‘...for we must remember that even the highest of mountains is capable of severity, a severity so awful and so fatal that the wiser sort of men do well to think and tremble even on the threshold of their high adventure.’


Introduction

In 1950, Maurice Herzog and Louis Lachenal became the first climbers to reach the summit of an 8000 m peak (Annapurna, 8091 m). In the half century since that pioneering climb, mountaineers have increasingly sought to climb the fourteen ‘8K peaks’ of the Himalayas and Karakoram, with remarkable success; they have made 5085 ascents of those peaks up to the year 2000. While seeking adventure on those great peaks, mountaineers are inevitably exposed to hypoxia, cold and dehydration as well as to the physical hazards of climbing. Those few mountaineers who successfully summit an 8K peak are likely to be at or near their physiological limits and probably confront an elevated probability of dying during their descent. We will briefly review some of the physiological challenges climbers face at extreme elevation and then compare success rates and death rates on mountains of different heights (Rainer, Foraker, Denali, K2, Everest). Success rates decline with summit height, but overall death rates and death rates during descent from the summit increase with summit height. Although these patterns are based on non-experimental and uncontrolled data, our findings are consistent with the hypothesis that increasing altitude is associated with decreased success and with increased risk of death.

We can now begin to address such questions. The necessary historical data derive from extensive interviews obtained over many years by E. Hawley and by X. Eguskitza supplemented by published records in the mountaineering literature (e.g. American Alpine Journal, Alpine Journal, Himalayan Journal and scores of books). To search for patterns in these data, we adopt analytical techniques that were developed for evolutionary studies of Natural Selection on organisms (Endler, 1986; Schluter and Nychka, 1994). For example, we attempt to determine whether rates of success or of death correlate with factors such as mountain height, use of supplemental oxygen or team size. Our analyses are still preliminary but nevertheless provocative.

We begin by reviewing some of the physical and physiological challenges associated with high-altitude mountaineering. Then, we will use available mountaineering data to test two hypotheses: (i) that the success rate in reaching the summit is inversely related to a mountain’s height and (ii) that death rates are directly related to mountain height.

Summary

Limits to human performance: elevated risks on high mountains

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In the ‘Death Zone’

Heights greater than 8000 m are well above the highest human habitation (5950 m; West, 1986a) and are inhospitable for sustained human life. Accordingly, such extreme heights are sometimes referred to as the ‘Death Zone’ (Krakauer, 1997). The primary limiting factor is barometric pressure, which declines exponentially with altitude (Fig. 1; West, 1996). Because oxygen makes up 21% of air at any altitude, the partial pressure and hence availability of oxygen decrease with altitude.

At Flagstaff, Arizona (2510 m), the barometric pressure is approximately 25% lower than at sea level (Fig. 1). To someone coming from a coastal city, the physiological impact of even that modest decrease is immediately evident. As one goes higher, physiology is increasingly stressed. On Mount Everest, a mountaineer has to tolerate extremely low barometric pressures. At Everest base camp (approximately 5310 m), the barometric pressure is half that at sea level; at the summit itself (8850 m), the barometric pressure is one-third that at sea level (West, 1999; Fig. 1). Because the partial pressure of oxygen is similarly low, arterial hemoglobin is poorly saturated with oxygen (Peacock and Jones, 1997). The resulting tissue hypoxia not only greatly restricts a climber’s ability to move (Edwards, 1936; Pugh et al., 1964; West et al., 1983; West, 1986b; West, 2000) but also induces serious physiological, medical, sensory and neurobehavioral problems (Hornbein et al., 1989; Houston, 1998; Hultgren, 1997; Richalet, 1999).

Low temperatures and strong winds compound physiological stresses at extreme altitude (although these are sometimes ameliorated by intense radiation loads). Some remarkable temperature data have recently become available for Everest (M. Hawley, personal communication). For example, temperatures measured on the South Col (approximately 960 m below the summit) during the spring 1999 climbing season averaged –28°C (Fig. 2). Winter 8K climbers thus face truly challenging conditions: colder temperatures, near-jet-stream winds (Peixoto and Oort, 1992, p. 154), even lower barometric pressure (West, 1996) and short days (approximately 10.5 h in December versus 13.5 h in May). Such conditions should be brutal enough to discourage most people: not surprisingly, very few climbers attempt 8K peaks in winter. Indeed, only 5% of all climbers have attempted an ascent of Everest in winter (total climbers 5268; 1953 up to spring 2000; R. Salisbury and E. Hawley, personal communication). Remarkably, one climber (Ang Rita, 22 December 1987) successfully reached the summit in winter without using supplemental oxygen.

