Rx Take two aspirin and call me in the morning

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pFunny little email interchange with my brother, a doctor, yesterday. He sent me an excerpt from an article fromp the New England Journal of Medicine---he so hates opacity and pedantry, and this (rightly) seemed an example of that to him:

Inflammation is risky. Leukocytes recruited to fight microbes cause collateral damage that is often more severe than that originally triggered by the pathogen. Moreover, inflammation takes place even in patients with sterile tissue injuries such as trauma and ischemia–reperfusion.

The immune system recognizes mitochondria released from dying tissues as the bacteria they (the mitochondria) once were, and it mobilizes its destructive potential to limit their proliferation and arrest a mistaken invasion. This tragic "misunderstanding" could have a role in several human diseases, leading to inflammation in conditions as clinically diverse as post-traumatic systemic inflammatory response syndrome, myocardial infarction, cerebral ischemia, and systemic and organ autoimmunity.

Mitochondria are membrane-bound organelles that produce energy in virtually all eukaryotic cells. They have evolved from an endosymbiont alpha-proteobacterium (a relative of brucella and rickettsia). Mitochondria have their own DNA, enriched in hypomethylated CpG-containing sequences, which is duplicated when mitochondria divide. The origin of the eukaryotic cell is still controversial, and transitional forms between prokaryotes and eukaryotes have not been persuasively documented.3 pThe amalgamation of two prokaryotes or the amalgamation of a prokaryote with an eukaryotic precursor cell are possible scenarios. Regardless, the merger would have occurred long before the existence of an immune system, which by definition is a feature that is unique to multicellular organisms.

Zhang et al. detected mitochondrial DNA in the blood of patients with systemic inflammatory response syndrome after major trauma. Intravenous injection of mitochondrial proteins into mice resulted in activation of circulating neutrophils, with random extravasation in peripheral organs such as the liver and lung. Acute lung injury developed in these mice. Mitochondrial constituents also selectively activate an inflammasome, suggesting a possible link with other sterile inflammatory conditions such autoinflammatory diseases

Zhang et al. reason that, by virtue of their evolutionary origin, mitochondria might be recognized by pattern-recognition receptors and thus might initiate inflammation. This event seems unlikely to occur in healthy tissues, in which membrane-bound mitochondria are contained within cells

Mitochondrial structures released by injured cells possibly prompt inflammation during heart, kidney, or brain ischemia–reperfusion injuries, in which local neutrophil activation and further tissue damage occur when the blood flow is restored. Finally, mitochondria are probably released in patients with infectious disease — in whom substantial cell death takes place — possibly contributing to the molecular pathology of sepsis.

I gulped when I read it, pand then replied to him with this:

I feel I owe you an apology
For my molecular pathology.
I thought I might have hypochondria
Turned out it's just my mitochondria!
I felt so ill, weak and sedated…
But my CPG was hypomethylated!
Wouldn't a good strong antibiotic
Keep my cells eukaryotic?

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