



DRDO

Press Release

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INHIBIT THE CALPAIN TO CLIMB THE MOUNTAIN

Scientists at Defence Institute of Physiology and Allied Sciences (DIPAS), DRDO have identified a novel mechanism for thrombosis induced by high altitude environment. The thrombosis developed at regions like Siachin Glaciers may lead to life threatening events such as pulmonary embolism, stroke, and limb amputation. Director, DIPAS, Dr. Shashi Bala Singh, describes this work as a key step towards understanding such disorders. The study by the Genomics lab, DIPAS, led by Dr. Zahid Ashraf using proteomic analysis of platelets and animal models elegantly demonstrated that enhanced activity of an enzyme 'calpain' significantly contributes to thrombosis under hypoxic conditions. Interestingly, the investigations on the soldiers who developed thrombosis while serving at extreme altitude also revealed an increased activity of calpain confirming the relevance of the novel preclinical findings for clinical applications. The findings have been published in current Feb 20, issue of medical weekly 'Blood', official journal of American Society of Hematology with editorial commentary. The novel findings could lead to development of therapeutics aimed at specifically preventing or treating thrombotic disorders induced at high altitude regions. The simple bioanalytic assay of 'calpain' could be developed for an early diagnosis test for such disorders.

Interestingly, Air Marshal DP Joshi, PVSM, AVSM, PHS, Director General Armed Forces Medical Services has commissioned a study on epidemiology of venous thrombotic disorders in lowlanders at high altitude. Maj Gen Velu Nair, AVSM, VSM**, Dean AFMC, Pune is supervising this ongoing multicentric study in collaboration with DIPAS. A cohort of more than 600 soldiers is being studied longitudinally during their induction and subsequent posting at extreme altitudes.

Reference: Tyagi T, Ahmad S, Gupta N, Sahu A, Ahmad Y, Nair V, Chatterjee T, Bajaj N, Sengupta S, Ganju L, Singh SB, Ashraf MZ. Altered expression of platelet proteins and calpain activity mediate hypoxia-induced prothrombotic phenotype. *Blood*. 2014;123(8):1250-1260.

<http://bloodjournal.hematologylibrary.org/content/123/8/1250>

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