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Paleophysiology and end-Permian mass extinction

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Abstract

Physiological research aimed at understanding current global change provides a basis for evaluating selective survivorship associated with Permo-Triassic mass extinction. Comparative physiology links paleontological and paleoenvironmental observations, supporting the hypothesis that an end-Permian trigger, most likely Siberian Trap volcanism, touched off a set of physically-linked perturbations that acted synergistically to disrupt the metabolisms of latest Permian organisms. Global warming, anoxia, and toxic sulfide probably all contributed to end-Permian mass mortality, but hypercapnia (physiological effects of elevated P_{CO2}) best accounts for the *selective* survival of marine invertebrates. Paleophysiological perspectives further suggest that persistent or recurring hypercapnia/global warmth also played a principal role in delayed Triassic recovery. More generally, physiology provides an important way of paleobiological knowing in the age of Earth system science.

Keywords: Permian; Triassic; mass extinction; physiology; paleontology

1. Introduction

Paleontologists have traditionally focused on morphology and diversity because these are the two aspects of ancient life most obviously recorded by fossils. In recent years, however, it has become increasingly clear that evolutionary trajectories reflect the physical dynamics of the Earth system as much as they do genetic innovation. Particularly during mass extinction, catastrophic environmental change has impacted both the diversity and ecological structure of marine and terrestrial biotas.

Physiology provides the proximal interface between organisms and their environment. Thus, physiological inferences gleaned directly from fossils or from their living relatives can illuminate the causes and consequences of major extinctions and other events in the history of life. In this paper, we explore the relationships among physiology, evolutionary history, and environmental catastrophe through the example of end-Permian mass extinction and its aftermath. We argue that extinction resulted from the synergistic effects of several

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environmentally-linked stresses on organismic physiology — with hypercapnia (the direct physiological consequences of increased P_{CO2}) imparting the principal selectivity observed in the Permian–Triassic (P–Tr) fossil record.

2. Paleophysiology: what can we know?

Not all aspects of organismic biology can be captured in the fossil record [1]. We cannot, for example, determine the precise basal metabolic rate of an Ordovician trilobite or the temperature optimum for a Devonian brachiopod. Nonetheless, many basic physiological attributes can be inferred from fossils because they are associated with preservable morphological structures and conserved within higher taxa. Thus, in marine invertebrates, gas exchange may be mediated by respiratory elaborations such as gills, whose presence or absence can generally be inferred for skeletal fossils of known systematic relationships.

Other physiological attributes can be known in a comparative sense, even if absolute measurements are impossible. We might not know the basal metabolic rate of a clam preserved in Jurassic rocks, but we can have confidence that its basal metabolism exceeded that of brachiopods in the same bed. And we can reason from physiological first principles that the clam's metabolic rate scaled with temperature roughly according to the Q_{10} law (rate increases exponentially with increasing temperature, generally doubling with every 10 °C increase [2]).

Paleobotanists have been differentially attuned to paleophysiology because plants are strongly biophysical entities in which important aspects of physiological function relate directly to anatomy and morphology. Fossilized leaves provide proxies for paleoclimate [3] and preserved stomatal distribution on these surfaces reflects ancient P_{CO2}, as well as the capacity of the vascular system for water transport [4]. Moreover, in combination, vascular anatomy and microscale organic geochemistry permit quantitative estimates of water conductance in fossil plants [5], facilitating integration of leaf and stem function in attempts to understand plant responses to ancient environmental change. Marine microplankton have also received increasing physiological scrutiny from geochemists striving to exploit (and not simply avoid) "vital effects" in paleoceanographic reconstruction [6–8].

Marine invertebrates may be the fossils for which paleophysiology is least developed but most promising. Fossil invertebrates chronicle evolution through more than 500 million years of Earth history, and the fates of faunas are commonly interpreted in the context of changing ocean circulation and chemistry [9]. In particular, mass extinctions have long been recognized as key biological events, yet biology has played a distinctly second fiddle to geological and geochemical efforts to evaluate these catastrophes. What more can we learn when fossils bear paleophysiological witness to a great extinction?

3. End-Permian extinction: trigger and kill mechanisms

The event that ended the Paleozoic Era is generally regarded as the most severe of all recorded mass extinctions [10]. Estimates of proportional diversity loss depend on the metric and time frame adopted, but compilations by Sepkoski [11,12] indicate that some 54% of latest Permian marine families, 68% of genera, and up to 92% of species did not survive into the Triassic. A major reorganization of marine ecosystems ensued [13,14]. Land plants [15] and animals [16] were affected as well, requiring extinction mechanisms that impacted terrestrial as well as marine environments, albeit in apparently distinct ways.

Fig. 1 illustrates marine animal diversity through the Late Paleozoic and first half of the Triassic Period. Late Permian diversity collapse contrasts markedly with its relative stability during the preceding 85 million years; globally, marine genus diversity dropped by about 76% from a Permian plateau of near 1100 to just 270 survivors that crossed the erathem boundary. Available radiometric dates suggest that the era-ending extinction was geologically rapid, occurring over less (and perhaps much less) than the *ca.* 0.5 million year precision of U–Pb dating [17–19]. Recovery, however, was prolonged, accelerating only some 4–5 million years after the extinction [see below].

In evaluating proposed explanations for end-Permian mass extinction, we need to draw a clear distinction between kill and trigger mechanisms. A kill mechanism is the physiologically disruptive process that causes death, whereas a trigger mechanism is the critical disturbance that brings one or more kill mechanisms into play. In general [but see [20,21]], Earth scientists have sought to identify trigger mechanisms for end-Permian extinction. Candidate triggers, in turn, can be associated with one or more kill mechanisms. Paleophysiology comes into play because each kill mechanism predicts a physiology-dependent pattern of extinction and survival, providing a test for geophysical hypotheses. A mechanistic understanding of mass extinction will only be attained when trigger mechanisms inferred from geological data become

