Letter to the Editor

Letters to the Editor will be published, if suitable, as space permits. They should not exceed 1000 words (typed double-spaced) in length and may be subject to editing or abridgment.

Letter by Calderón-Gerstein Regarding Article, "Environmental Determinants of Cardiovascular Disease"

To the Editor:

I read with great interest the article of Bhatnagar[1] on the environmental determinants of cardiovascular disease. Concerning high-altitude influence on disease, some precisions are needed to make. In first place, Andean natives develop right ventricular hypertrophy, not left ventricular hypertrophy. This phenomenon has been observed at all ages,[2] and is noticeable since the sixth month of age, when the right ventricle fails to regress and, especially at altitudes higher than 4000 m, the cardiac axis remains shifted to the right permanently.[3]

The author also sustains that leptin is decreased at high altitude, but this has been found only in few studies, whereas in the majority of reports, leptin is usually increased at high altitude, especially in conditions of chronic hypoxia,[4] and >2500 m above sea level. This dissimilar findings may be because of a lack of adjustment of leptin measurement to environmental changes like temperature or humidity or variations in the diet or activity intensities of the evaluated subjects, as all these factors can alter its levels.[4] Current research shows that ghrelin values are always decreased after acute hypoxia and, henceforth, produce a decline in appetite and food ingestion, promoting weight loss at high altitude. Nevertheless, its role in the long term has not been well established.

As a final point, an important cardiac modification secondary to high-altitude exposure is the development of multiple collaterals of the coronary vessels at the microcirculation level. First described by Recavarren and Arias-Stella,[3] this anatomic modification is universal for all animals and individuals born at high altitude, failing to appear in elderly subjects who adapt to high altitude.[5,6] Angiogenesis secondary to chronic hypoxia has been observed in the coronary circulation of both ventricles and has been related to the AT1 (angiotensin II receptor type 1) receptor pathway in rats, while being linked to a simultaneous increase in VEGF (vascular endothelial growth factor) and caveolin-1, a capillarization marker.[7] This anatomic adaptation of the heart to chronic hypoxia conditions may be one of the main protection mechanisms against coronary ischemia, explaining in part the low rate of acute coronary events at high altitude.

Disclosures

None.

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