hypoxia (Seeley et al. 2022) and may reduce fatigue, as shown during isometric contractions (Allois et al. 2023).

Repeated ischaemic preconditioning at rest may induce local adaptations that facilitate muscle vasodilation and trigger adaptations that improve O_2 extraction via enhanced capillarization, antioxidant capacity, and mitochondrial biogenesis, although the evidence for such effects is weak (Jeffries et al. 2018; Martinez-Canton et al. 2024). Certainty regarding these contentions would require evaluation of central and peripheral haemodynamics using advanced invasive procedures (Skattebo et al. 2025). It also remains to be determined how ischaemic preconditioning may trigger haematological adaptations during subsequent training. Would ischaemic preconditioning alone, without training, elicit the observed haematological adaptations? Likely not. Alternative explanations include indirect mechanisms, such as attenuation of muscle cues that elicit pain, an effect that may be mediated by the release of adenosine in the ischaemic muscle and its subsequent binding to A_1 receptors in nociceptive endings (Goldman et al. 2010). Attenuating pain perception may allow training at higher intensity.

The study by Loukas et al. (2026) advances current knowledge of the interaction between ischaemic preconditioning and endurance training and offers new potential mechanistic explanations. Whether similar effects would be observed in top-level athletes is a much more challenging question for future studies.

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Declarations

Conflict of interest The author has developed a new device, "TRAIN-IRS" (Patent pending), which can be used to produce limb ischaemiaor blood flow restriction in humans.Improving

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