Altitude Does Not Reduce Concussion Incidence: Letter to the Editor

Dear Editor:

I read with interest a recent article published by Smith and colleagues[8] in which the authors suggested that altitude reduces the incidence in concussion rates by 30% in high school students. Indeed, these results sound very novel and exciting, and it is not surprising they were featured in multiple mainstream media reports.[1,2,6] Due to this widespread media attention, I feel compelled to discuss some serious issues with the study, especially focusing upon statistical illiteracy and the physiology of altitude exposure.

The first major problem with the study is that it confuses the reader (and media) into thinking the results are clinically meaningful, and because of this, erroneous conclusions are met concerning their findings. This sort of statistical illiteracy (the inability to understand the meaning of numbers) in the population has been documented elsewhere,[4] and the interpretation of this study is no different. The authors show a 30% reduction in the relative risk in the incidence of total reported concussions at altitude in high school athletes paying a variety of sports compared to that at sea level.[8] However, what is critical to understand is that the relative risk reduction they report is a gross exaggeration of the findings and that it is the absolute risk reduction that should be meaningful to readers and reported.[4] The overall (absolute) risk of concussions in high school athletes is already very low to being with, as Smith et al[8] report: 0.3 incidents per 1000 athlete-exposures or 0.03% chance of a concussive event per exposure. However, what needs to be understood is that a 30% reduction in the relative risk does not change the already low absolute risk significantly. The absolute risk of concussions at the authors' definition of altitude ($\geq$600 feet) is reduced to 0.25 incidents per 1000 exposures (see Table 1 of Smith et al[8]). This means that the absolute risk reduction for concussive events at altitude is 0.08 incidents per 1000 exposures, or 8 concussive events for every 100,000 exposures ([0.33 per 1000 exposures] – [0.25 per 1000 exposures] = 0.08 per 1000, or 8 per 100,000 events). In simple terms, there would be 8 fewer concussive events in these high school athletes for every 100,000 exposures if they were playing at an altitude of about 600 feet compared to sea level. While statistically significant, this is hardly clinically meaningful. This goes the same for the incidence of concussions in high school football. The absolute risk reduction by playing at "altitude" is 0.016%, or 16 events per 100,000 exposures (see Table 2 of Smith et al[8]).

The second major problem with this study is that their definition of altitude is 600 feet with an interquartile range of 200 to 935 feet. This is a rather arbitrary cut-off point, which is not based on physiological response to altitude or atmospheric science but rather to an artifact of the database used. Using similar logic, one could examine a database of morbidly obese patients and arbitrarily classify those below the median weight as "thin individuals." The authors do indeed describe many of the physiological effects of altitude exposure throughout the paper, but these effects do not occur at these low altitudes, and therefore their references are completely inappropriate for this particular study. The difference between the inspired partial pressure of oxygen (P_IO_2) at an altitude of 600 to 935 feet compared to sea level is only $\sim$6 mm Hg. Thus, at 0 to 1000 feet of altitude, the differences in P_IO_2 is negligible compared to sea level. While the authors describe changes in air pressure with slight changes in altitude (eg, a baseball thrown in Denver, Colorado at $\sim$5000 feet), these changes do not significantly influence arterial oxyhemoglobin saturation, and therefore hypoxemia simply does not occur at most of the altitudes described within the study. Maximal oxygen uptake only begins to decline at altitudes of $\sim$2000 feet, but performance declines are noticed only at $\sim$3300 feet.[3] Anaerobic events lasting <2 minutes are minimally affected up to $\sim$7000 feet.[3] So the statistical analyses should have compared sea level incidence of concussions to the incidence of concussions at <3300 feet (at a minimum), not at 600 to 935 feet.

The third major problem with the study is their supposition of the mechanism for the "reduction" in the incidence of concussions at 600 feet of altitude. They hypothesize that there is increased intracranial stiffness at that altitude, decreasing the "sloshing effect" of the brain against the cranium and thus reducing the chance of concussive injury. The authors cite papers from the *Lancet* and *Journal of the American Medical Association* describing the pathophysiology behind acute mountain sickness, including the life-threatening condition of cerebral edema, to support their contention that the brain experiences some swelling at altitude. However, these conditions typically take place at altitudes around 10,000 feet,[7] and they do not occur in all individuals. Thus it is preposterous to contend that at a mere 600 to 1000 feet, there is sufficient swelling to induce a protective effect. Likewise, the authors cite research describing increased cerebral blood flow at high altitude,[9] however, these studies examined individuals exposed to altitudes >17,000 feet. Further, the individuals describe the chronic adaptations that occur at altitude, including increased erythropoietin (EPO) production leading to polycythemia, as potential mechanisms that can contribute to these effects, yet numerous studies have shown that expansion of red blood cell volume and increases in total hemoglobin mass

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typically requires altitudes ≥7000 feet for prolonged periods (ie, 20 days)[5]. The authors continue to describe the relationship between temperature and pressure with changing altitude, but surely it is possible for athletes to experience cold and dry days at sea level as well, and consistent differences in ambient conditions 1000 feet above sea level seem rather unlikely when the large geographic region of the study is considered. Perhaps, though, another interpretation of this finding is that altitude reduces effort (ie, speed and movement are reduced, thus impact forces are reduced), and the reduction of effort reduces concussive events. Of course, this is conjecture and in my view unlikely, as the physiological effects of altitude are not noticeable until a much higher elevation (≥3300 feet).

In conclusion, the paper by Smith and colleagues[8] is filled with overzealous interpretations, wrong conclusions, and a blatant misrepresentation of the literature, such that true high-altitude physiology is inappropriately extrapolated to low-to-mild altitude for the convenience of supporting the authors' hypothesis.

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