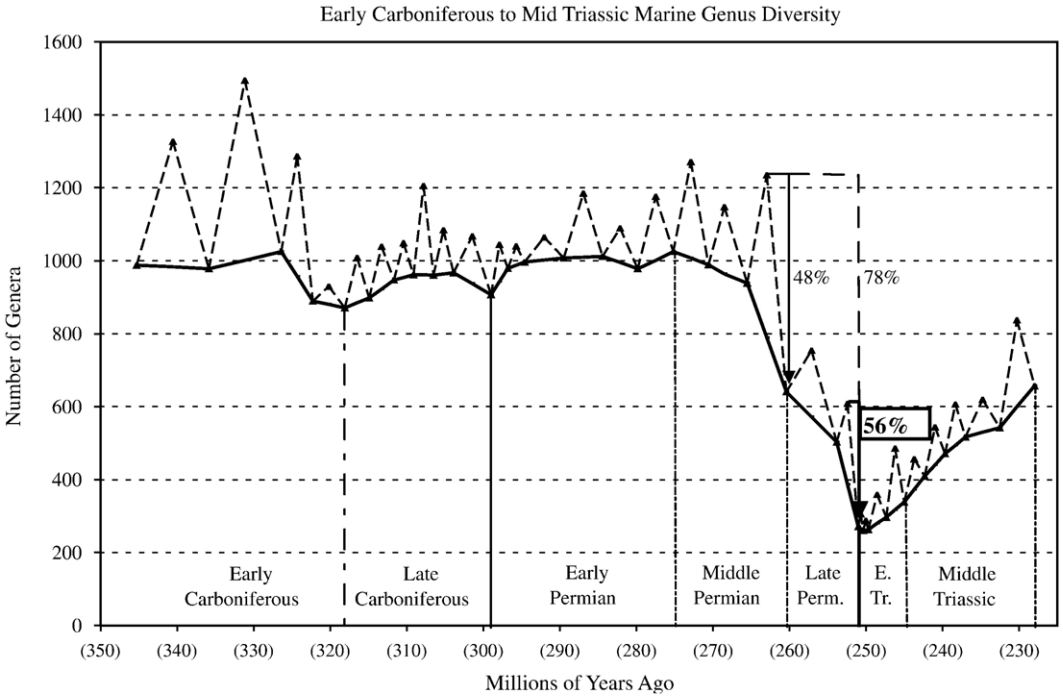

Fig. 1. Number of marine animal genera in stages and substages from the Visean (Mississippian) through the Ladinian (Middle Triassic). The display illustrates all available data compiled in [12]. Solid dots (connected by the heavy solid line) are the number of genera ranging from one stage into the next. The heavy solid line is the minimum standing diversity integrated between stage boundaries. Open dots at peaks along the dashed line are the total number of genera recorded in each time interval. The dashed line, thus, indicates the range of faunal turnover in each interval — positive slopes record the number of originations in each time interval, negative slopes the number of extinctions in each time interval. The number of genera ranging from one interval to the next is an approximation of standing diversity. Total diversities for time intervals are probably not measures of standing diversity because, if diversity were to equal the total number of genera that are recorded from that interval, all originations in an interval would need to occur before any extinctions occurred. In the same vein, the heavy line is an approximate minimum diversity path within an interval. It would only be followed if extinctions followed originations in lockstep.

integrated with kill mechanisms buttressed by paleophysiological support.

Many triggers have been proposed for end-Permian extinction, but in recent years, geologists have focused primarily on four: bolide impact, Siberian flood basalt volcanism, anoxia in the oceans, and catastrophic release of seafloor methane.

3.1. Bolide impact

Following the discovery that bolide impact coincides with mass extinction at the end of the Cretaceous Period, geologists asked whether impact might also account for P–Tr catastrophe and perhaps even mass extinction in general. Fullerenes containing extraterrestrial gases, shocked quartz, metallic particles with meteoritic elemental abundances, and nanophase iron oxides have all been interpreted as evidence of end-Permian impact, and the enigmatic Bedout structure, off the coast of current day northwestern Australia, has even been proposed as the impact site [22]. Sharp criticism has met each of these claims [23,24], however, leaving the impact hypothesis in a state of skeptical limbo.

What kill mechanisms might be associated with extinction triggered by bolide impact? Impact is widely accepted as the trigger for Cretaceous–Paleogene mass extinction, but the associated kill mechanism(s) are still debated. In the absence of well substantiated mechanisms, we might naively guess that extinction should be random with respect to any discernible distribution of organismic characters. With more justification, we might use observed K–P extinction patterns [summarized in [25]] to predict end-Permian selectivity. In fact, neither provides a close match to end-Permian survivorship patterns [26].

3.2. Siberian trap volcanism

The largest known eruption of continental flood basalts coincided at least in part with end-Permian extinction [27–29], and global atmospheric and oceanographic changes facilitated by trap-associated volatiles

are commonly proposed to account for the biological catastrophe [*e.g.*, [30–32]].

Estimates of the original extent of Siberian flood basalts remain somewhat speculative, and the volatile load they carried is poorly constrained. Suspected basalt volumes are on the order of 10^6 km^3 [29], corresponding with an estimated release of 10^{17}–10^{19} mol CO_2 [*e.g.*, [32]]. At first glance this influx appears an astonishingly large, given that the late Permian atmosphere probably contained $10^{16–17}$ mol CO_2 [33]. Because the carbon cycle is in constant motion, however, the biological and climatic importance of such a large volatile release depends heavily on the timing and duration of the eruptions, as yet inadequately constrained. For example, integrated over a half million years, the estimated release would cause only a 10% to two-fold increase in annual volcanic outgassing. On the other hand, CO_2 fluxes from the Siberian Traps might have been augmented significantly by particular circumstances of emplacement and eruption. Basalts flooded onto and extruded through thick Proterozoic and Paleozoic carbonates and a late Paleozoic succession rich in coals and dispersed organic carbon. Decarbonation from these materials, perhaps especially Tungusskaya Series coals that nearly everywhere underlie the basalts [10], may have greatly increased the volatile load carried by Siberian Trap magmas and lavas.

Global kill mechanisms stemming directly from Siberian Traps volatile flux might, thus, include acid rain (CO_2 and SO_2), poisoning from halogen and halide gases, hypercapnia, and immediate but transient global cooling (increased particle and SO_2 flux) followed by global warming (CH_4 and CO_2). Additional kill mechanisms would extend to the oceanographic consequences of global climate change; for example, warming would lead to lower O_2 solubility in the oceans, facilitating the expansion of anoxia in oxygen-minimum zones (see below).

3.3. Shallow water anoxia

The wide distribution of black shales in lowermost Triassic shelf and platform successions has prompted interest in anoxia as a trigger for end-Permian mass extinction [34,35]. Biomarker lipids diagnostic of anoxygenic photosynthetic bacteria also occur in latest Permian and basal Triassic marine strata, providing independent evidence that many ocean basins were prone to anoxia within the photic zone [36]. Moreover, geochemical evidence suggests that the shallow water anoxia developed at the erathem boundary was superimposed on a longer term development of oxygen-poor deep waters, at least in the hemisphere-scale Panthalassic Ocean [37–39].

The atmospheric reservoir of oxygen is large relative to plausible fluxes of reducing agents on short (<1 Ma) timescales. Thus, while boundary-level marine anoxia may have been enhanced by a long-term decline in P_{O2} [40], it probably needs a proximal trigger to set it in place. Berner's [40] model estimates of Permian and Triassic P_{O2}, for example, show large and protracted Late Permian decline, but suggest little change coincident with the near-global expansion of shallow marine black shales. Moreover, his modeled P_{O2} nadir (0.13 bars) in the Middle Triassic occurs after shelf anoxia receded to background levels. Other estimates of Permo-Triassic P_{O2} show trajectories comparable to Berner's, but with abundances that never decline below present day levels [41].

Perhaps the proximal trigger for the expansion of shallow water anoxia was global increase in sea surface temperature induced by CO_2 influx into the ocean/atmosphere system. The oxygen content of subsurface ocean waters reflects the balance between downward organic flux, which drives the respiratory consumption of oxygen, and the upward diffusion of oxygen supplied to bottom waters by downwelling [42]. Global warming would have driven up ocean temperatures, increasing both rates of respiration (see below) and the temperature of downwelling waters — thereby decreasing the oxygen content of the O_2 minimum zone. Such a scenario implies that anoxia developed dynamically due to relative rates of oxygen supply and demand, not principally by the breakdown of physical mixing, which is difficult to sustain [*e.g.*, [43]].

Prolonged disruption of primary production would have the opposite effect, limiting available substrates for aerobic respiration and, hence, respiratory oxygen consumption. For this reason, the nearly global occurrence of black shales in basal Triassic successions provides *prima facie* evidence for continued photosynthesis in surface waters.

Unless the atmosphere itself were depleted of O_2, which it clearly was not, it would be impossible to make the shallow mixing zone of the oceans anoxic on a global scale. Thus, although anoxia may have been severe and widespread below the mixing zone, shallow waters would have served as biological refugia except at sites of pronounced upwelling. Anoxia, therefore, can be viewed as neither universal killer nor independent actor in this play. Trigger mechanisms for anoxia are intimately related to those that produce warming, and once in place, anoxic waters would develop increasing abundances of CO_2 and sulfide [21,44]. Thus, kill mechanisms linked to

anoxia would include not only regional asphyxia, but also global hypercapnia, H_2S poisoning, and perhaps, under extreme conditions, sulfide-driven loss of stratospheric ozone [21].

3.4. Catastrophic methane release

Catastrophic destabilization of seafloor methane clathrates has been suggested to account for the pronounced negative excursion in $\delta^{13}C$ recorded in P–Tr boundary carbonate rocks and organic matter [*e.g.*, [45], but see [46]]. Methane release provides an attractive explanation for the excursion because biogenic methane is depleted by as much as −60‰ in ^{13}C; like expanded marine anoxia, however, it probably requires a triggering event.

