

EXERCISE-INDUCED CALCITONIN GENE-RELATED PEPTIDE (CGRP) INCREASE DURING NORMOXIA AND HYPOXIA

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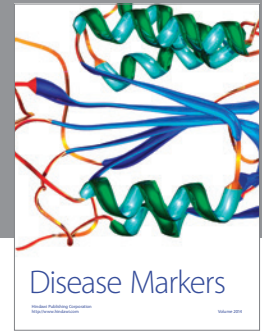
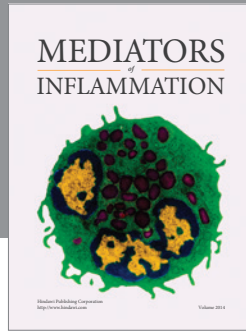
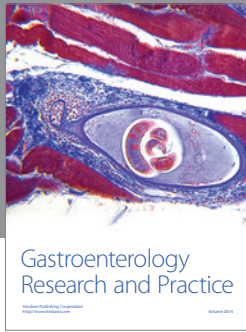
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To date calcitonin gene-related peptide (CGRP) is the most potent vasodilatory peptide described. Evidence indicates that the level of CGRP increases during exercise in humans. Furthermore, hypoxia, lactic acid (LA) and sympathetic activity affect the release of CGRP. We therefore studied how CGRP levels would be influenced by maximal exercise in humans during normoxia, acute and prolonged hypoxia.

Twelve healthy volunteers were investigated at sea level (SL), day 1 (HA1) and day 5 (HA5) in high altitude (4559 m). CGRP, calcitonin (CT), noradrenalin (NA), adrenalin (A), LA, and heart rate (HR) were measured at rest and during maximal exercise (W_{max}) on a bicycle ergometer. At SL, HA1, and HA5, CGRP increased significantly from resting levels to W_{max} along with NA, LA, HR, whereas A only increased significantly at sea-level and HA1. CT did not change at W_{max} . The CGRP levels at W_{max} were significantly decreased at HA1 (74.3 ± 6.1 pmol/l; $p = 0.008$) and HA5 (69.6 ± 6 ; $p = 0.003$) compared to CGRP (80.9 ± 5.1 pmol/l) at SL. This pattern was similar for LA and HR at W_{max} during acute and prolonged hypoxia, whereas the NA and A responses were not. At rest neither acute nor prolonged hypoxia changed the CGRP, CT, or LA levels, whereas NA, A, and HR changed significantly compared to SL.

The exact signal for release of CGRP cannot be stated from this study, but we hypothesize that the release of CGRP and LA are correlated and that CGRP does not solely act as a counter regulatory mechanism to the sympathetic vasoconstrictor NA.



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