

## Attenuated skeletal muscle metabolism explains blunted reactive hyperemia after prolonged sitting

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**Introduction:** Although reduced post-occlusive reactive hyperemia (PORH) after prolonged sitting (PS) has been reported as impaired microvascular function, no specific mechanism(s) have been elucidated. One potential mechanism, independent of microvascular function, is that an altered muscle metabolic rate (MMR) may change the magnitude of PORH by modifying the oxygen deficit achieved during cuff-induced arterial occlusions. We speculated that if MMR changes during PS, this may invalidate current inferences about microvascular function during PS. **Objective:** Therefore, the objective of this study was to examine if peripheral leg MMR changes during PS and to ascertain whether the change in the oxygen deficit explains the change in PORH after PS. **Hypothesis:** We hypothesized that peripheral leg MMR would decrease during sitting and that the reduced oxygen deficit would explain the attenuation in PORH after PS. **Methods:** 31 subjects sat for 2.5 hours. A near-infrared spectroscopy (NIRS) sensor was placed on the medial gastrocnemius. PORH was quantified with the reoxygenation rate of the NIRS tissue oxygenation index (TOI) after 5-min of a cuff-induced arterial occlusion. MMR was quantified with the TOI slope during a cuff-induced arterial occlusion. Measurements were made before and after sitting. To determine if changes in MMR and the oxygen deficit explains the change in the reoxygenation rate, an artificial neural network (ANN) was trained to define the stimulus-response relationship between the oxygen deficit (i.e., difference in TOI during arterial occlusion ( $\Delta$ TOI) and the area under baseline TOI during arterial occlusion (AUC)), and the reoxygenation rate with 65 retrospectively collected PORH recordings. The ANN was then used to predict the experimentally collected reoxygenation rates at baseline and post-sitting. Therefore, if the ANN can replicate the experimental results, this provides evidence that the reoxygenation rate after sitting is dependent on MMR and the oxygen deficit during an arterial occlusion. **Results:** Consistent with other studies, the reoxygenation rate was reduced after PS ( $\Delta -0.27 \pm 0.55$  ln TOI% $\cdot$ s $^{-1}$ ,  $p < 0.01$ ). Unique to the current study, we discovered that MMR was reduced ( $\Delta -0.46 \pm 0.61$  ln TOI% $\cdot$ s $^{-1}$ ,  $p < 0.01$ ),  $\Delta$ TOI was reduced ( $\Delta -0.34 \pm 0.62$  ln TOI%,  $p < 0.01$ ), and AUC was reduced ( $\Delta -0.42 \pm 0.68$  ln TOI% $\cdot$ s,  $p < 0.01$ ) after PS. Furthermore, there were no differences between the ANN-predicted and experimental reoxygenation rates at baseline ( $p = 0.47$ ) or post-sitting ( $p = 0.33$ ). **Conclusions:** Since the oxygen deficit plays a significant role in the reduced reoxygenation rate after PS, current inferences about microvascular function with PORH after PS are not valid. Therefore, these data provide evidence that microvascular function may be preserved during PS. Interestingly, the blunted MMR after PS may not be trivial and could be an important mechanism underlying the relationship between PS and disease development.

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