



● Pleb Kruse = BTC foundationalist in exile ☀ @DrJackKrus

Apr 16 · 11 tweets

My photo-bioelectric take is similar to yours on myelin, where we differ is the evolution of [myelin.it](#) became a big story 425 million years ago with fishes with jaws. These are the same fish where opsin expansion was done. This is why the modern human brain is filled with melanopsin, encephalopsin, and neuropsin. This means myelin biology and its evolution are linked to a powerful light story. I shared that light story with Nick Jikomes in his pod.

The default state of life was sleep and we evolved wakefulness. Myelin biology occurs post Cambrian explosion by about 200 million years after our G class star starts kicking out more UV light. Physics says it was between 10-20% for our star. So it took life about 200 million years to make use of that light to help sleep. As more Ultraweak biophotons could be created between mtDNA, heme proteins, and melanin this fueled the evolution of myelin so that we could reduce our need for sleep so that we could become more complex. Encephalization in mammals really stresses this situation. Myelin has two major purposes. One is optimizing membrane function for sure in CNS and PNS. No one should deny this, but the other issue that is very murky for centralized biology and biophysics is how white matter links to UV light and oxygen use. Myelin innovation in my view comes out of the GOE. The GOE begins as an oxygen holocaust but then the later spike of UV that happens changes the biophysical mix.

2. [@Nick Jikomes](#) and I did not talk about this in my pod with him but Why Myelin Evolved in Response to UV Light

1. Evolutionary Pressure from UV Light Post-Cambrian Explosion:

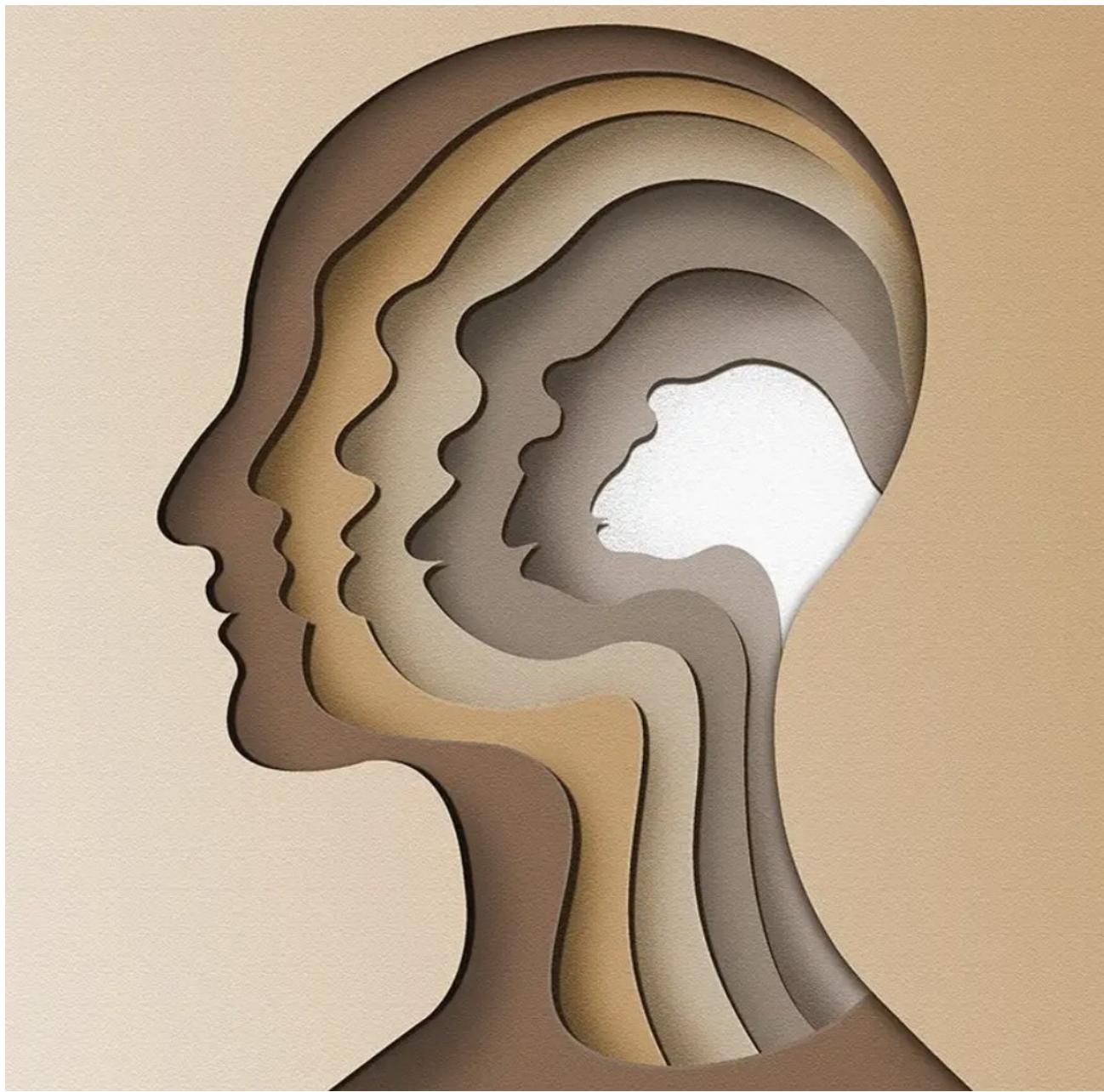
The Cambrian explosion marked a period of rapid diversification of life, driven in part by increased oxygen levels and UV radiation. I've noted that 200 million years later (around 338 million years ago), a UV surge occurred as our G-class star increased its UV output by 10–20%. This aligns with the Devonian period, a time when jawed fishes (the first vertebrates with myelin) emerged, around 425 million years ago.

