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Peer Review

Review of: "Atmospheric CO_2 as a Biophysical Driver of Mesozoic–Cenozoic Biodiversity"

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1. Independent researcher

General Assessment

The manuscript proposes an original and thought-provoking hypothesis linking the long-term decline of atmospheric CO_2 to the increase of Phanerozoic biodiversity and vertebrate encephalization. The proposed physiological mechanism, based on the thermodynamic framework of Buxton (2024), is potentially relevant to questions of metabolic performance and perhaps to the evolution of energetically expensive traits such as large brains. However, the extension of this mechanism to explain long-term biodiversity trends is not convincingly justified.

In my opinion, the manuscript's central weakness is that it conflates several distinct evolutionary phenomena — biodiversity increase, cladogenesis, biological complexity, encephalization, and intelligence — and treats them as different expressions of a single underlying process. While these phenomena may be correlated in some circumstances, they operate at different evolutionary levels and do not necessarily share the same causal mechanisms.

1. Biodiversity and Encephalization Operate at Different Evolutionary Levels

The manuscript implicitly assumes that biodiversity increase and encephalization can be explained through a common biophysical mechanism. However, these processes correspond to fundamentally different aspects of evolution.

Biodiversity primarily reflects the balance between speciation (cladogenesis) and extinction. Encephalization, by contrast, is a phenotypic trait that evolves within particular lineages. In evolutionary

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terms, biodiversity concerns the branching structure of the tree of life, whereas encephalization concerns changes occurring along individual branches.

Consequently, even if atmospheric CO_2 were shown to influence metabolic efficiency and brain evolution, it would not automatically follow that it also controls biodiversity. The manuscript does not provide a mechanism linking these two processes.

2. Absence of a Speciation Mechanism

The manuscript attempts to explain long-term increases in marine genus diversity through a physiological mechanism involving metabolic scope. However, no mechanism is presented through which atmospheric CO_2 could systematically increase rates of cladogenesis.

The classical theory of speciation, from Wright onwards, requires some form of population subdivision, reduction of gene flow, and eventual reproductive isolation. The manuscript does not explain how a globally distributed atmospheric parameter such as CO_2 could generate these conditions.

The missing causal chain is therefore:

Environmental change
→ population isolation
→ reduction of gene flow
→ cladogenesis
→ biodiversity increase

Without such a mechanism, the manuscript establishes at most a correlation between CO_2 and biodiversity, not an explanation for biodiversity.

3. Methodological Framing

The three criteria proposed by the author for a candidate "complexity driver"

1. long-term temporal change,
2. connection to metabolic performance,
3. global distribution,

are appropriate for evaluating physiological drivers of organismal performance, but not necessarily for evaluating drivers of biodiversity.

Processes known to generate biodiversity are often regional rather than global and operate through population structure and geographic isolation rather than through metabolism.

Thus, the manuscript begins from a framework tailored to physiological evolution rather than to speciation dynamics.

4. Use of Previous Literature

Several cited works appear to be interpreted more broadly than their original scope supports.

- Dawkins' *The Blind Watchmaker* explains how cumulative natural selection can generate complex adaptations. It does not argue that biodiversity, encephalization, and intelligence inevitably increase through time.
- Lovelock and Margulis (1974) discuss atmospheric homeostasis and planetary habitability, not mechanisms of biodiversity increase or encephalization.
- Salles et al. (2023) primarily address carrying capacity, productivity, and habitat availability. Their study concerns the maintenance and support of biodiversity rather than the generation of new lineages through speciation.

5. Biodiversity Dataset

The biodiversity analysis relies heavily on the Sepkoski diversity curve. However, the long-term increase recorded in this curve has been challenged by later studies that identified substantial sampling biases, including the "pull of the recent" and unequal geographic sampling through time.

The manuscript acknowledges these concerns but does not repeat its analysis using corrected datasets such as those developed by Alroy or Close et al. Consequently, the robustness of the biodiversity correlation remains uncertain.

6. Encephalization Dataset

The encephalization analysis is based on Russell's (1983) compilation of maximally encephalized organisms through time.

Importantly, this dataset does not measure the average encephalization of vertebrates, nor the overall cognitive evolution of the biosphere. Instead, it records the single highest encephalization quotient (EQ) achieved at different times.

Therefore, the observed correlation concerns the upper envelope of encephalization rather than a general trend affecting vertebrates as a whole. The manuscript should acknowledge this limitation more explicitly.

Conclusion

The manuscript presents an interesting physiological hypothesis linking atmospheric CO_2 to metabolic performance and perhaps to encephalization. However, its extension to biodiversity is not adequately supported because no mechanism is provided linking atmospheric CO_2 to cladogenesis or extinction dynamics.

The central issue is not whether CO_2 may influence organismal physiology. The central issue is whether a physiological mechanism can be transferred directly to explain a macroevolutionary pattern that ultimately depends on the origin and persistence of evolutionary lineages. In its present form, the manuscript does not demonstrate that connection.

Suggestions for Improvement

The manuscript would be substantially strengthened by:

1. Explicitly separating the biodiversity hypothesis from the encephalization hypothesis and evaluating them independently.
2. Providing a mechanistic explanation linking atmospheric CO_2 to speciation or extinction dynamics rather than relying primarily on correlations with biodiversity curves.
3. Repeating the biodiversity analyses using sampling-corrected datasets (e.g., Alroy 2010; Close et al. 2020) in addition to the Sepkoski curve.
4. Clarifying that the Russell dataset represents maximum encephalization (upper-envelope evolution) rather than average vertebrate encephalization through time.
5. Testing whether the reported correlations remain significant after detrending, autocorrelation correction, and other standard time-series procedures.
6. Exploring whether the proposed mechanism is better interpreted as affecting extinction resistance, carrying capacity, or metabolic performance rather than speciation itself.

Declarations

Potential competing interests: No potential competing interests to declare.

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