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Effect of Hypoxia in the Pharmacological Treatment of Chronic Mountain Sickness (monge sickness) in Native Residents of High Altitude

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ABSTRACT

Introduction: Altitude is a natural medical research laboratory which provides results with important implications for the understanding of diseases that affect millions of people living at altitude, as well as for the treatment of hypoxemia-related diseases in patients living at low altitude. Monge described a disease in the Peruvian Andes that is characterized by excessive polycythemia and congestive symptoms that are dramatically alleviated in the descent to lower levels.

Materials and methods: The research is descriptive, non-observational, non-experimental, cross-sectional, retrospective. A search was carried out in Pubmed for scientific articles published in both the Andean and South American countries. This search was done in both Spanish and English. The search for scientific articles included all articles that spoke of "mountain sickness", "hypoxia". The main exclusion criteria were articles that did not include information on altitude sickness in South America.

Results: Hemoglobin concentrations and hematocrits of normal native Cerro de Pasco were in the expected range but were found to be much higher in natives with chronic mountain sickness whose artery oxygen saturations of hemoglobin were 59.6, 74.2, 75.9, 78.8 and 80.0% or, in contrast to 81%, which is the mean value for that in Morococha (4540 m).

Discussion: To our knowledge, this is the first study to evaluate the effect of ACZ on ventilation and ventilatory control in subjects with CMS. We seek to determine whether chronic carbonic anhydrase inhibition could show similar therapeutic efficacy in chronic hypoxia, as occurs in subjects exposed to acute hypoxia, due to its effects on improving blood oxygenation.

Conclusions: Administration of ACZ provides a beneficial effect on the respiratory function of high-altitude natives with CMS and, therefore, may be an effective therapy for the disease. Defective synthesis of no by the pulmonary endothelium and alveolar epithelium has been shown to be the central event in the pathogenesis of exaggerated hypoxic pulmonary vasoconstriction.