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PERINATAL ORIGINS OF CHRONIC MOUNTAIN SICKNESS

The Role of Perinatal Hypoxia in the Development of CMS

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Chronic mountain sickness (CMS) is a significant public health problem for the more than 140 million persons residing at high altitudes (>2500 m) yet its etiology remains incompletely understood because, in part, the effects of normal aging obscure the identification of causal factors. Excessive erythrocytosis (EE, Hb ≥ 18.3 g/dL) has been shown, with time, to lead to CMS and therefore a preclinical form of CMS. We hypothesized that perinatal hypoxia increased susceptibility to CMS by impairing development of pulmonary structure and/or respiratory control. From a survey of 820 young (18-25 yr) male residents of La Paz/El Alto (3600-4100 m) of normal body weight, height and BMI, 8% had EE, arterial hypoxemia ($SaO_2 \leq 2$ SD altitude-specific mean) and/or pulmonary hypertension (>35 mmHg, systolic) with no known, or identifiable underlying disease. Among the 30 EE and 29 controls selected for detailed study, 63% of EE cases experienced perinatal hypoxia as manifested by being preterm (29%), born to a preeclamptic (PE) mother (38%), or experiencing neonatal hypoxia (38%) vs. a frequency of these conditions in controls of 24%, 11% and 6%, respectively. Birth weights of EE subjects were 378 g lower at birth than controls but this difference was not statistically significant. Alveolar ventilation and $FEF_{75\%}$ values were lower in EE than control subjects, whereas lung diffusion capacity, HVR, FVC, FEV_1 and FEV_1/FVC were equivalent. The majority of EE subjects showed evidence of pulmonary hypertension (62%), electro or echocardiographic evidence of right ventricular hypertrophy (67%). We concluded that exaggerated perinatal hypoxia may increase the susceptibility to CMS via alterations in pulmonary development and/or respiratory control. Impaired growth *in utero* has been shown to raise susceptibility to adult disease; these are the first data from a well-controlled study to demonstrate a possible influence of exaggerated perinatal hypoxia on susceptibility to CMS.

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