DISCUSSION AND REPLY

Reply

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Our hypothesis of terminal-Permian vertebrate mass extinction by pulmonary edema (Retallack et al., 2003) rested on observations of victims (1) and survivors (2) of mass murder, and a plausible lethal weapon (3), as follows: (1) mass mortality of vertebrates at the Permian-Triassic boundary; (2) improved respiratory capacity of earliest-Triassic survivors (development of secondary bony palate, shortened internal nares, high dorsal vertebral spines, thickened ribs, barrel chests, shortened limbs, reduced lumbar ribs); and (3) evidence of dramatically lowered earliest Triassic atmospheric oxygen levels (bertherine in paleosols, lowered stomatal index of seed ferns from increased carbon dioxide, declining carbon isotope values unique for methane outburst). Now Milo Engoren questions the lethality and nature of the weapon with a variety of back-of-the-envelope calculations. The main problems with his estimates are that they do not take into account analytical uncertainty or interactions between causes or dose exposure times, as outlined below. What is known about acclimatization in humans, birds, and frogs supports our hypothesis, and I thank Engoren for the stimulus to look into this further.

Analytical uncertainties do not rule out concentrations and rates proposed as lethal by Engoren. For example, the rate of the global atmospheric crisis can be tracked from the decline of carbon isotope values, which has been dated at ~618 k.y. from radiometry (Bowring et al., 1998), 600 k.y. by cyclostatigraphy (Rampino et al., 2000), and 10,000 yr from varve counting and coal accumulation rates (Retallack and Jahren, work in progress). Successive outbursts of methane over thousands of years best explain multiple isotopic minima (Morante and Herbert, 1994). The amounts of methane and carbon dioxide in the earliest Triassic greenhouse are very uncertain. The magnitude of the methane outburst can be estimated from the magnitude of the isotope excursion at the Permian-Triassic boundary. My compilation of 22 marine and non-marine sections shows an average isotope (δ13C) excursion of ~7.6 ± 4.7% and range of ~22.2 to ~2.9% with larger excursions at higher latitudes (Krull et al., 2000). The isotopic values (δ13C) of methanogenic carbon range from ~110 to ~35%, and ~65% is a conservative amount used for calculating terminal-Permian methane emissions of 4200 Gt (Berner, 2002). Another order of magnitude could be estimated by assuming ~35% and an isotopic excursion at the upper edge of observed range. The upper limit for methane pollution of the atmosphere by clathrate release is probably close to the 11,000 Gt currently stored in global reservoirs (Krull et al., 2000). A magnitude of the earliest Triassic CO2 greenhouse of 7876 ± 5043 ppmV can be estimated from a stomatal index of seed fern leaves near Sydney, Australia (Retallack, 2002). The new, physically based, transfer function giving CO2 partial pressure from the stomatal index was fit to data from modern (280 ppmV) to twice modern (580 ppmV) greenhouse results (Wynn, 2003). Extrapolation from these results to many thousands of ppmV CO2 is fraught with uncertainties, which could embrace much higher values. Thus, atmospheric hypoxia could well have been fast and profound enough to induce pulmonary edema and to kill most, but not all, vertebrates. Refining these estimates is an important task for the future.

Engoren’s estimates can also be accommodated by interactions between causes revealed by models for the terminal Permian atmospheric crisis, such as those of de Wit et al. (2002), Berner (2002), Ryskin (2003), and Kidder and Worsley (2004). Ryskin’s (2003) model is the boldest in proposing atmospheric concentrations of 5%–15% methane, presumably with atmospheric mass increase. Berner’s (2002) model is more conservative and tuned to geological evidence. It maintains atmospheric mass balance, releases methane over 20,000 yr, and reaches a low of 12% atmospheric O2 only after methane outbursts are supplemented by wetland die-back and volcanic emission. Berner’s model suggests that a variety of causes, including starvation, global warming, and hypoxia, contributed to the extinction. More sophisticated models will be needed.

Engoren raises the issue of CO poisoning. Concentrations of CO rise by 0.03 ppm to reach 0.14 ppm in the northern summer in sympathy with a summer rise of CH4 by 0.03 ppm to 1.8 ppm, suggesting efficient production of CO by the reaction of CH4 with OH, which in turn comes largely from reactions of water vapor and ozone (Warneck, 2000). Atmospheric levels of CO above 1500 ppmV are rapidly fatal to humans, with collapse induced by concentrations above 800 ppmV, and a decided headache above 220 ppmV (Raub, 1999). Most models for the Permian-Triassic (de Wit et al., 2001; Berner, 2002; Kidder and Worsley, 2004) postulate increases in CH4 and CO2 by a factor of 10–100. Thus, increases in CO by factors of 1,000–10,000 needed for lethal effect seem unlikely, unless Ryskin’s (2003) suggestion of 5%–15% methane and an atmosphere above 1.5% CO2 is correct. Furthermore, decreasing oxygenation would lead to decreasing CO2 (Berner, 2002) and thus decreasing production of CO. Nevertheless, CO poisoning is also a hazard of mountaineering exacerbated by altitude (McGrath, 2000), and its role in terminal Permian extinctions deserves further attention.

Finally, Engoren suggests that acclimatization to a new atmosphere over days to months would have enabled most reptiles to survive the Permian-Triassic life crisis. Duration of gas dosages is critical in environmental toxicology. As a general rule for assessing fire fatalities (Parson, 2000), humans are incapacitated after an hour of exposure to doses of CO in excess of 800 ppmV, CO2 greater than 4%, and O2 levels lower than 14%, but milder doses can lead to unconsciousness over many hours, and stronger doses are tolerated for times less than an hour (Fig. 1). Living monotremes (Nicol and Andersen, 2003), turtles (Frische et al., 2000), and frogs (Navas, 1996; Hou and Huang, 1999) can tolerate a half hour of as little as 5% O2 when hibernating, but this induces distress and hyperventilation. Terminal Permian hypoxia may have developed and been sustained for several thousand years (Berner, 2002), like a very slow climb to altitude. Humans could survive Berner’s modeled atmospheric crisis by acclimatization, which is well known among mountaineers, but not without limits, and it would not be possible for everyone (Hultgren, 1997). Acclimatization also is possible in Himalayan bar-headed geese (Anser indicus) and Canada geese (Branta canadensis) because

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Figure 1. Time to incapacitation of humans under varied levels of ambient CO, CO2, and O2 (from equations of Purser, 2000).

hypoxia induces increased capillary density in these high-flying species (Snyder et al., 1984). However, domestic cattle grazing in the high pastures of the Colorado Rockies aclimatize poorly and commonly require relocation due to edema ("brisket disease"); Reeves and Grover, 2001). Frogs have no significant capacity for hypoxic aclimatization, as demonstrated by Hou and Huang (1999) for a single species of toad (Bufo bankorensis) at elevations of 0–3000 m in Taiwan, and by Navas (1996) for several species of frogs at 2900–3500 m in the Colombian Andes compared with sea level congeneric in Panama. Thus, there is a range of dose and time tolerance to CO, CO2, and O2 among humans and other vertebrates, but aclimatization has only been demonstrated in birds and humans, not in amphibians more closely related to Permain vertebrates. Acclimatization to altitude may have been yet another respiratory adaptation selected during vertebrate evolution by atmospheric oxygen crises like those of the terminal Permian.

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