Also, 1 participant was diagnosed with high altitude pulmonary edema (HAPE) at 2900 m on day 2 and was treated with dexamethasone. We were interested to learn if this patient was experiencing concomitant AMS/high altitude cerebral edema. As per Wilderness Medical Society consensus guidelines, dexamethasone is indicated for prevention of HAPE in a susceptible subject and prevention/treatment of AMS and high altitude cerebral edema. Recommendations on its use for treatment of HAPE has been graded weak with low or very low quality evidence.³

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In Reply to Drs Sikri, Bhattachar, and Thapa

To the Editor:

We thank Drs Sikri, Bhattachar, and Thapa¹ for their interest in our recent article exploring the risk determinants of acute mountain sickness (AMS) on Mount Kilimanjaro.² They raise a number of valid points that we agree with and aim to clarify further.

Our cohort demonstrated a lower incidence of AMS than those in 2 previous studies on Kilimanjaro using shorter ascent profiles. The populations studied in the 3

research articles are quite heterogeneous, most notably in the use of prophylactic acetazolamide. Our cohort had the highest use of acetazolamide at 88%. It is therefore not possible to directly compare the outcomes of these studies, as we discussed in our article. Interestingly, despite an ascent over 6 days, and high utility of acetazolamide, we still reported an incidence of AMS of 52.6% (using Lake Louise Score [LLS] ≥ 3), which would be considered high on most other tourist treks.

We did not include a subgroup analysis of those who used acetazolamide in our original article in the interest of brevity. Additionally, the sizes of the subgroups were too small and low powered to permit a meaningful comparison. In our cohort, of those who took acetazolamide, only 1.3% started the regimen the day before ascent, as recommended for prophylaxis.³ On the first day of ascent, 33.8% started acetazolamide; 64.9% started after the first day while already at altitude. All users continued to take the medication until after summiting. As Sikri et al highlighted, prophylactic acetazolamide is most efficacious when started the day before ascent.³ In that respect, the majority of our group was using acetazolamide suboptimally, and this could be reflected in the AMS incidence that, despite being lower than that in other studies, remains relatively high. We would also highlight that although acetazolamide generally is considered to be effective,⁴ there have been no conclusive results from its use on Mount Kilimanjaro specifically that demonstrate effective prophylaxis. Any protective effect is therefore hard to quantify. To that extent, it is possible that the reduced AMS incidence in our study is more related to the slower ascent profile than to any medication use.

Sikri et al also raise the logistic issue of recording LLS at the summit, which is a challenge in all field-based research in this area. The reality is that it is impractical for the expedition doctor to wait at the summit while all individuals complete their ascents. In our experience, the climbing group is spread out during the final stages of the ascent so that individuals summit in a staggered fashion over a time frame of a couple of hours or more. We acknowledged in our article that recording the LLS once the group had returned to base camp is a limitation. However, we also consider that the subjective nature of the LLS may mean that recording it while in a relatively elated emotional state, immediately after successfully reaching the summit, could potentially be unreliable. We note that other studies based on Mount Kilimanjaro have used the same approach.^{5,6}

The ascent profile was illustrated in our original article, where it was compared to shorter Kilimanjaro ascent profiles. Participants walked at a slow pace from the first day of the trek, as dictated by the head guide.

Trekkers carried a light day-pack only; other equipment was carried by porters. No physical activity other than the trek and movement around the camps was undertaken. As Sikri et al describe, the rate of ascent and actual altitude reached are the primary risk factors for development of AMS.³ In our study, the rate of ascent was highest on summit night, during which time participants ascended 1165 m over a period of 8 to 10 hours. This is far higher than the recommended ascent rate of no more than 500 m/day,³ although this does refer to sleeping altitude specifically. On summit night, ascent was immediately followed by descent to base camp over ~2 hours, followed by a further descent of ~1000 m over the subsequent 6 to 8 hours. The fast rate of ascent on Kilimanjaro is likely the major factor in the high rate of AMS described, both in our study and previously. We maintain that a slower ascent profile is likely to be safer.

Sikri et al specifically inquire about the use of dexamethasone in a trekker reported in our study, who was treated for high altitude pulmonary edema. In reference to that case, the climber was a 42-year-old woman with no medical comorbidities. She was taking prophylactic acetazolamide, having started it on the first day of the trek. She presented with worsening dyspnea at a relatively low altitude of 2900 m, which progressed over a period of a few hours. The main clinical feature was pulmonary edema, with basal crepitations on auscultation. She also experienced worsening headache throughout this time and was therefore treated empirically with dexamethasone, despite not having any other clinical features of high altitude cerebral edema. As has been described by others, climbers should be treated for both high altitude pulmonary edema and high altitude cerebral edema if there is any doubt in the diagnosis.⁷

Once again, we appreciate the interest in our article and hope to have provided some clarification.

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