Recent observations show that the boundary excursion was just the first of a series of strong C-isotope fluctuations that continued throughout the Early Triassic [47]. It is difficult to explain all of these by simple repetition of methane storage and release because the intervals between excursions are too short regenerate methane reservoirs [47]. Thermogenic methane production during the intrusion of Tungusskaya coals by Siberian Trap sills provides an alternative mechanism perhaps more in line with geological observation [10,30,48].

Aside from short-term global warming, the kill mechanisms associated with large methane flux, be it from marine clathrates or Siberian coals, are unlikely to differ much from those associated with elevated carbon dioxide, as methane in the atmosphere would oxidize to CO_2 on a timescale of decades. As outlined above, such possibilities include global warming, hypercapnic stress, and the facilitation of marine anoxia [49].

3.5. Summary of triggers and associated kill mechanisms

The late Permian world constituted an extreme state of the Earth surface system, with maximum aggregation and minimum marine flooding of continents, an unusual east–west trending ocean basin in the tropics, and, after the melting of Gondwanid ice sheets and Kazanian glaciers in Angara, a low equator to pole temperature gradient [50]. This initial condition may have profoundly influenced the course of Permo-Triassic boundary events, maximizing the likelihood that anoxia would develop in the oxygen minimum zone (and perhaps through a much larger swath) of earliest Triassic marine basins. End-Permian eruption of massive flood basalts delivered large amounts of CO_2 and possibly CH_4 to the atmosphere, inducing global warming and, in consequence, the spread of anoxic water masses in the oceans. Anoxic water masses in which sulfate-reducing bacteria were active would have generated further abundances of carbon dioxide and hydrogen sulfide, both of which would be delivered to the surface ocean and atmosphere by upwelling. In consequence, the biota would have been subjected to asphyxia, hypercapnia, H_2S poisoning, thermal stress, and, possibly, increased UV radiation associated with H_2S-facilitated stripping of ozone.

4. Paleophysiology: the present as a key to the past

The scenario outlined above identifies a number of candidate kill mechanisms, and it is hard to envision the establishment of one without imposing most or all. Can the paleobiological record help sort out the relative impacts of these potential killers?

Studies addressing the individual and combined effects of hypoxia, hypercapnia, sulfide toxicity, and increased temperature on marine invertebrates are accumulating rapidly, not so much because the end-Permian extinction provides a compelling intellectual problem as because we ourselves live in a world where all of these effects are increasing, at least in part due to human activities. But, physiological research undertaken to illuminate contemporary environmental issues can also inform our thinking about ancient extinctions.

4.1. Oxygen depletion and asphyxia

Over the past half century, anthropogenic increases in nutrient fluxes have resulted in dramatically expanded eutrophication in the North and Baltic seas, the western Gulf of Mexico and elsewhere [51]. Most animal phyla contain species able to tolerate hypoxia and transient anoxia by decreasing oxygen consumption and scaling up anaerobic metabolism [52]. Tolerance to hypoxia can also be conferred by complex anatomical or molecular features that maximize oxygen extraction and/or suppress ATP demand [53,54]. Such features, however, are difficult to infer from fossils. In modern oceans, populations with different tolerances are distributed along the environmental gradient from oxygen saturation to anoxia; the most pronounced effects set in when P_{O2} falls below *ca.* 10% of saturation [55]. Exceptions exist, but in general, empirical studies suggest that polychaetes show the highest tolerance for oxygen depletion, and arthropods the least, with mollusks and echinoderms in between [55]. Other systematic changes along the seafloor redox gradient include decreases in biomass, size, skeletal mass and diversity.

Most physiological adaptations confer only transient tolerance to anoxia. Prolonged absence of oxygen (>60 days) causes total mortality, without selective survival [55]. Thus, hypotheses invoking anoxia to explain the selectivity of end-Permian mass extinction must focus on transient anoxia and the hypoxic margins of anoxic water masses, or on the differential survival of shallow water invertebrates most likely to have avoided oxygen-depleted waters. (Loss of habitat hypotheses requires consideration of species-area effects, as well. Island biogeographic models vary, but the rule of thumb that 90% loss of habitat area causes 50% species depletion [56] provides useful perspective — nearly all habitable shelf and platform area would have to be inundated by anoxic waters to account for the observed magnitude of extinction, leaving little opportunity for selectivity.)

4.2. Elevated P_{CO2}/hypercapnia

The greenhouse effect of CO_2 is widely appreciated by Earth scientists, but high P_{CO2} can also have profound effects on animal physiology. These include a decrease in the capacity of respiratory pigments to oxygenate tissues and disruption of internal pH balance, which affects the precipitation of carbonate skeletons and, at high concentrations, induces narcosis [57]. Death occurs on time scales of minutes to hours when marine animals are exposed to P_{CO2} of 10,000–30,000 ppm [58], levels likely to have been to be reached at the P–Tr boundary only under extreme conditions where CO_2-charged anoxic deep waters welled up to the surface [*e.g.*, [21,44]]. Even if CO_2 peaked at lower levels, however, chronic deleterious effects would have been likely. In modern oceans, decreased growth rate, survival, and reproduction can all occur when animals are exposed chronically (for weeks or more) to P_{CO2} as little as 200 ppm above ambient levels, depending on species [58,59]. For example, copepod and sea urchin populations subjected to P_{CO2} above 1000 ppm did not immediately die but exhibited both reduced fertilization rates and skeletal pathologies [60]. Also, experimental increase in P_{CO2} sharply reduced rates of protein biosynthesis in sipunculid muscle tissue [61]. In this context, it is worth emphasizing that consignment to extinction does not require the instantaneous death of all individuals in a species; a decrease of only 1% per generation will reduce animal populations to unsustainable sizes in little more than a century — an instant by geological standards.

In addition to its direct biochemical effects on skeletal physiology, increased P_{CO2} influences skeletal biomineralization by decreasing the pH of ambient waters, lowering $[CO_3^{2-}]$ and the degree of oversaturation with respect to carbonate minerals [62–65]. This increases the energetic cost of producing a carbonate skeleton. Skeletal physiology appears to be particularly vulnerable to acid–base perturbations, at least for organisms that precipitate calcite or aragonite. Experiments on organisms as varied as corals, mollusks, and coccolithophorids have documented decreases in the capacity for biomineralization when high P_{CO2} is imposed [62,66–68]. Andersson et al. [65] estimate that rates of biogenic calcification may have decreased by 11–44% in association with P_{CO2} rise since preindustrial times.

Geologists will note that Ordovician invertebrates precipitated carbonate skeletons beneath an atmosphere thought to contain 10–15 times as much carbon dioxide as today [33]. For organisms, however, the key variable is rate of change, not magnitude. It is the rapid, unbuffered increase in P_{CO2} and not its absolute value that causes important associated changes such as reduced $[CO_3^{2-}]$, pH, and carbonate saturation of seawater. Subjected to gradual increase over millions of years, marine carbonate chemistry will adjust and organisms can adapt their physiologies. Marked change over a few generations, however, leaves populations with only three options: tolerance, migration to more felicitous environments (if they existed), or death.

Organisms vary in their ability to compensate for hypercapnic stress and reduced $[CO_3^{2-}]$, and this provides a basis for predicting the effects of rapid, massive CO_2 increase at the P–Tr boundary. For example, O_2 and CO_2 diffuse similarly in air, but in water CO_2 is about 28 times more soluble than O_2. Thus, at comparable P_{O2}, air-breathers can ventilate at much lower rates than water-breathers to obtain the same amount of oxygen. Concomitantly, for respiratory CO_2 to be eliminated as metabolic waste, P_{CO2} in the body fluids and cells of air-breathers must build up to relatively high levels (15–40 Torr for tetrapods, 10–15 Torr common in terrestrial invertebrates) so that the pressure gradient across respiratory surfaces will be sufficient to force outward diffusion of CO_2 [71]. The reverse is true for water-breathers — internal P_{CO2} of aquatic organisms is never much higher than ambient conditions ($\sim$0.3 Torr in the modern ocean). Dejours [57] reported internal P_{CO2} of only 1–3 Torr in most marine animals studied, ranging from octopus to dogfish.