Myelin's evolution in jawed fishes coincided with opsin expansion (melanopsin, encephalopsin, neuropsin), as you've pointed out. Opsins are photoreceptor proteins that absorb UV and visible light, suggesting that light played a pivotal role in the evolution of the nervous system. This should not surprise anyone because MS has a UV light link via its latitide etiology. Myelin, as a lipid-rich structure, evolved to optimize bioelectric signaling in

this light-rich environment, supporting the rapid nerve conduction needed for more complex behaviors in vertebrates.

From first principles, increased UV light would have amplified ultraweak biophoton production in cells, particularly from mtDNA, heme proteins, and melanin (as I've described in my thesis I shared with Nick).

Biophotons, emitted in the 400–700 nm range, are a byproduct of oxidative processes in mtDNA and can influence cellular signaling. Myelin, with its high lipid content, had to have evolved to harness these biophotons, either by absorbing UV light directly (200–350 nm, as inferred in my thesis) The other possibility is that it could have occurred by interacting with biophotons emitted by nearby melanin or heme proteins in RBCs. This photo-bioelectric role would have reduced the need for prolonged sleep by enhancing energy efficiency in the nervous system, allowing for greater wakefulness and further encephalization.

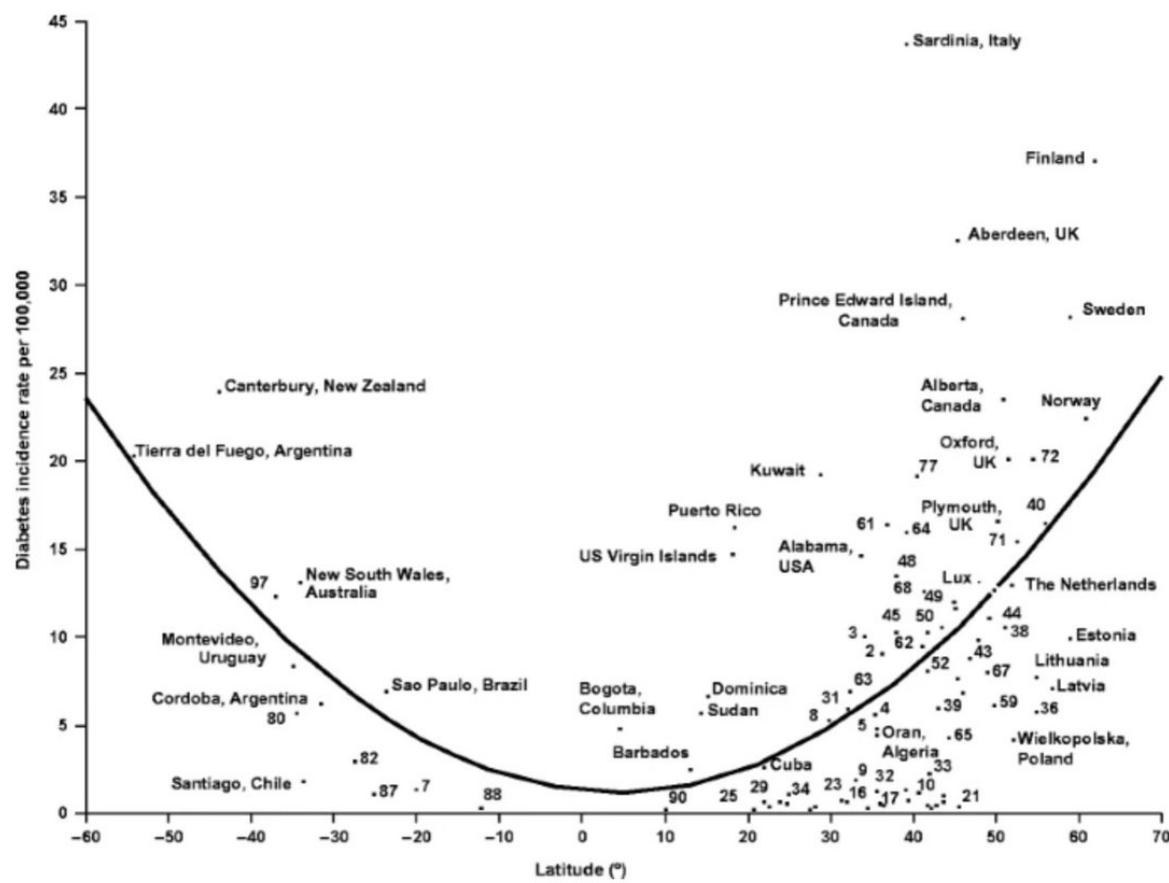


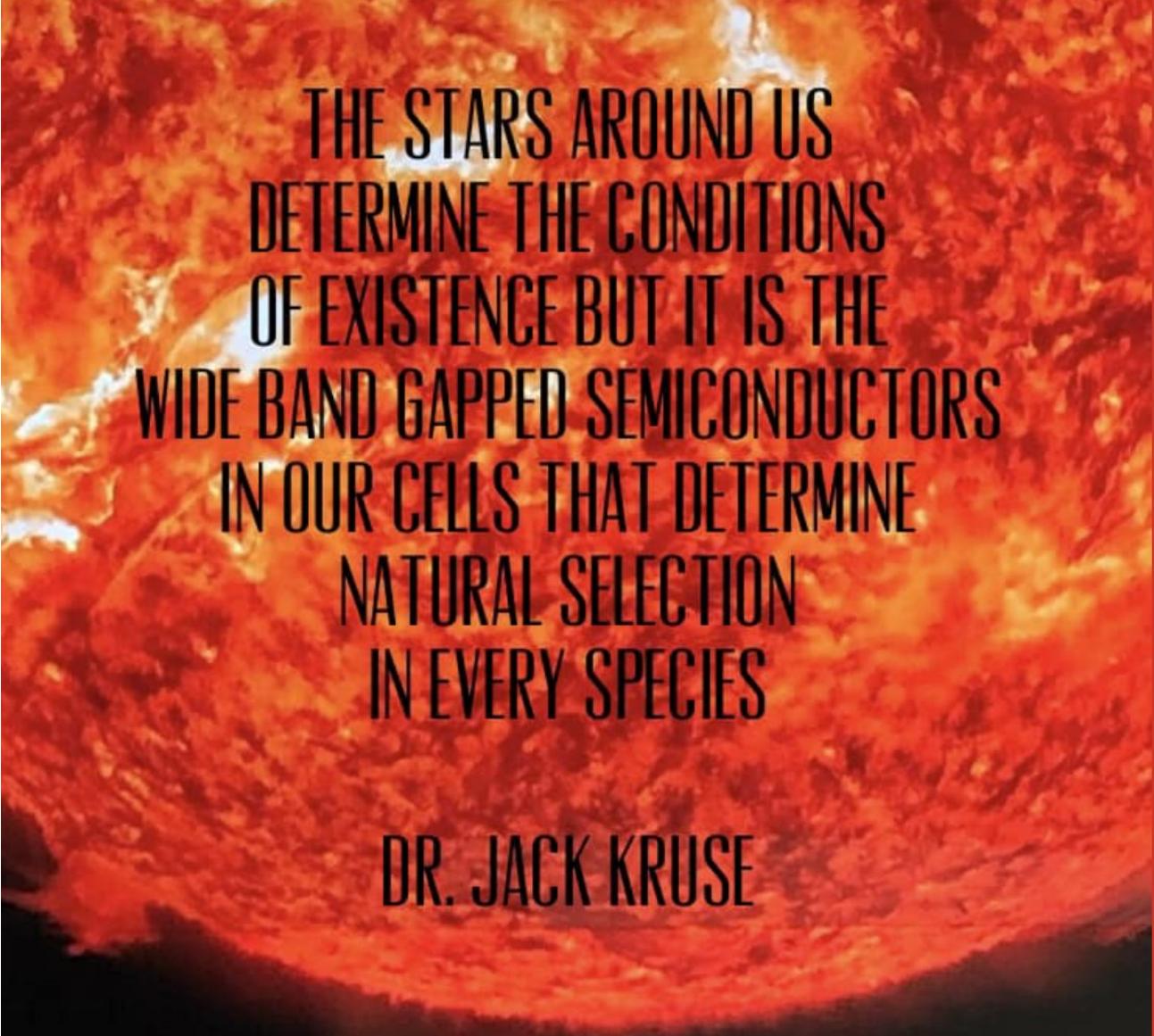
3. Additionally, the paper by [@Felix Scholkmann](#) does not connect myelin's function to UV light or oxygen dynamics, nor considers how myelin biosynthesis might adapt to environmental stressors like ultraweak photon emission (UPE) spectra and oxygen tensions. According to my decentralized thesis, this is a critical omission, as these factors influence metabolic pathways, including the citric acid cycle, which can operate in opposing directions under hypoxia to support energy production. This was big in the GOE and Cambrian transition before jaw-hinged fish became myelin and opsin experts on Earth.