Success and death versus altitude

Physiological stress from hypoxia, cold, wind and dehydration increases with altitude, and physiological performance inevitably drops. Consequently, the risk of exhaustion, a serious medical problem, a fall or mental error might well increase the higher a climber goes (Pollard and Clarke, 1988; Hornbein et al., 1989; Peacock and Jones, 1997). Consequently, we predict that the probability of successfully reaching the summit will be inversely related to the altitude of that peak and that the probability of dying will increase with altitude, all else being equal.

Testing these hypotheses is easier said than done, in part because the probabilities of success and of death must be influenced by many other factors, not just altitude. For
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example, peaks (and even different routes on a given peak) obviously differ in difficulty, danger and even with other conditions (see below). A full analysis of the probabilities of success and death should consider the influence of as many such factors as possible; but of necessity here, we begin with a single-factor (altitude) approach.

First, do death rates of trekkers increase with altitude? Indeed, death rate increases with altitude (Burtscher et al., 1995): 2.3 deaths per 10^6 days of exposure for England and Wales (Avery et al., 1990), 5.7 for Austria (Burtscher et al., 1995), compared with 10.6 for Nepal (Shlim and Gallie, 1992; Shlim and Houston, 1989). Even so, as Burtscher et al. (Burtscher et al., 1995) cautioned, trekking death rates will be influenced by many factors other than by altitude (medical services are less accessible in Nepal, for instance), so these data are suggestive at best. Nevertheless, they are consistent with the hypothesis that risk increases with altitude.

Next, does success rate decrease with the altitude of a peak and does death rate increase? Success and death rates have been compiled for very few peaks so far, but we have been able to obtain reasonably complete data for five peaks [Table 1: the two highest peaks in the world (K2, Everest) versus three lower-altitude peaks (Rainier, Foraker and Denali, all in North America)]. These five mountains all involve major altitudinal gains from base camp to summit (Table 1), but differ strikingly in success (i.e. reaching the summit) and mortality (Table 1).

The probability of success of individual climbers drops suggestively with altitude (Table 1; \( r = -0.70, P = 0.09 \)), and success rate on the two 8K peaks is significantly lower than on the three higher peaks (Wilcoxon–Mann–Whitney test, \( P = 0.04 \)). Nevertheless, success rate can be low even on a relatively low-altitude peak such as Foraker (Table 1), reflecting the inherent difficulty of this mountain irrespective of its lower altitude.

Overall death rate increases with altitude (Table 1; \( r = 0.84, P = 0.04 \)) and is significantly higher for the two 8K peaks (\( P = 0.04 \)). Similarly, death rate during descent from the summit increases with a summit’s altitude (Table 1; \( r = 0.83, P = 0.04 \)) and is higher for the two 8K peaks (\( P = 0.04 \)). Note that the

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Table 1. Summary of tentative mountaineering statistics

<table>
<thead>
<tr>
<th>Peak (census period)</th>
<th>Summit (m)</th>
<th>Elevation gain (m)</th>
<th>Permit fee (US$)</th>
<th>Success rate (%)</th>
<th>Overall death rate (%)</th>
<th>Death rate for descent from summit (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rainier (1987–1996)</td>
<td>4392</td>
<td>2740</td>
<td>15</td>
<td>52.9</td>
<td>0.02</td>
<td>&lt;0.04</td>
</tr>
<tr>
<td>Foraker (1990–1900)</td>
<td>5306</td>
<td>3780</td>
<td>150</td>
<td>17.8</td>
<td>0.70</td>
<td>0</td>
</tr>
<tr>
<td>Denali (1990–1900)</td>
<td>6193</td>
<td>4060</td>
<td>150</td>
<td>48.4</td>
<td>0.24</td>
<td>0.17</td>
</tr>
<tr>
<td>K2 (1954–1994)</td>
<td>8616(^*$)</td>
<td>3411</td>
<td>1714</td>
<td>&lt;13</td>
<td>Approx. 4.0</td>
<td>18.8(^*)</td>
</tr>
<tr>
<td>Everest (1953–Spring 2000)</td>
<td>8850</td>
<td>3538</td>
<td>10000</td>
<td>16.5(^\d)</td>
<td>2.0(^\d)</td>
<td>8.3(^*)</td>
</tr>
</tbody>
</table>

On the two 8000 m peaks, success rates are relatively low and death rates are relatively high. Data sources: Rainier (Christensen and Lacsina, 1999); Denali and Foraker (National Park Service); K2 (Eguskitza, 1996); Everest (E. Hawley and R. Salisbury, personal communication; Huey and Eguskitza, 2000).