In consequence, marine animals are far more sensitive to hypercapnic stress than vertebrates living on land. Land plants are subject to CO_2 stress at very high levels, in no small part because of the effects of soil acidification on root aquaporin function [69], but more

moderate increases in carbon dioxide should not impact negatively on the core physiology of vascular plants and might actually increase net primary production [70]. From this simple perspective, it might be argued that increasing P_{CO2} should be beneficial to plant life; however, soil acidity may increase [71] and plant populations will change patterns of biomass allocation in ways that alter competitive abilities in unpredictable ways [72]. More importantly, plants *and* animals will be sensitive to climatic changes associated with increased P_{CO2}.

In aquatic environments, the animals most likely to be affected by rapid P_{CO2} increase include those characterized by high sensitivity of respiratory pigments to P_{CO2} (the Bohr and Root effects), limited elaboration of organs for circulation and gas exchange, and/or limited environmental exposure to chronically high or fluctuating P_{CO2} [44,57]. Invertebrates that secrete carbonate skeletons should be particularly vulnerable, especially organisms with limited physiological capacity to buffer against decreasing $[CO_3^{2-}]$ in ambient fluids. Indeed, Pörtner et al. [59] have argued that the differential vulnerability of calcifying marine organisms constituted the principal cause of mortality in end-Permian oceans (see below).

4.3. Hydrogen sulfide poisoning

The same environmental factors that motivate interest in marine hypoxia, plus problems of waste disposal and sewage treatment, fuel increasing physiological attention to sulfide tolerance in animals. In anoxic marine waters depleted in nitrate, sulfide (summed H_2S, HS^- and S^{2-}) levels increase as a function of anaerobic respiration by sulfate-reducing bacteria. Sulfide is almost universally toxic to eukaryotic cells in micromolar or higher concentrations, in part because it inhibits the activity of cytochrome *c* oxidase, the terminal electron acceptor in mitochondria [73]. Humans succumb almost instantly when exposed to H_2S at concentrations of 700–1000 ppm, about twice the lethal level for laboratory mice; more prolonged (hours or more) exposure can damage nervous tissues at much lower levels [74].

Animals display widely varying abilities to detoxify ambient sulfide, with organisms that live in association with organic-rich mud, hydrothermal vents, or other sulfide sources exhibiting the greatest degree of tolerance [75]. These mechanisms are predominantly molecular and may involve symbiotic association with sulfide-oxidizing bacteria. Notably, they commonly involve molecular oxygen [76] and so come into play in hypoxic, not anoxic environments; where oxygen is totally depleted, anaerobic metabolic capability is key to short-term survival. The relevant observations here are that (1) sulfide tolerance is more or less randomly distributed with respect to phylogenetic characters that can inferred from fossil morphologies and (2) tolerance is relative, not absolute. Prolonged exposure to sulfide causes death in most eukaryotes, just like prolonged exposure to anoxia. Perhaps the best paleobiological tests of P–Tr sulfide hypotheses are environmental: survival should be highest among marine animals from muddy/organic-rich habitats and lowest on land. More specifically, infaunal organisms chronically exposed to elevated sulfide should be more tolerant of systemic sulfide increase than epifauna that lack biochemical adaptations.

Kump et al. [21] argued that if sulfide levels in anoxic water masses built up to >1 mmol/kg, H_2S fluxes to the atmosphere would rise to levels 2000 times greater than today, triggering the loss of tropospheric OH radicals and, consequently, a major increase in tropospheric methane and destruction of stratospheric ozone. Atmospheric H_2S concentrations modeled for the onset of ozone depletion are 100 ppm, the level chosen by the National Institute for Occupational Safety and Health [77] as the threshold level that is "immediately dangerous to life and health" in humans. Elevation of sulfide concentrations to levels that deplete stratigraphic ozone would lead to radiation damage, with land animals and developmentally complex organisms being more vulnerable than marine and developmentally simpler organisms [78]. If fluxes from the oceans rose to levels beyond this threshold, H_2S in end-Permian air would soon reach generally acute levels of toxicity for vertebrate life.

4.4. Global warming and its physiological consequences

Global warming is probably the most widely discussed environmental issue of the early 21st century. Temperature affects a range of physiological and behavior activities, including aerobic metabolism in animals [79,80], time of flowering and other phenetic responses in plants, and sex determination in reptiles. Through these and other effects, temperature helps to determine the geographic distribution of species. As for other environmental parameters, rate of change is as important as magnitude.

Thomas et al. [81] modeled extinction risks posed by climate change over the past thirty years for land plants and animals, concluding that 15–37% of the species in their sample would be committed to extinction by 2050. Extinction probabilities were highest in temperate environments, intermediate in the tropics, and lowest in boreal and tundra regions. Thus, at a minimum, one might predict that extinctions driven by

the "post-apocalyptic greenhouse" [82] should show a distinct pattern of latitudinal variation.

4.5. Productivity collapse/starvation

Save perhaps for bolide impact, the trigger mechanisms outlined above do not necessarily predict the collapse of primary production in the oceans. Nonetheless, Martin [83] has proposed that the end-Permian extinction was governed by a crisis in primary production, and Rampino and Caldeira [46] have argued that the P–Tr C-isotope excursion can be modeled adequately in terms of short-term collapse of photosynthetic populations. More generally, Vermeij [84] has proposed that *all* extinctions begin with productivity collapse.

We cannot reject the hypothesis that primary production was transiently disrupted at the P–Tr boundary (for an interval below the limits of geological resolution), but global starvation seems hard to reconcile with the observation that taxa with high rates of basal and exercise metabolism survived much better than those with lesser oxygen demands [44]. As noted above, the observed expansion of anoxia in subsurface ocean waters suggests and may require sustained or rapidly resumed rates of primary production.

Eukaryotic algae might well have decreased in abundance both because they are more vulnerable to extinction than prokaryotic photoautotrophs and because remineralization of nitrate and ammonia in anoxic subsurface waters would favor primary producers capable of nitrogen fixation [85]. Photosynthetic bacteria, especially cyanobacteria, would have multiplied rapidly to pick up the slack, however, as evidenced by high 2-methylhopanoid indices in basal Triassic shales [36,86]. The nutrient status of cyanobacteria differs markedly from that of eukaryotic phytoplankton, particularly with regard to toxins and sterol abundances. Moreover, invertebrate animals in many different phyla lack the physiological capacity to synthesize sterols from low molecular weight precursors [87] and therefore rely on dietary sterols produced by algae. Cyanobacteria do not produce sterols and experimental evidence shows that arthropods and mollusks grow and reproduce poorly on cyanobacteria-rich diets [88,89]. Thus, the changing composition of marine phytoplankton may have contributed to P–Tr extinction and its aftermath even if changes in the amount of primary production did not [90].

4.6. Time scale and synergistic effects

One can draw two broad conclusions from the preceding discussion. First, rate and timescale are key to any consideration of kill mechanism. Sustained change in environmental parameters over tens of thousands to millions of years may permit genetic adaptation, enabling populations to accommodate to magnitudes of change that would be lethal if imposed rapidly — effective kill mechanisms act fast.

