High VO_{2\text{MAX}} with no history of training is due to high blood volume: an alternative explanation

In a paper published in Medicine and Science in Sports and Exercise in 2002, Martino and colleagues concludethat some untrained subjects achieve high VO_{2\text{MAX}} values because their naturally high blood volumes optimise cardiac filling pressures, spontaneously producing higher maximum cardiac outputs, even without physical training.

It appeared to me that their data failed notonly to prove the authors’ hypothesis butthey could also not exclude an alternative interpretationalong the lines of our opposing hypothesis. However, the editor of the journal considered that my letter was too delayed (more than 12 months) after the publication of the original article and hence could not be considered for publication. This came as something of a surprise as I was unaware that there is now a “statute of limitations” within which published scientifiwork can be critiqued, where after it becomes the irreputable truth, immunised against further scrutiny. As it is possible that the British Journal of Sports Medicine does not subscribe to this “statute of limitations”, I would appreciate the publication of this letter so that it might add to the scientific debate. The crucial point in any analysis of the conclusions of Martino et al. is that two of the six subjects with high VO_{2\text{MAX}} had only “average” blood volumes. Hence their conclusionthat the high VO_{2\text{MAX}} values measured in some untrained subjects are “primarily” due to high blood volumes can only be 66% correct.

Indeed a crucial scientific challenge is to obviate the risk that incorrect conclusions of causality will be drawn from cross sectional studies simply because relatively few biological measurements are made. For example, the weakness of this study was that cardiac filling pressures were not measured. Thus the stated hypothesis could not be, and was not, tested. Hence any conclusions drawn remain purely speculative.

Furthermore, their interpretation is based on the popular cardiovascular/ananaerobic/catastrophic model, which holds that the cardiac output determines oxygen delivery to the exercising skeletal muscles and hence the exercise performance—the “oxygen push” explanation. But the authors’ finding that stroke volume and cardiac output increased linearly from rest to maximal exercise—in fact the slope of the stroke volume versus VO_{2} relation became increasingly positive at the two highest workloads (Figure 1; p 969)—shows that neither cardiac output nor stroke volume “plateaued” in these subjects. Yet, as argued at length in this journal, without a plateau in cardiac output at VO_{2\text{MAX}} it is difficult to understand how a maximal (limiting) cardiac output sets the upper limit for the VO_{2\text{MAX}} and performance in these subjects. Indeed, Dr. Gledhill’s group as well as others have consistently reported that the cardiac output does not “plateau” at VO_{2\text{MAX}}.

An alternative possibility is that the high cardiac outputs measured in some untrained subjects are the result and not the cause of their high VO_{2\text{MAX}} values. This contrasting “oxygen pull” model proposes that muscle performance is regulated by recruitment of fewer or a larger number of motor units in the active skeletal muscles. The increased number of motor units recruited by the central nervous system then increases oxygen demand and cardiac output. According to this interpretation, during maximal exercise testing, untrained athletes with higher VO_{2\text{MAX}} values must recruit a greater number of muscle fibres with high contractility than do those with lower VO_{2\text{MAX}}. As a result, the superior exercise performance is the cause of the high cardiac output and high VO_{2\text{MAX}} rather than their consequence.

That the athletes with higher VO_{2\text{MAX}} in the study of Martino et al. appear to recruit a larger muscle mass is suggested by the substantially lower total peripheral vascular resistance in the athletes with high VO_{2\text{MAX}} values. Indeed this finding could be used to argue that an altered ventricular afterload, and not just an altered preload, might contribute to the higher cardiac output of the high VO_{2\text{MAX}} group.

In summary, the “oxygen pull” model predicts that some untrained subjects can achieve high VO_{2\text{MAX}} values because they recruit a larger proportion of the available motor units in their active skeletal muscles. As a consequence, they achieve higher maximum work rates, producing lower total peripheral vascular resistances, higher venous returns, and greater maximum cardiac outputs, regardless of their blood volumes. The reason why some subjects may be able to recruit a larger proportion of their muscle mass at exhaustion during maximal exercise is unknown because it has yet to be studied.

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References


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