

Relationship between intelligence and spectral characteristics of brain biophoton emission: Correlation does not automatically imply causation

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Despite enormous efforts, any correlation between “intelligence” and cognitive or physiological/anatomical properties of animal brains is still poorly understood because intelligence depends on multiple factors in parallel and not a single determinant (1). The study by Wang et al. (2) is both novel and intriguing, but we believe that the experimental results presented do not directly support their conclusion. Unfortunately, a mechanistic explanation of the correlation between intelligence and the spectral properties of biophotonic emission is not given in ref. 2, raising concerns that the observed correlation does not reflect a causal relationship but rather an accidental coincidence. Based on the reported spectral characteristics of the biophotonic emission (2) we calculated the coherence length (3), given as $l_c = \lambda_{avg}^2 / \Delta\lambda$, with $\Delta\lambda = \lambda_{max} - \lambda_{min}$ in Table 1.

The obtained values for the coherence lengths show that the biophotons in the human brain have the shortest coherence length (i.e., 1.893 μm) among other species (Fig. 1). This is in contradiction to the expectation that the longest coherence length would be favorable for efficient information processing from which a higher degree of intelligence could emerge.

Interestingly, we have found a strong correlation ($r = 0.86$) between the values of λ_{max} and the mass of each of the six species, indicating that intelligence may

simply correlate with size. Additionally, glutamate-induced biophotonic emission does not necessarily correlate with the change in aerobic metabolism. Reactive oxygen species (ROS) can be produced by neural mitochondria as well as by the NADPH oxidase (4, 5). NADPH oxidase can produce ROS whether mitochondrial cytochrome c oxidase is completely or incompletely blocked. Glutamate-induced ROS generation in neuronal presynaptic terminals is caused by the activation of the NADPH oxidase and nitric oxide synthases (6). NMDA receptor (NMDAR) activation by the glutamate increases NADPH oxidase activity, which is the key source of superoxide formation ($\text{O}_2^{\cdot-}$) (4). We conclude that the glutamate-induced NMDAR activation and NADPH oxidase activity can lead to overproduction of ROS that causes an increase of biophoton in the brain (7), which seems at odds with the reasoning behind the claims made by Wang et al. (2). Finally, protein phosphatase (PP) 2A comprises a family of serine/threonine phosphatases that play roles in cell-cycle regulation, cell morphology and development, and specific signal transduction (8). However, okadaic acid can also inhibit the activity of PP1, PP2A, PP4, PP5, and PP6 phosphatases, which is an underappreciated fact (9). Hence, the claim that the inhibition of PP2A induces the hyperphosphorylation of MAP tau and interferes with the function of microtubules is highly speculative.

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