The relationship between environmental challenge and physiological response is, itself, time-dependent, albeit on much shorter time scales than those associated with adaptation. Acute and instantaneous physiological arrest may occur only at steeply elevated levels of CO_2 or H_2S, but chronic impairment of performance will accompany smaller increases that persist for hours, days or longer. Thus, the relevant environmental thresholds for P–Tr mass extinction need not be the high levels associated with acute trauma but rather much lower values that have sublethal but persistently detrimental effects. Rapidly imposed environmental changes apparent in the geologic record must have been long relative to the physiological response and generation times of organisms.

The second major conclusion is that the candidate kill mechanisms do not act in isolation. Hypercapnia, hypoxia, increased temperature, and bacterial sulfide generation are yoked together by environmental circumstance and have physiologically interactive effects. Pörtner et al. [59] explicitly emphasized the synergistic effects of temperature, CO_2 and hypoxia on the physiological performance of marine invertebrates. As noted above, hypercapnia can reduce respiratory capacity, exacerbating the physiological effects of environmental oxygen limitation. Temperature also appears to set limits on aerobic metabolism in marine animals, with temperatures above (or below) a species-specific threshold causing organismic oxygen demand to exceed supply [59,80]. Temperature, P_{O2}, and P_{CO2} interact synergistically to increase the vulnerability of organisms to coupled environmental change (Fig. 2) — and at lower thresholds than those associated with each parameter viewed in isolation. Higher P_{CO2} would also exacerbate the physiological effects of elevated sulfide in the oceans, because declining pH increases the proportion of total sulfide resident as H_2S, the chemical species able to diffuse freely across biological membranes [75].

A decade of physiological research on such synergistic effects has been summarized as follows: "all of these findings imply that high CO_2-induced shifts in cellular and organismic equilibria synergistically reduce the functional capacity of the whole organism with consequences on behavior, growth, reproduction and thus long-term survival in a changing ecosystem" [59].

5. Paleophysiology at the Permian–Triassic boundary

5.1. Extinction in the oceans

Each candidate kill mechanism predicts a specific pattern of P–Tr extinction and survival. How do paleontological data compare with these predictions?

Knoll et al. [44] divided Late Permian marine fauna into two groups based on predicted vulnerability to a particular kill mechanism, hypercapnic stress. In modern ecosystems, the highest vulnerability to hypercapnic stress is exhibited by marine animals characterized by low basal metabolic rate, limited or no circulatory system, little elaboration of respiratory surfaces, and precipitation of $CaCO_3$ skeletons under conditions of minimal physiological buffering. This group includes calcareous sponges, corals, calciate brachiopods, bryozoans, and most echinoderms: 81% of Changhsingian genera thus characterized disappeared at the end of the Permian Period. In contrast, organisms characterized by relatively high basal metabolic rate, elaborated circulatory systems and respiratory surfaces, and formation of calcium carbonate skeletons from physiologically well buffered fluids or without carbonate skeletons — principally mollusks, arthropods, and chordates — suffered only 38% loss of Changhsingian genera. Knoll et al. [44] also compared five other pairings of more and less vulnerable taxa *within* major clades and found that in every case the group predicted on the basis of physiological inference to be more vulnerable to hypercapnic stress did, in fact, experience significantly higher levels of extinction. The numbers of taxa involved in these comparisons are sometimes low, limiting the statistical power of individual comparisons; nonetheless, the likelihood that all would favor the same result by chance is $1/2^5$, or 0.03.

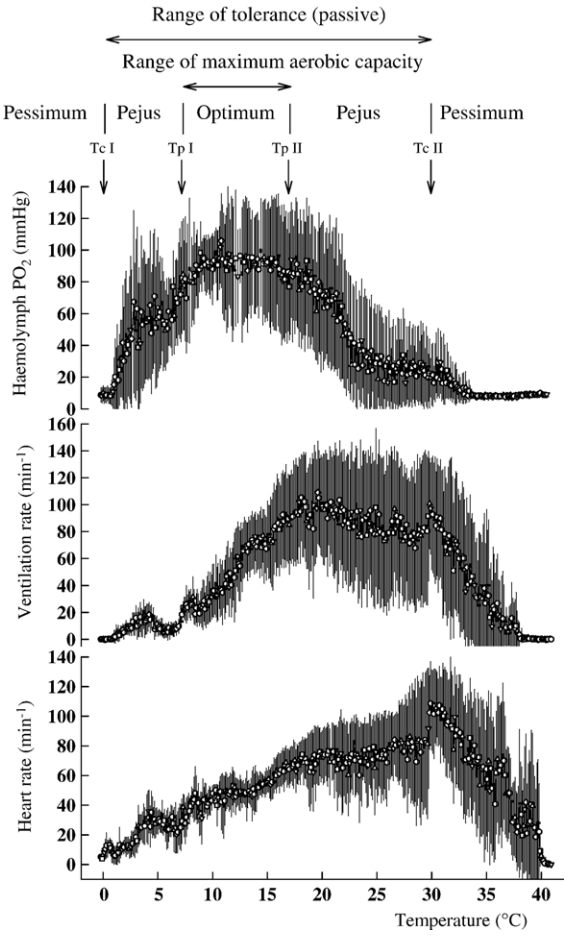

Fig. 2. Diagram showing the relationship between aerobic metabolism and temperature in the spider crab *Maja squinado*. The top graph shows that aerobic performance (indicated by blood P_{O2}) is maintained at an optimum level within a specified temperature range, falling off (pejus) to unsustainable levels (pessimum) beyond lower and upper thresholds. The middle and lower graphs show that the spider crab can maintain optimum aerobic performance in the face of rising temperatures by changing ventilation and heart rates, but only within specified limits — as temperature falls below 6 °C or rises above 16 °C, optimum performance cannot be maintained. Reproduced from [136] with kind permission of Springer Science and Business Media.

Pörtner et al. [58,59] provided physiological support for the hypothesis that skeletal physiology determined extinction vulnerability, but suggested that other factors related to hypercapnia were of secondary importance, largely serving to increase the stress on skeletal physiology. Inadvertently anticipating this criticism, Bambach and Knoll reanalyzed the Sepkoski database, this time parsing late Permian marine animals into three groups based solely on skeletal physiology: (1) animals with a calcium carbonate skeleton that was massive with respect to supporting organic tissue and formed from fluids minimally buffered by physiology (for example, corals and calciate brachiopods), (2) animals with a calcium carbonate skeleton of moderate mass with respect to supporting living organic tissue and formed from fluids that are relatively well buffered with respect to the factors that govern carbonate precipitation (principally mollusks and arthropods), and (3) animals with skeletons made of materials other than calcium carbonate (lingulid brachiopods, conodonts, cartilaginous fish) or with minimal, internal calcium carbonate spicules not used for mechanical support.

The results are striking (Table 1). Over 85% of the genera in group one went extinct at the P–Tr boundary, whereas group two lost about 54%. In contrast, only about 5% of the genera tabulated in group three disappeared — skeletal physiology really was destiny during the end-Permian catastrophe. Some taxa in group

Table 1
Extinction intensity and skeletal mineralogy

Taxon	Diversity	Extinction	% Extinction
Heavy carbonate load			
No physiological buffering			
Rugosa	19	19	100.0
Stenolaemata	17	6	35.3
Rhynchonelliform brachiopods			
Orthida	8	8	100.0
Strophomenida	67	62	92.5
Spiriferida	33	30	90.9
Rhynchonellida	17	14	82.4
Terebratulica	10	8	80.0
Acrotretida	1	1	100.0
Crinoidea	1	1	100.0
	173	149	86.1
Moderate carbonate load			
Potential physiological buffering			
Gastropoda	85	36	42.4
Bivalvia			
Infaunal burrowers	16	4	25.0
Epifaunal, attached	42	22	52.4
Nautiloidea	8	3	37.5
Ammonoidea	18	14	77.8
Ostracoda	53	41	77.4
Malacostraca	5	2	40.0
Echinoidea	2	1	50.0
	229	123	53.7
Little or no carbonate load			
Ctenostomata	4	0	0.0
Lingulida	1	0	0.0
Polychaeta	17	0	0.0
Holothuroidea	12	0	0.0
Conodontophorida	6	2	33.3
Chondrichthyes	3	0	0.0
	43	2	4.7

Taxa are divided into three groups, based on skeleton formation (see text). Genus diversity in the Changhsingian, number of genera that became extinct at the Permian–Triassic boundary, and percent extinction are given for each taxon listed. Data compiled from [13].

three had low diversity and/or limited abundance, so one might argue that taphonomic and taxonomic issues compromise direct comparison of this group with the other two. Regardless of this concern, the contrast between groups one and two is highly significant. Moreover, a number of further comparisons all point in the same direction-selective extinction with regard to skeletal physiology during the end-Permian event. Among foraminiferans, for example, the massively calcified fusulinids were annihilated, with only two of 40 genera persisting into the Olenekian [91]. Genus diversity in other calcareous foraminiferans dropped from 53 to 8 from the Changhsingian to the Induan, with subsequent Middle Triassic rediversification [91]. In contrast, textulariinids, which have agglutinated tests, lost only four of 33 genera [12], paralleling the pattern of selectivity exhibited by animals.