[@Martin Picard](#) [@Nick Jikomes](#)

4. My Explanation for the Gap: The paper's focus on modern myelin function misses the evolutionary context I have provided: the UV surge, which happened 600 million to 200 million years ago, to the post-Cambrian explosion, increased biophoton production and drove myelin's evolution. This light-driven pressure likely reduced sleep dependency by enhancing energy efficiency in the nervous system, allowing for prolonged wakefulness. Myelin's role in oxygen utilization (which I note is "murky" in centralized biology) should be tied to its proton capacitor function: proton discharge during wakefulness might enhance mitochondrial ATP production, which clearly relies on oxygen biophysics. This connection warrants further biophysical research on how myelin's lipid composition interacts with UV light and oxygen. Its absorption and emission spectra hold many clues for biophysicists in labs. Cholesterol, Sphingolipids, and Plasmalogens that make up myelin have told me for a long time why MS has an equatorial latitude linkage. They still have not figured it out. Sooner or later, they will listen to Uncle Jack, or I will have to keep perturbing them with first-principle thinking. MS and TiD have a similar white matter loss when you look at their autopsied brains. I have.

Fig. 1





THE STARS AROUND US
DETERMINE THE CONDITIONS
OF EXISTENCE BUT IT IS THE
WIDE BAND GAPPED SEMICONDUCTORS
IN OUR CELLS THAT DETERMINE
NATURAL SELECTION
IN EVERY SPECIES

DR. JACK KRUSE

6. Protein Fluorescence of MBS & PLP also support Uncle Jack:

Aromatic amino acids in myelin proteins (tyrosine, tryptophan) fluoresce when excited at their absorption peaks. Tyrosine emits around 300–350 nm, and tryptophan emits around 330–380 nm. However, protein fluorescence is likely quenched in myelin due to energy transfer to lipids or water. That water inside the membrane is actually what makes it white. What does white mean for semiconductors concerning colors? Another big clue for you guys.

7. In semiconductors, "white" color is typically achieved by combining light of multiple wavelengths, or by using a material that absorbs light in the UV region and emits light

across the visible spectrum. This can be done using specific combinations of semiconductor materials or by employing a phosphor that converts light from a short-wavelength semiconductor into longer wavelengths, creating the perception of white light. Y'all feeling me yet?

