significance. Methods 90 male SD rats were randomly divided into 10 groups, three experimental animals brought by the Golmud, Qinghai, Xi'an time-consuming and 1d (elevation 2,700 m), 2d to the Tibetan Tanggula (elevation 5,000 m), 3d to the Naqu (elevation 4,500 m), two experimental groups 1d directly to Lhasa, Tibet (altitude 3,658 m) and Naqu, feeding more than five groups to plateau after 24 h of slaughter; three different experimental groups 1d directly to high altitude (above sea level in Qinghai Golmud 2,700 m, Lhasa, Tibet, 3,658 m, Naqu 4,500 m) after 4 weeks of feeding killed, two group (in Xi'an, elevation 5 m), with the Western blot and conventional RT-PCR and real-time fluorescence quantitative PCR (real-time PCR detection) measured at different altitudes SD Heat shock protein in rat brain 70 (Hsp70) expression and brain differences in the natural expression of Hsp70 gene, light microscopy and transmission electron microscopy observation of animals in each group structural changes in brain tissue. Results Mammals have a different elevation of heat shock response genes, stress, high altitude when the rapid increase in the expression of mammalian Hsp70; Hsp70 can be high altitude (high altitude) induced. Conclusion: Hsp70 heat shock response in the rapid synthesis of high altitude hypoxic stress is conducive to maintaining the normal physiological function when the cells, Hsp70 and cell formation is proportional to hypoxia tolerance.

Keywords: High altitude, SD rats, Heat shock protein 70 (Hsp70), Western blot, RT-PCR real time quantitative PCR

Project: Tibet, the Office of Science and Technology Research Fund (2010), the State Ethnic Affairs Commission research project (2011), initial results.

Antibodies to hemeoxygenase-2 tagged with nanowires enhances neuroprotection in traumatic brain injuries

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Previous reports from our laboratory showed that traumatic brain or spinal cord injuries upregulates hemeoxygenase-2 (HO-2) proteins in various regions in the CNS. Drugs downregulating HO-2 protein expression resulted in neuroprotection. Thus, in present investigation the neuroprotective role of HO-2 antibodies in brain injuries was investigated in a rat model. Since nanowiring of drugs enhances then neuroprotective capabilities, influence of nanowired HO-2 antibodies was also examined in present investigation.

Traumatic brain injury (TBI) was produced by making a longitudinal incision into the right parietal cortex under Equithesin anesthesia and the rats were allowed to survive 5 h after the lesion. In separate group of rats HO-2 antibodies (1:20, monoclonal) were applied 10, 30 or 60 min after TBI. Another group of rats received identical HO-2 antibodies but tagged with TiO2 nanowires applied over the lesion at the same time intervals after trauma. Topical application of HO-2 antibodies given 10 or 30 min after TBI resulted in marked neuroprotection in terms of reduction in the blood–brain barrier (BBB) breakdown to Evans blue and radioiodine traces, edema formation and neuronal injuries. However, normal HO-2 antibodies did not give any effects when they are applied 60 min after TBI. On the other hand nanowired HO-2 was able to significantly reduce neuronal injuries and BBB disruption or brain edema even applied after 60 min TBI. The neuronal damages were tightly correlated with upregulation of HO-2 expression in both untreated and following HO-2 antibodies treatments with or without nanowiring in TBI. Taken together our results for the first time show that HO-2 antibodies could induce marked neuroprotection in TBI and these effects are further potentiated by nanowiring of the HO-2 antibodies.

Arginine transport in the human pathogen *Leishmania* and its possible role in parasite-host interactions

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Arginine is an essential amino acid for the intracellular parasitic protozoan *Leishmania* but not for its host. Thus, maintaining cellular homeostasis of arginine is critical for parasite survival and virulence. Previously, we cloned and functionally characterized a high affinity arginine-specific transporter, LdAAP3, from *Leishmania donovani*. Here we characterized the relationship between arginine transport via LdAAP3 and amino acid availability. Exposing parasites to amino acids starvation decreased the cellular level of most amino acids including arginine, while the abundance of LdAAP3 mRNA and protein greatly increased. Consequently, arginine transport activity was up-regulated ~fivefold. We also found that genetic obliteration of the polyamine biosynthesis pathway, for which arginine is the sole precursor, caused a significant decrease in the rate of arginine transport. Cumulatively, we established that LdAAP3 expression and activity changed whenever the cellular level of arginine changed, and thus hypothesized that *L. donovani* promastigotes have a signaling pathway that senses cellular concentrations of arginine and subsequently activates a mechanism that regulates LdAAP3 expression and activity.

Although starvation for amino acids is an artificial condition examined in an in vitro system, it is likely that *Leishmnania* also experience starvation in vivo. Inside macrophage phagolysosomes the parasites proliferate as non-motile amastigotes that need to compete for host arginine, possibly by employing their starvation response mechanism. Therefore, we hypothesize that LdAAP3 plays a vital role in the parasites survival within its host.

Association study of hypoxic gene single nucleotide polymorphism with the susceptibility of acute mountain sickness

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Background and objective: Acute mountain sickness (AMS) is apotentially serious affliction to health in immigrants to Tibetan Plateau above 3,000 m. There are no effective treatment and prevention strategy of AMS now. Interindividual variation in acclimatization and adaptation to high altitude suggest that the probability of developing AMS depends on genetic and environmental factors. So we speculated that genetic factors may be associated with AMS susceptivity. The research aimed to explore the association between single nucleotide polymorphism (SNP) of hypoxic gene and AMS by comparing the difference of hypoxic gene SNP between AMS-susceptible and acclimatized individuals.