Further analysis of group two provides additional support for the hypothesis that hypercapnic stress governed the selectivity of end-Permian mass extinction. Within the bivalve mollusks, sedentary epifauna, which neither generate high levels of metabolic CO_2 from burrowing activity nor encounter elevated P_{CO2} in their normal environments, had twice the proportion of extinction (52 *vs.* 25%) found among infauna that live where ambient P_{CO2} is high and generate large amounts of respiratory CO_2.

Another, more speculative example leans in the same direction. Seventy-nine percent of ammonoid genera became extinct at the end of the Permian, whereas the related nautiloids lost only two of seven genera (29%). Ammonoids are extinct and so cannot be directly evaluated physiologically, but they are now known to lie within a derived clade that also includes the extant coleoids [92,93]. Coleoids exhibit a severe Bohr effect, in which CO_2 inhibits the transport of oxygen by respiratory pigments [94]; therefore, they are sensitive to hypercapnic stress. In contrast, the living tetrabranch cephalopod *Nautilus* shows no strong Bohr shift [95]. If Late Permian ammonoids and nautiloids had physiological tolerances similar to those of their closest living relatives, hypercapnic stress provides a reasonable explanation for their differing evolutionary fates.

Other candidate kill mechanisms might also be expected to select against anatomically simple organisms — which include most skeleton builders that lack strong physiological buffering capacity — but kill mechanisms other than hypercapnia cannot account for some paleontological patterns:

- Late Permian corals disappeared but their unskeletonized relatives, the sea anemones (which would give rise to scleractinian corals in the Triassic) did not;
- skeletonized dasyclad green algae disappeared, but multiple unskeletonized sister groups did not [96];
- with few exceptions, calcified red algae disappeared, but a large number of uncalcified clades persisted, including the progenitors of Mesozoic and Cenozoic coralline taxa;
- as noted above, calcareous foraminiferans were decimated, but agglutinated forams were not.

If calcareous skeletons represent the common theme among these victims *vs.* survivors, CO_2 provides a reasonable kill mechanism, whereas oxygen and H_2S stress by themselves do not. Neither does productivity collapse predict the broad pattern of extinction, as

groups with higher metabolic demands survived differentially well.

Does this mean that anoxia and sulfide had no effect on end-Permian extinctions? Not at all. Euxinic water masses persistent enough to leave a stratigraphic record would have decimated faunas wherever they developed. Such water masses may have killed a majority of the animals in latest Permian oceans, but they would not have been selective killers; nor could they have been ubiquitous — oxic refugia persisted, and some 30% of latest Permian marine genera survived into the Triassic. In contrast, hypercapnic stress on organisms not subjected to lethal anoxia would have been both selective and globally pervasive. Indeed, hypercapnia would be expected to have particularly strong effects on taxa well represented in the rocks — the producers of robust carbonate skeletons. Multiple killers were undoubtedly at work, but it is principally hypercapnia that could impart the observed signal of *selectivity* to the paleontological record. Hypoxia, sulfide generation, and temperature increase likely operated *synergistically* with elevated P_{CO2} to cause selective extinction in end-Permian oceans, with low oxygen and high temperature accentuating the pattern of selective extinction associated with hypercapnic stress on $CaCO_3$-precipitating invertebrates [59].

5.2. Extinctions on land

Any viable kill mechanism must be consistent with extinction and survival on land as well as in the sea. Patterns of change among land plants and vertebrates are relatively difficult to identify with confidence because most regions do not preserve continuous records across the extinction horizon. Plants show evidence of rapid boundary-level extinction and floral reorganization in both Gondwana [97] and the peri-North Atlantic region [15]; however, data compiled by Rees [98] show an *increase* of floral diversity in the northern temperate regions of Angara (see also [99]), suggesting that plant responses to end-Permian events were region-specific. Indeed, major elements of the Triassic flora that evolved in Gondwana appear to have originated in paleotropical Permian habitats and migrated poleward following the extinction event [100].

Widespread teratologies in earliest Triassic lycopod spores have been interpreted in terms of environmental mutagenesis, possibly related to enhanced UV radiation [101]. Foster and Afonin [102] demonstrated aberrant development in earliest Triassic gymnosperm pollen, as well, but cited literature showing that developmental mutations can reflect many environmental insults, including acid rain, sulfur dioxide, and elevated P_{CO2}, as well as harmful radiation. Thus, developmentally aberrant spores and pollen document acute short-term perturbation of terrestrial environments, but do not specify the type of perturbation.

Visscher et al. [103] also recorded the widespread occurrence of unusual microfossils interpreted as the spores of fungi that proliferated following end-Permian mass mortality of woody plants. Phylogenetic interpretation was based on fluorescence, not morphology, which is not diagnostic. Fossil algae can also fluoresce [104], however, and isotopic and microchemical signatures convinced Foster et al. [105] that these fossils record bloom-forming green algae — putting a very different spin on their occurrence.

The best known record of vertebrate changes across the P–Tr boundary occurs in the Karoo Supergroup, South Africa. Tetrapod faunas sustained marked diversity decrease through the Late Permian, but relatively few extinctions appear to have occurred at the P–Tr boundary. Smith and Botha [106] suggest that 69% of latest Permian tetrapods disappeared through the last 300,000 years of the period, with 31% of survivors becoming extinct *ca.* 160,000 years later. In contrast, Ward et al. [16], who used the same data but provided confidence estimates on first and last stratigraphic appearances, show pronounced turnover but no significant or persistent dip in species diversity from 60 m below to 140 m above the interpreted boundary. Ward et al. concluded that end-Permian tetrapods experienced protracted, not instantaneous, ecosystem change. Marshall's [107] reanalysis of Ward et al.'s confidence intervals suggests that the Karoo record is compatible with boundary level extinction followed by repopulation, but the implied time scale for new species introductions is so rapid that it would have to reflect immigration rather than evolution. *Lystrosaurus maccaigi*, a therapsid that disappears at the hypothesized P–Tr boundary in South Africa, occurs in Antarctica with Triassic fossils [108], underscoring the importance of regional survival and immigration for the vertebrate record.

Triassic insects differ markedly from those in Permian rocks but the interval between the youngest informative Permian entomofauna and the oldest Triassic example is *ca.* 15 million years, leaving it (once again) unclear whether insects experienced marked extinction and subsequent reradiation or simply accelerated turnover at the boundary [109]. Interestingly, putative insect burrows in fluvial sandstones from Antarctica do not show the pronounced P–Tr decrease in size and burrowing depth observed in contemporaneous marine ichnofossils [110].