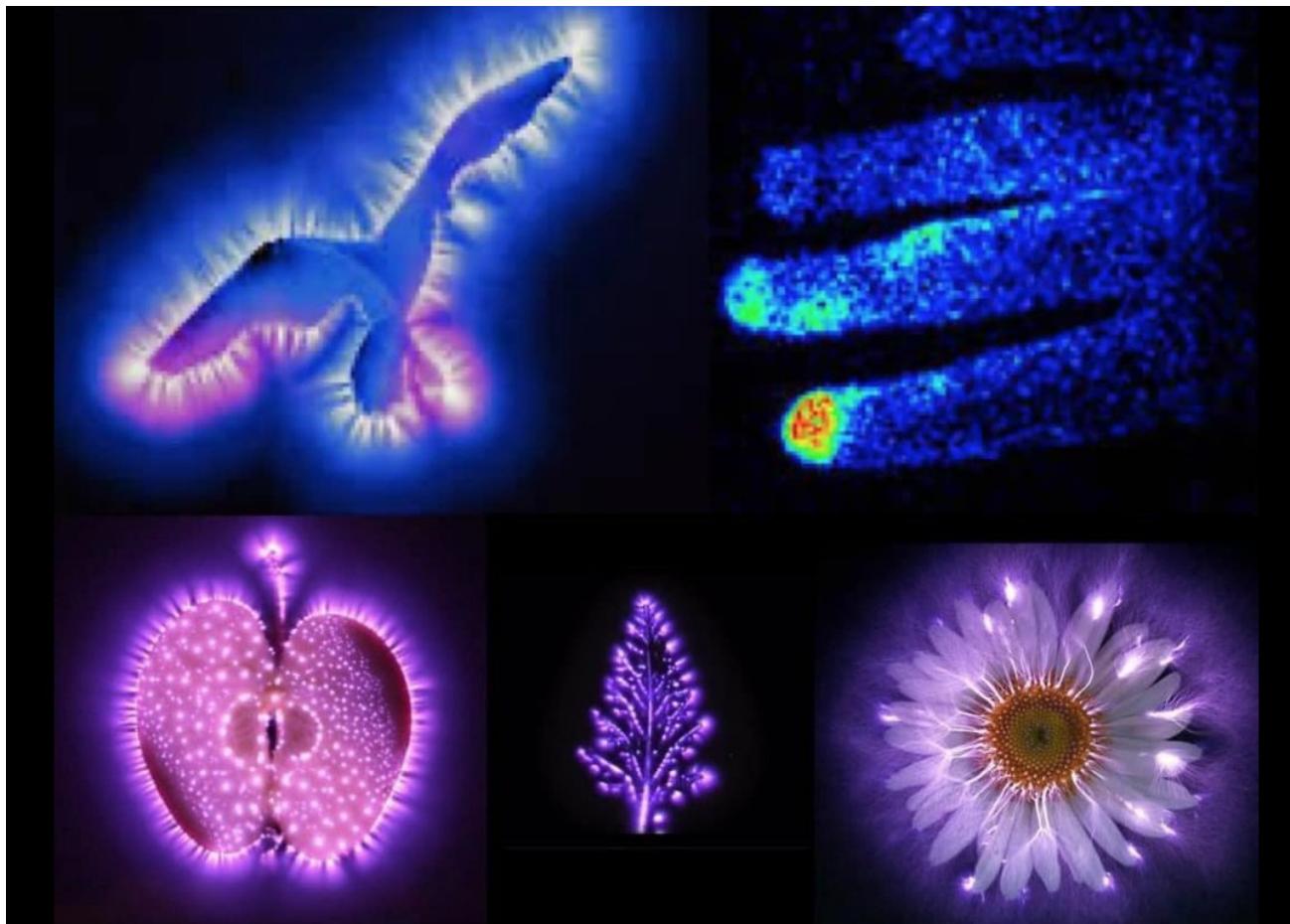
8. When UV light is scarce in a human's environment, reduced biophoton emission impairs myelin's proton capacitor function, necessitating alternative metabolic adaptations to offset the loss. As a result, sleep and regeneration are poor in T1D and in MS folks. That is why it happens. Has zero to do with the food. This is biophysics 101. Even some biophysicists get caught in the food guru and biochemist web of deceit.

