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Skiing Injuries in Perspective

To the Editor:

Congratulations to Flores, Haileyesus, and Greenspan with regard to their excellent article on National Estimates of Outdoor Recreational Injuries Treated in Emergency Departments, United States, 2004–2005.[1] The article calls attention to recreational injuries, an important component in the need for injury control. However, when we first looked at their table 6, we thought ‘‘Where is skiing?’’ The omission of skiing in the table might leave the casual reader with the impression that skiing is not an important source of outdoor recreational injuries. The authors apparently excluded skiing injuries, as they were ‘‘concentrated in a few hospitals.’’ It is not surprising that there are more skiing injuries in hospitals near ski areas and that the hospitals selected in the Consumer Product Safety Commission’s sample do not provide a reasonable estimate of skiing injuries.

A major problem in measuring the importance of recreational injuries is the following: What is to be used as a denominator? The authors have used rates per 100 000 population, which gives an estimate of the societal importance of injuries resulting from each activity. Equally important is measuring the relative danger of persons exposed to the various activities. For skiing, the denominator is particularly difficult. Is it the number of people skiing? The days of skiing? Hours spent skiing? ‘‘Double black diamonds’’ as compared to hours on ‘‘bunny slopes’’? Although the number of ‘‘ski visits’’ to the northeast areas decreased from 14.7 million in 1986–1987 to 11.8 million in 2006–2007, the number of skiers still exceeds the number of snowboarders.[2]

Although China does not seem a likely place for skiing injuries, the problem of ski injuries in the United States may be a forewarning of greater problems in China. Surprisingly, the number of people in China who skied went from 300 000 in 2000 to 3 million in 2005.[3] China has great opportunities for ‘‘extreme skiing,’’[4] which is growing in popularity and should be of interest to the readers of *Wilderness and Environmental Medicine.* In conclusion, we call attention to an article on skiing injuries by William Haddon, Jr, ‘‘The father of injury, epidemiology and control,'' and colleagues, whose landmark 1962 paper described the frequency of specific injuries and variations with age, sex, and skiing experience.[5]

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Persistent Elevation of VEGF and Prostacyclin Following Poor Cardiopulmonary Adaptation to High Altitude

To the Editor:

Hypobaric hypoxemia is a potent stimulus for the release of erythropoietin and for some markers of angiogenesis, such as vascular endothelial growth factor (VEGF).[1] Vascular endothelial growth factor stimulates angiogenesis, and angiopoietin-2 acts with VEGF to stimulate angiogenesis in the myocardium.[2] Similarly, prostacyclin (PGI_2) is a member of the prostaglandin family of lipid mediators, which have potent vasodilator and antithrombotic activities.[3] Some limited data have been reported on the release of selected markers of angiogenesis, including VEGF in climbers exposed to high altitude with or without symptoms of acute mountain sickness (AMS). However, no study has explored the concomitant changes in VEGF, angiopoietin-2, and PGI_2, as well as selected markers of inflammation and oxidation, concomitantly in relationship with good vs poor adaptation to high altitude.

We investigated the changes in selected markers of angiogenesis, prostaglandin, and some markers of subclinical inflammation and oxidative stress 24 hours prior to departure for the Bolivian Altiplano and within 24 hours of return to sea level. The objective of this expedition was to summit Mount Sajama, the highest peak in Bolivia, at an altitude of 6522 m. Participants were exposed to an altitude between 3600 and 6522 m for 19 days.

The study population consisted of 5 participants (4 males/1 female) aged 30 to 64 years living chronically at sea level. The diagnosis of AMS was performed using the Lake Louise[4] and the Hackett[5] scoring systems at the highest point reached during the expedition.

From the 5 climbers, 2 participants developed moderately severe symptoms of AMS. One climber, aged 64 years, experienced mild headache, recurrent difficulty sleeping, profound fatigue, and dyspnea on minimal exertion at an altitude of 4800 m (Hackett score = 4; Lake Louise score = 6). This climber stopped his climb at 5300 m. One participant developed some fatigue and severe intractable headache at 5680 m and returned to base camp at 4800 m (Hackett score = 3; Lake Louise score = 4). Data are presented in the Table.

Table. Changes in plasma markers prior to departure and within 24 hours upon return to sea level*

	No AMS (n = 3)		*AMS-CP* (n = 1)		*AMS-CNS* (n = 1)	
	BSL	*Post*	*BSL*	*Post*	*BSL*	*Post*
VEGF ($pg \cdot mL^{-1}$)	204 ± 38	210 ± 43	222	553	259	244
Ang-2 ($pg \cdot mL^{-1}$)	1605 ± 601	1257 ± 280	1739	1539	1800	1750
6-Keto-$PGF_{1\alpha}$ ($pg \cdot mL^{-1}$)	37.7 ± 36.1	49.5 ± 40.0	118	311	85.4	62.0
IL-6 ($pg \cdot mL^{-1}$)	12.7 ± 15.9	12.2 ± 8.1	11.3	13.7	40.3	51.9
TBars ($pg \cdot mL^{-1}$)	10.8 ± 2.6	9.42 ± 3.70	6.26	13.7	10.0	11.2

*AMS indicates acute mountain sickness; CP, cardiopulmonary; CNS, central nervous system; BSL, baseline; Post, postexpedition; VEGF, vascular endothelial growth factor; Ang-2, angiopoietine-2; 6-Keto-$PGF_{1\alpha}$, 6-keto-prostoglandin F_{α}; IL-6, interleukin 6; and TBars, thiobarbituric acid-reactive substances.