A drop in global atmospheric P_{O2} sufficient to drive major marine extinctions (*ca.* 85–90% decrease) would

kill land animals indiscriminately, which didn't happen. Huey and Ward [111], however, argued that declining P_{O2} throughout the later Permian Period imposed hypoxic stress on tetrapod communities adapted to high oxygen tensions. Unfortunately, this intriguing and physiologically explicit hypothesis cannot be extended to include boundary events *per se*, as model estimates of P_{O2} show little change at this critical juncture [40,41].

We know of no mechanism by which the direct effects of anoxia documented in shallow seaways could have propagated onto land. Nor does it seem that plants or land vertebrates should have been decimated by CO_2 levels likely to have affected calcifying animals in the oceans. Rather, regional patterns of plant extinction and accelerated taxonomic turnover among vertebrates appear to be consistent with *climatic* effects stemming from sharply increased P_{CO2}. The land record is not sufficiently complete to evaluate potential effects of H_2S efflux from upwelling in the oceans, but the survival of multiple plant and animal clades indicates that any sulfide effect must have been limited.

6. Paleophysiology in Early Triassic Oceans

Several features of Lower Triassic rocks reflect pronounced but relatively short-lived environmental perturbation (Fig. 3). Geologic evidence of marine anoxia, for example, is widespread in Griesbachian shelf and platform successions, but not thereafter — regional recurrences of anoxia are recorded in some Smithian and Spathian basins, but not in the shallow facies affected by earliest Triassic perturbation [39]. Euxinic facies are neither universal nor strictly correlative, suggesting a stratigraphically limited global *propensity* toward shallow anoxia that was realized regionally.

Similarly, precipitated microbialites and macroscopic carbonate crystals precipitated on the seafloor are most abundant immediately above the P–Tr boundary, although they recur episodically throughout the Early Triassic [[112] and references therein]. Upwelling of alkalinity-charged anoxic waters provides a mechanism capable of linking black shales and seafloor carbonate precipitates [113].

Pollen and spore teratologies likewise faded after the Griesbachian, as did biomarkers indicative of photosynthetic bacteria. However, other biological phenomena initiated at the P–Tr boundary persisted until Middle Triassic times, an interval of at least 4–5 million years. These include:

- low diversity [114]
- small size of marine animals [115–117]
- reduced abundance of skeletal benthic invertebrates [118]
- reduced bioturbation and the consequent expansion of flat pebble conglomerates and associated facies more typical of Lower Paleozoic and older rocks [112,119,120]
- absence of biogenic deposits such as coals [121], cherts [122], and skeletal framework reefs [123].

6.1. Diversity and abundance patterns

By definition, low diversity is the outcome of mass extinction, and in the absence of other data it would be hard to determine whether the persistence of low diversity faunas reflects sustained environmental inhibition or simply the depth of the wound to latest Permian diversity. Simple rebound *via* population growth, however, does not predict the observed *compositions* of Early *vs.* Middle Triassic marine communities. Early Triassic communities were dominated by mollusks and phosphatic brachiopods, with echinoderms becoming more abundant late in the Early Triassic [114,118]. Taxa with the least capacity to modify seawater by pumping ions into and out of the fluids from which carbonate skeletons are precipitated — cnidarians, sponges, and calcareous algae — are absent or nearly so from this Early Triassic record [123]. It has been proposed that relatively diverse faunas from well-oxygenated environments record early recovery in Early Triassic oceans [124], but insofar as the assemblages in question share their major features of organism size, taxonomic composition, and diversity with other Early Triassic faunas, they are better interpreted in terms of ecological variation among habitats in a persistently depauperate Early Triassic world [125].

Quantitative analyses of carbonate sediments in South China [118] support qualitative observations of widespread reduction in skeletal contributions to Early Triassic carbonate sediments. Radiometric constraints on depositional rates in South China further indicate that the observed reduction is not merely a dilution effect caused by higher input of non-skeletal components. Potentially, the decrease in skeletal biomass could have been offset by a commensurate increase in the abundance of non-skeletal organisms, say, of worms or sea anemones. Documented decreases, however, in the both the size and penetration depth of trace fossils [119,120] and a dramatic expansion of microbial mats across the seafloor [126,127] suggest that non-skeletal animals did not increase in abundance. Thus, a strong case can be made for reduced animal biomass in post-extinction oceans that persisted for several million years.

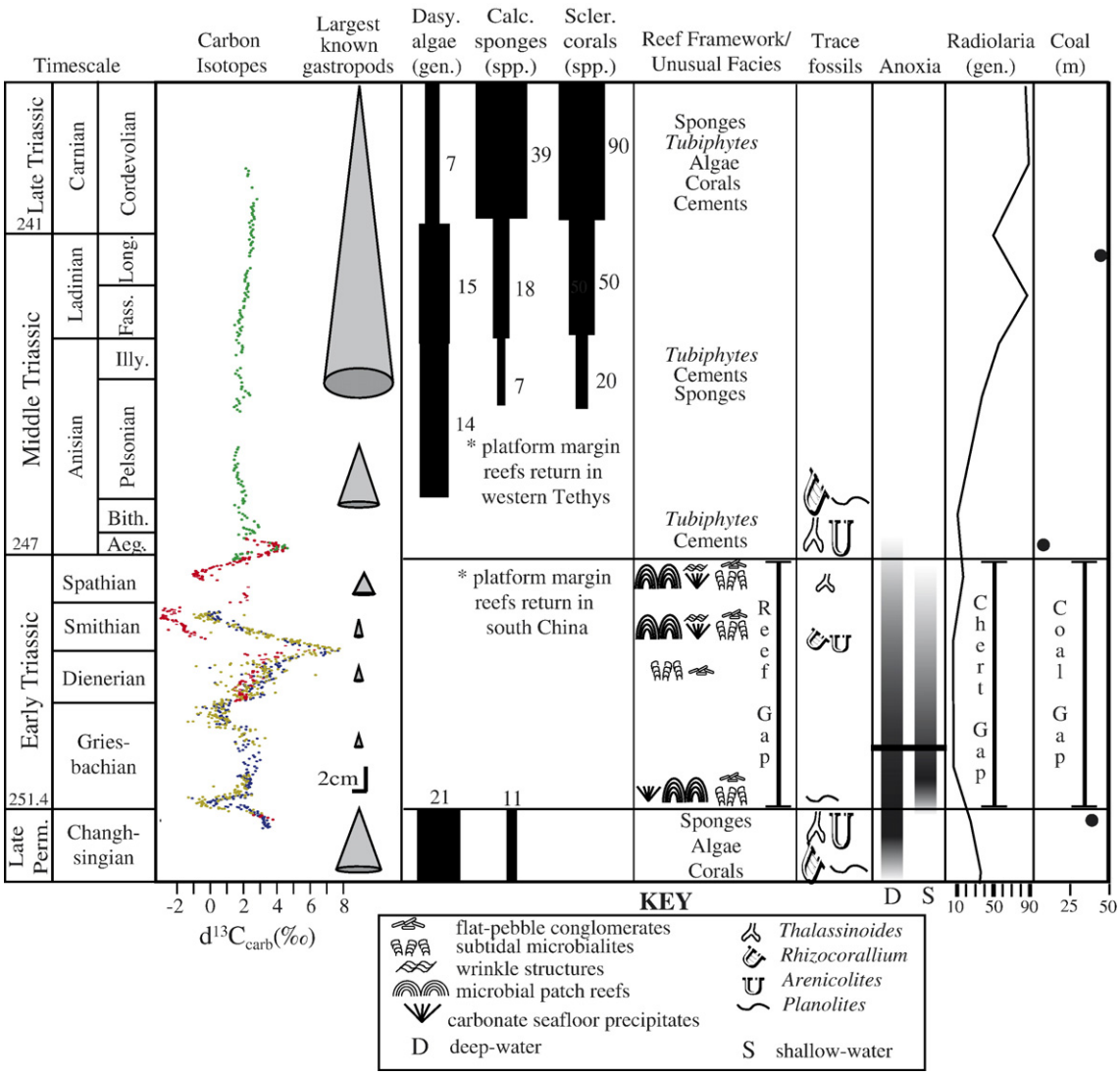

Fig. 3. Compilation showing characteristics of the Early Triassic record in comparison to preceding and following intervals. Sources of data: C-isotopes showing the repeated excursions through the Early Triassic, which end near the beginning of the Middle Triassic [47]; maximum gastropod size showing the absence of large adults in Early Triassic assemblages [116]; dasyclad algal diversity [96]; calcareous sponge diversity (includes sphinctozoan and inozoan genera) [133]; scleractinian coral diversity and reef fabric data [123]; ages of the oldest Triassic reefs [123,134]; unusual facies [112]; trace fossils showing the disappearance and delayed recovery of some equatorial trace fossils [119]; anoxia [34] — maximum level of anoxia in mid-Griesbachian indicated by dark horizontal line; radiolarian diversity [135] — note that the chert gap also reflects a dearth of siliceous sponges; coal seam thickness [121].