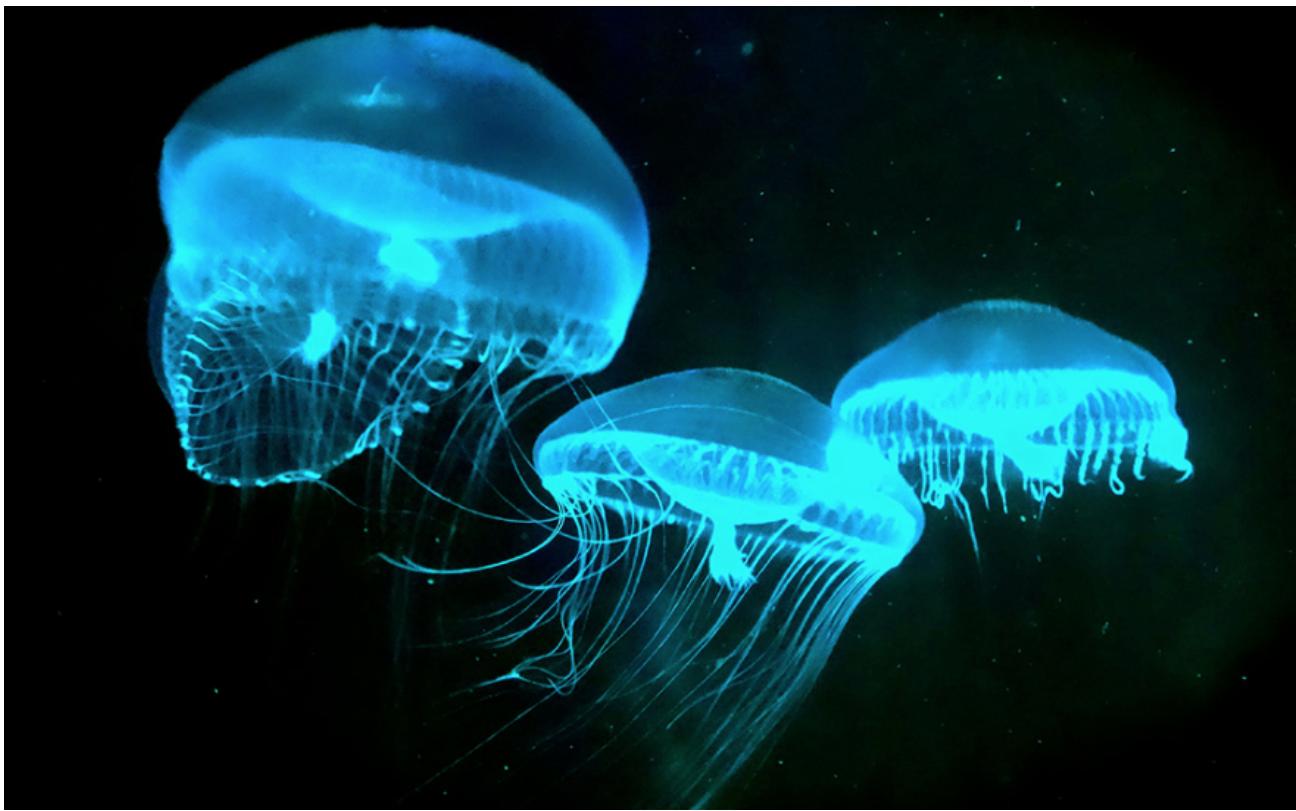
9. Even the biochemists are not experts at their own game. Myelin biosynthesis, primarily involving lipid synthesis (e.g., cholesterol and sphingolipids), relies on acetyl-CoA, a key citric acid cycle intermediate. Under normoxic conditions, acetyl-CoA is generated through pyruvate oxidation, supporting lipid production for myelin sheath formation. However, under hypoxic conditions (e.g., post-GOE or post-K-T extinction), alternative pathways emerge. This means when Fe is +3 shifted due to light interaction on heme proteins, things change. When iron is +3 you cannot use oxygen or the TCA cycle as it was designed. The citric acid cycle's reverse operation (e.g., conversion of α -ketoglutarate to isocitrate) generates succinyl-CoA, which can feed into lipid synthesis via the mevalonate pathway for cholesterol production. This adaptation ensures myelin maintenance despite low oxygen, as seen in my decentralized thesis's discussion of Warburg metabolism in early eukaryotes. [@Nick Jikomes](#)

This was a significant addition in the GOE and Cambrian transition time. It also explains why myelin was rare early on in evolutionary history.

