Letters to the Editor

In response to HMOX1 microsatellite polymorphism by Cao et al

To the Editor:

We read the article titled "HMOX1 promoter microsatellite polymorphism is not associated with high altitude pulmonary edema in Han Chinese" by Cao et al with profound interest.[1] The authors of this study have concluded that the microsatellite polymorphism in the HMOX1 gene promoter is not associated with high altitude (HA) pulmonary edema (HAPE) in Han Chinese. There has been a worldwide shift in focus among the scientific community toward research involving the proteomics and genomics aspects of this disease process. This is also evident in the present study, but the presentation of the physiologic/clinical aspects of the data appears to have taken a backseat.

We would like to seek more information regarding the status of the non-HAPE lowland participants in terms of the time spent at HA before their clinical examination was done and blood samples collected. Hematological parameters like hemoglobin level are definitely affected by the duration and quantum of hypoxia exposure in all individuals at HA.[2] Elaboration on this aspect would enable better understanding of the results of the comparison of the 2 groups. Similarly, a word on the average time for presentation of HAPE symptoms in patients after entry in HA before they were hospitalized could have elucidated their clinical presentation further. It would also be interesting to know the duration and details of HAPE treatment (oxygen/nifedipine) provided to these patients and the timing of clinical examination measurements in the hospital. If it was not done at the time of hospitalization, then clarification on cessation of treatment before the examination was carried out would be remarkable. It is evident that from the mean resting heart rate (118±17 beats · min^{-1}) of HAPE participants (n=83) that they had a moderate grade of HAPE.[3] Especially in the background of the very low blood oxygen level of HAPE participants (54±19%), additional information on respiratory rate of these participants would complete their clinical profile.

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In Reply to Drs Sikri and Bhattachar

To the Editor:

We thank Drs. Sikri and Bhattachar for their response[1] to our recent study, "HMOX1 promoter microsatellite polymorphism is not associated with high altitude pulmonary edema in Han Chinese," published in *Wilderness & Environmental Medicine*.[2] Our study has clarified the link between microsatellite polymorphism in the heme oxygenase-1 (HMOX1) gene promoter and high-altitude pulmonary edema (HAPE) in Han Chinese and may attempt to explore the pathogenesis of HAPE. The exact pathophysiologic mechanisms of HAPE are still unclear. Recently, increasing evidence has shown that HAPE is the result of a combination of genetic and environmental factors, but the role of genetics has not been clearly determined.[3,4] We assumed that heme oxygenase-1 promoter region microsatellite polymorphism may be related to HAPE.

The pathogenesis of HAPE is not only exaggerated hypoxic pulmonary hypertension, but may also involve the inflammatory response and the reduction of glucocorticoid release. We fully agree that the diagnosis and definition of HAPE require very strict criteria. In the present study, HAPE was diagnosed based on clinical symptoms and chest radiograph. All patients experienced illness within 3 to 5 days after arrival from low altitude to high altitude (3800 m). Blood samples were collected soon after patients were hospitalized. In the non-HAPE patients, measurements including hemoglobin, white blood cell count, oxygen saturation, and pulmonary artery pressure were collected at a hospital after they had been in Yushu for 5 to 7 days. Before travel to high altitude, all patients were healthy and had no clinical symptoms. According to our experience, the most satisfactory treatments of HAPE are field treatment using high-flow oxygen (8–12 $L \cdot min^{-1}$) or a hyperbaric chamber. At the same time, patients were given drug treatment including dexamethasom,[5] nifedipine/Regitin,[6] and furosemide. Clinical observations of heart rate, blood pressure, respiratory rate, and body temperature were periodically recorded. After treatment using these methods, all patients improved significantly within 24 hours and continued treatment in the hospital using Tibetan herbs, such as *Rhodiola rosea*,[7] ginkgo leaf tablets, and echinacoside[8] until complete recovery.

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Is the Water Rescue Service in Poland Heading in the Right Direction? Preparing Lifeguards to the Standards of First Aid in Europe

To the Editor:

Drowning is one of the leading causes of death among children under the age of 15 years.[1] The role of a lifeguard is to save people from drowning and to perform first aid[2] where necessary. Resuscitation methods and lifesaving techniques have changed and developed over the years, as knowledge expands and technology advances.[3] In Poland, however, changes in the law and a lack of regulation has seen the quality of lifeguards deteriorate.

In 2009, the voluntary water rescue service introduced a new division of rescue degrees: voluntary and professional degrees. The preparatory course for the junior lifesaver examination gave training in basic first aid skills, basic life support (BLS), and how to use the automated external defibrillator. The preparatory course for the lifesaver examination also taught basic resuscitation in simulated classes using standardized patients. Both these degrees were voluntary degrees. A candidate wanting to start a professional course to become a lifeguard (pool lifeguard, inland open water lifeguard, or surf lifeguard) not only had to have completed the volunteer lifesaver degree, but also had to hold the title of "rescuer," having completed the qualified first aid course (QFAC), which is an advanced first aid course using the R1 first aid kit.[4] The QFAC was a separate course, with classes being held in the practice room only and not in the water. The R1 rescue kit includes equipment for cardiopulmonary resuscitation, dressings,