Reduced animal biomass could result from two different processes — diminished food supply or reduced consumption and growth efficiencies of animals. Low density of epifaunal and burrowing organisms and greatly reduced tiering [128] suggest that the consumption efficiency of Early Triassic communities was reduced relative to their Late Permian counterparts. The dominance of mollusks, which have substantially higher metabolic rates and, consequently, lower growth efficiencies than most brachiopods [129] and other heavily calcified animals, indicates that the aggregate growth efficiencies of organisms in Early Triassic communities were likewise diminished, with the expected result of reduced animal biomass. Because per gram metabolic rates are inversely correlated with body size [*e.g.*, [130]], the small mean size of Early Triassic organisms would further decrease the aggregate growth efficiency of Early Triassic communities.

All of the above would be exacerbated by high P_{CO2} and global warming. Temperature increase would not be

likely to increase primary production governed in the first instance by nutrient availability. They would, however, increase rates of respiration according to the Q_{10} rule, among the most universally observed of all physiological relationships [2]. Noting that many marine invertebrates feed on bacteria that are themselves heterotrophic, increased respiratory rates in warm Early Triassic oceans would markedly affect the conversion of primary production into animal biomass. Moreover, as noted above, rates of protein synthesis in at least some marine animals decline under conditions of elevated P_{CO2} [61]. Thus, basic physiological and ecological principles can account for greatly reduced abundance of Early Triassic marine invertebrates, without invoking lower primary productivity.

Accelerated ecological and evolutionary recovery took place in Spathian to Anisian oceans. Within a brief interval characterized by the return of C-isotopes to long-term stability, robust skeletons reappeared in a number of poorly buffered groups, including calcareous sponges, cnidarians, green algae, and red algae (Fig. 3). Indeed, the Early Triassic "reef gap" simply reflects the paucity of massively calcifying invertebrates and algae. During the recovery interval, taxa in our poorly-buffered group one actually diversified at higher rates than the well buffered group two that dominated Early Triassic assemblages [15,131]. The proportional contribution of group one organisms to carbonate accumulations increased, as well [118].

The pattern and timing of recovery, thus, shows evidence of physiological selectivity comparable to that of end-Permian extinction. Taxa least sensitive to hypercapnic stress dominated most Early Triassic deposits, whereas physiologically sensitive taxa were rare or absent until the Spathian and Anisian. Together, the diversity and composition of Early and Middle Triassic marine communities suggest that some version of the environmental conditions established at the time of mass extinction persisted or recurred for the next 4–5 million years, even if the acute disruption that precipitated the crisis did not.

6.2. Additional observations and summary

Persistent or recurring hypercapnia/global warming helps to explain other phenomena observed in Lower Triassic rocks. For example, the widely recognized coal gap might reflect increased rates of soil respiration as well as shifting climates. In the oceans, the observed Early Triassic expansion of microbialites requires only that animals which graze on mats or compete for substrate space decline in abundance. Precipitated stromatolites and macroscopic seafloor cements, however, are not default conditions in skeleton-poor oceans — except during cap carbonate formation following Snowball glaciation, macroscopic precipitates were not common features of carbonate deposition during the 300 million years leading up to the Cambrian explosion [113]. Such features require unusual sources of carbonate alkalinity that, as noted above, are consistent with a warm, CO_2-rich ocean prone to anoxia beneath the mixed layer.

In summary, Early to Middle Triassic patterns in marine biological diversity, community composition, size and abundance of fossils, and stratigraphic features of carbonate and other depositional systems are consistent with hypotheses of sustained or recurring anoxia, hypercapnia and global warming in the Early Triassic biosphere. If correct, this poses a challenge. Carbon cycle models predict that a rapid infusion of CO_2 would be removed by chemical weathering on time scales of millennia to a few hundred thousand years [*e.g.*, [40]]. Sustaining high P_{CO2} over million year time scales would require that the Earth entered a new steady state following the extinction, as suggested by Broecker and Peacock [132] (although the state they appear to have had in mind was not established before the Middle Triassic). Alternatively, elevated influx of CO_2 into the Earth surface system might have continued *episodically* through Early Triassic time, perhaps reflected in the record of large C-isotopic variations that ended only with Middle Triassic biotic recovery. Mechanisms for recurring CO_2 influx are speculative, but insofar as deep water anoxia persisted or recurred until the end of the epoch, it provides a potential source for episodically upwelling CO_2. Also, we do not yet know whether all Siberian trap basalts formed at the same time or erupted rapidly but episodically in discrete geographic locations over several million years. Comprehensive dating of these massive deposits will help to constrain hypotheses about Early Triassic life and environments.

7. Conclusions

Inferences from geochemistry and paleontology concur in discouraging simple one-parameter explanations for mass extinction at the Permian–Triassic boundary. Debate must shift from hypotheses of hypercapnia *vs.* hypoxia *vs.* sulfide poisoning *vs.* global warming to consideration of a world in which all were simultaneously in play. The new focus is not a resurrected "Murder on the Orient Express" scenario in which unrelated bad things happen together by chance, but rather the coordinated imposition of physical perturbations that are inextricably linked in the Earth system and

which would have acted synergistically to disrupt the metabolism of end-Permian organisms. In our view, hypercapnia best accounts for the pattern of selectivity recorded by marine fossils, not because it acted alone but because its effects were global and focused on the skeletal organisms that dominate the record. At the same time, high P_{CO2} appears to have exerted a primary influence on P–Tr land life as well, in this case *via* global warming. The unusual biological and physical features of Lower Triassic rocks are consistent with the hypothesis that environmental changes set in motion at the P–Tr boundary persisted or recurred for 4–5 million years, governing the timing and pattern of biotic recovery in the oceans and, perhaps, on land.

We reemphasize that the observed pattern of selective survival does not arise because some groups were little affected by the boundary event while others suffered greatly. Nearly all major taxa in the oceans suffered high proportions of extinction, and a majority of species disappeared even in the groups that survived differentially well and dominated Early Triassic ecosystems. Nonetheless, survivors preferentially fall into a distinct group united by basic features of anatomy and physiology, and these seeded new ecosystems. Earliest Triassic land vertebrates similarly display anatomical features such as respiratory turbinals and behavioral characters such as burrowing that would have facilitated survival in the face of hypercapnia and rapid global warming [44,49,111].

The pattern of selectivity associated with P–Tr extinction is unique [26], but the logic developed here can be applied more broadly to investigations of Earth and life through time, including both other major extinctions and long-term changes in the state of Earth's atmosphere and oceans. The important point is that physiology offers a critically important paleobiological way of knowing in the age of Earth system science. Physiological insights developed to improve understanding of the present and near term future help, as well, to illuminate our planetary past.

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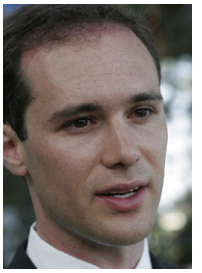

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