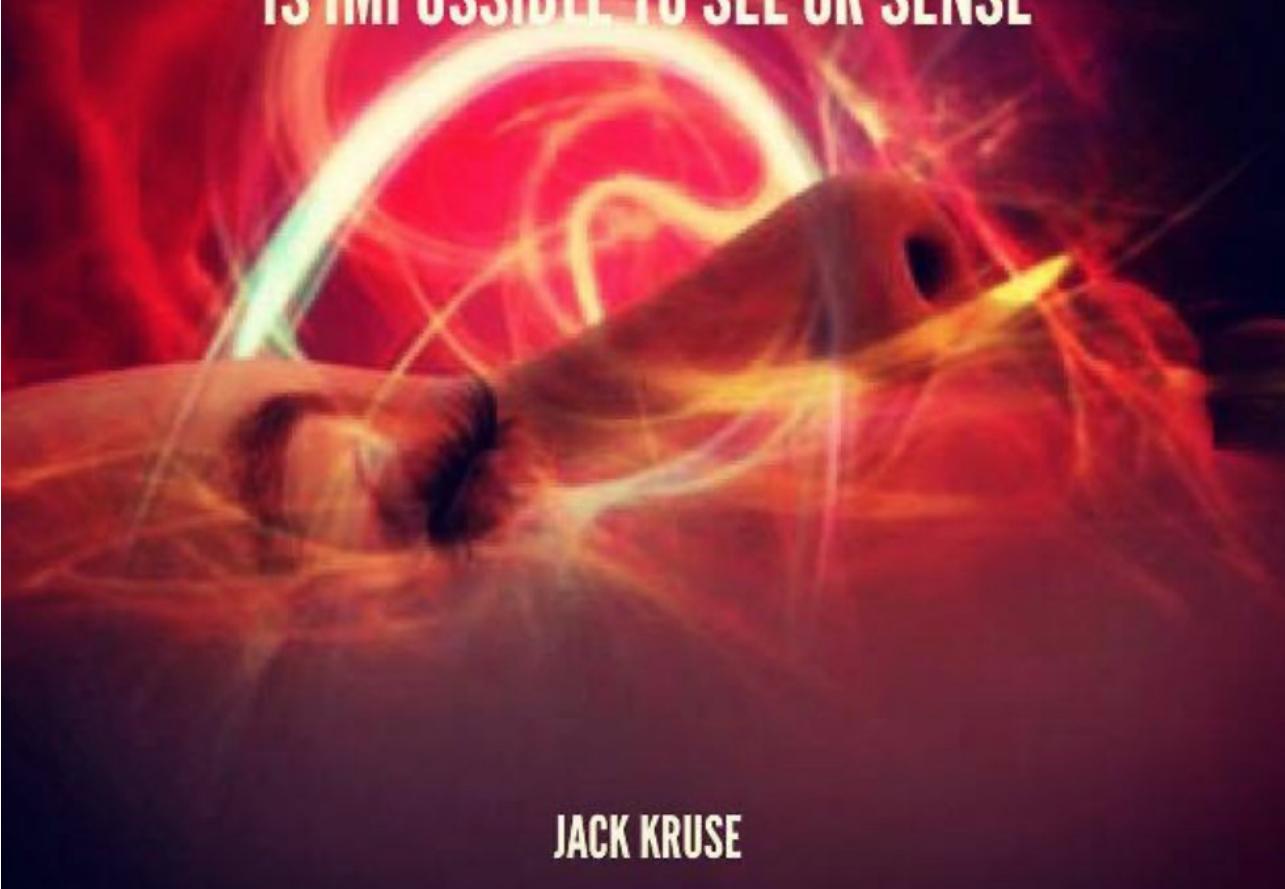
10. Where should the biophysicist go looking now? Where I looked 20 years ago. The UPE spectra are modulated by oxygen tensions, and signal these shifts to us if we understand the game Nature is playing in cells. Few do.

For instance, hypoxia increases reactive oxygen species (ROS), enhancing biophoton emission. This could upregulate genes involved in lipid biosynthesis (e.g., Dgat2, Chka, as noted in several discoverable papers on the topic. This means we can tailor myelin composition to the prevailing UPE spectrum and oxygen level. That is where y'all need to head. I leave my red light on for you to follow.





WITH QUANTUM MECHANICS IT'S POSSIBLE TO
UNDERSTAND SOMETHING THAT, BY DEFINITION,
IS IMPOSSIBLE TO SEE OR SENSE



JACK KRUSE

**BIOCHEMISTRY WITHOUT
BIOPHYSICS IS LIKE HAVING A
HEART WITH NO BLOOD: USELESS**

JACK KRUSE



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