LETTER

REPLY TO SALARI ET AL.:

Toward understanding the deep mechanisms regarding the biophotons related to human intelligence

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We would like to thank Salari et al. for their interest in our paper (1) and to give a response to their concerns on the causation of spectral redshift of biophotons related to human intelligence (2). First, it is inappropriate to consider a brain slice (or whole brain) as a single light source to calculate the coherence length because a brain slice contains different types of neurons. We have particularly emphasized in the discussion section in our paper that "the present imaging technique could not distinguish and determine what types of neurons emit what types of spectral biophotons or whether a neuron or type of neurons emit different spectral biophotons," which implies that we should classify the types of neurons in functional neural circuits if considering efficient biophotonic information processing. It is necessary to study the spectral features in functional neural circuits related to high intelligences, and we propose that it may be highly possible that a narrowband spectral characteristic besides the redshift of biophotons from animals to humans exists. Therefore, for example, if λ_{max} is 865 nm and λ_{min} is 765 nm in a functional neural circuit in human brain, then the coherence length (l_c) is ~6,642 μm, which is much greater than that (1,893 μm) calculated by Salari et al. Second, they have also found a strong correlation ($r = 0.86$) between the values of λ_{max} and the mass of each of the six species (2). We think such a correlation analysis may be meaningless without consideration of biomedical relations, and we suggest that it may be more important if there indeed exists a strong negative correlation between the values of λ_{max} and energy consumption (glucose) per unit of weight of brain tissue. Third, Salari et al. mentioned that the biophotonic emissions could be the results of glutamate-induced oxidative stress, which then generates various types of free radicals such as reactive oxygen species (ROS), and so on. We have addressed such a doubt in our previous paper (3) and also emphasized it in this paper (1), showing that biophotonic activities and transmission along neural fibers or in neural circuits induced by 50 mM glutamate are not due to the generation of free radicals. One of the key pieces of evidence is that the concentration of glutamate less than 12.5 mM is not possible to induce obvious biophotonic activities and transmission (3), whereas even much less than 12.5 mM glutamate would result in strong oxidative stress in primary cultured neurons in other studies (4). It was proposed in our previous study that the ultraweak intensity of biophotonic emissions due to the generation of free radicals may contribute to background biophotons (5), and we speculate that the maintenance of background biophotonic emissions may play a role in biophotonic energy storage in particular molecules such as proteins that may be involved in biophotonic signal transmission and encoding (action biophotons). Finally, protein phosphatase 2A (PP2A) is expressed in axons of neurons, and okadaic acid has been extensively studied as an inhibitor of PP2A to disturb the function of microtubules via hyperphosophorylation of Tau (6).

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The authors declare no conflict of interest.

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