\(^*$\)Death rates descending from the summit of K2 or of Everest are for mountaineers who did not use supplemental oxygen and are for the period 1978–1999 (see Huey and Eguskitza, 2000). Climbers using supplemental oxygen had lower death rates (0 % and 3.0 %, respectively, see Huey and Eguskitza, 2000). In the analyses of the impact of summit altitude, we excluded data on O₂-aided ascents because the use of supplemental O₂ lowers the ‘physiological altitude’ of a peak (Messner, 1979). Rates for K2 are based on incomplete data (see Eguskitza, 1996).

\(^\d\)Success and overall death rates on Everest exclude high-altitude porters, sirdars and commercial guides (R. Salisbury, personal communication).

\(^\d\)Altitude for K2 from Poretti (Poretti, 1999).
probability of death during descent from either of the two highest Himalayan peaks is more than an order of magnitude higher than that from Rainier, Foraker or Denali (Table 1). Moreover, death rates during descent from the summits of all other 8K Himalayan peaks [range 0.5% (Cho Oyu) to 7.4% (Annapurna), data from Eguskitza, in Sale and Cleare, 1999] are also very high relative to the three lower peaks in Table 1, further reinforcing the evidence in Table 1 that big peaks are more dangerous. [Note: the permit fee for climbing ‘scaler’ positively with a peak’s altitude (Table 1), and this pattern holds even within the Himalayas (Huey, 2001). Obviously climbers are willing to pay a premium for attempting big peaks, despite low success and high risk.]

These comparisons are simplistic and based on only a few peaks. Moreover, these five mountains differ in many ways (not just altitude) that might influence or dominate success and death rates. For example, helicopter rescue, which could reduce death rates, is an option on the three North American peaks (weather permitting), but only at ‘low altitude’ on Everest and not at all on K2. Moreover, the experience and skill of the climbers that attempt these peaks are undoubtedly far from homogeneous (see below). Even so, the available data support the expectation that climbing high peaks is in a different league from climbing low ones.

**Concluding caveats**

Before we started these analyses of mountaineering outcomes, we questioned whether meaningful patterns could in fact be discerned from available data. After all, conclusions concerning causality might be confounded by several issues.

First, mountaineering data will be intrinsically noisy. Weather and snow conditions can change rapidly, as can a mountaineer’s physical state, adding variability to any real pattern.

Second, such analyses are inherently retrospective, and so inferring causation is additionally risky (Hill, 1965). The standard way to evaluate causation is, of course, to do an experiment. That is not an option here: imagine how K2 mountaineers would respond to being randomly assigned into groups ‘using’ versus ‘not using’ supplemental oxygen! Accordingly, we must interpret any observed pattern with caution (see Huey and Eguskitza, 2000) using standard epidemiological criteria (Hill, 1965; Susser, 1991) to evaluate causality.

Third, we must assume that climbers on different mountains are comparable in experience, skill and willingness to accept risk, but this is certainly not the case (climbers on Rainier are less skilled and experienced than are climbers on K2). Climbers also differ in ability to acclimatize to altitude, concurrent illness and tolerance of physiological adversity. Such individual differences, which unfortunately are difficult to ascertain retrospectively, can potentially confound or mask comparisons across peaks (see Huey and Eguskitza, 2000). In the present examples, however, the induced biases should be in the opposite direction to the observed patterns: thus, for example, climbers on 8K peaks have relatively low success rates and relatively high death rates despite their greater skill and experience.

Finally, the most readily accessible data are for individual climbers (e.g. Table 1); yet, because climbers are usually in groups (and sometimes die in groups), data for individuals are not entirely independent, as assumed by traditional statistical methods. An alternative is to use a climbing team as the ‘unit’ of analysis (see Huey and Eguskitza, 2000). Unfortunately, such team-based data are not yet available for most peaks.

Despite these and other concerns, our work so far suggests that patterns can indeed be detected. Mountaineering data do appear to give strong signals (Huey and Eguskitza, 2000; herein), despite their inherent noisiness. Because the observed patterns are consistent with physiologically based expectations, we gain some reassurance that cause and effect may well be involved. Nevertheless, more rigorous studies will be necessary to validate causality. At the present time, a cautious skepticism is appropriate.

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**References**


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