Pareidolias in idiopathic RBD—an author response letter to the Editorial "What the 'man in the moon' can tell us about the future of our brains"

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In our original paper (1), we aimed to clarify the clinical significance of pareidolia in patients with idiopathic RBD. Prior to our work, Dr. Uchiyama and his coauthors from Professor Mori's research group from Department of Behavioral Neurology and Cognitive Neuroscience, Tohoku University School of Medicine established a practical and useful test, the pareidolia test, for evoking pareidolic response (2), which is a surrogate marker of visual hallucination. We conducted the test in idiopathic RBD patients, in whom more than half of them showed pareidolias. Following the comments to pareidolia in idiopathic RBD in the Editorial "What the 'man in the moon' can tell us about the future of our brains" from Professor Birgit Högl, we gratefully continue to discuss this topic, which further highlights the potential need to clarify the clinical benefit of the phenomenon in idiopathic RBD in terms of prediction of future conversion to Lewy body diseases and pathological course.

In research series reported by Professor Mori's research group, the pareidolia test that quantify pareidolias has been indicated to be beneficial to differentiate Lewy body diseases from other neurodegenerative dementing disorders such as Alzheimer's disease (2). Their studies demonstrated (I) pareidolias are more frequently observed in patients with dementia with Lewy bodies (DLB) in whom the number of pareidolias correlates with the severity of visual hallucination (3); (II) pareidolias are observed even in patients with DLB without visual hallucinations or those with Parkinson's disease without dementia, in whom the number of pareidolia are smaller than in DLB with visual hallucinations (4); and (III) cholinergic insufficiency or hypometabolism in posterior cortical region is common neural mechanism of pareidolia and visual hallucination (3-5).

In idiopathic RBD, more than half of patients showed pareidolia; the proportion was higher than the value of controls (1). Unfortunately, the number of pareidolia in the idiopathic RBD patients cannot be compared with those in DLB or PD without dementia in the prior studies because we used the ten scenery picture version of the pareidolia test although longer version of the test was used in the prior studies (25 scenery pictures). However, in the idiopathic RBD the rate of patients with one or more pareidolia was 53.5% (1), which was similar to the rate in PD without dementia without visual hallucination (57.1%) (4) and lower than those in DLB or PD without dementia with visual hallucination (100%) (2,4). The order of the occurrence rate of pareidolia corresponds to the fact that idiopathic RBD predates PD, DLB or PDD in the ascending disease pathway from brainstem to cortical region. Furthermore, the idiopathic RBD patients who gave pareidolic responses had a greater level of motor dysregulation during REM sleep, deteriorated sleep stability and lower cognitive function than those did not. These findings may suggest that iRBD patients with pareidolias represent a subgroup of

more advanced pathological status, namely closer to clinical Lewy body diseases, in the ascending neuropathological course than iRBD patients without pareidolias do.

In our prior study, decreased regional cerebral blood flow in the parieto-occipital lobe (precuneus), limbic lobe, and cerebellar hemispheres in patients with iRBD, which is commonly seen in patients with PD/DLB (6). In addition, the presence of RBD symptoms in PD is associated with relative neocortical, limbic cortical, and thalamic cholinergic denervation (7). Given these findings, in idiopathic RBD cerebral hypometabolism/hypoperfusion especially in posterior cortical region and cholinergic degeneration may underlie the mechanism of pareidolia.

In the recently updated diagnostic criteria of DLB, RBD was reassigned as one of core clinical features. Further, the Pareidolia task was also recommended to assess spatial and perceptual difficulties of DLB especially for perceptual discrimination (8). The specificity of the pareidolia test is limited; about 20% of controls showed pareidolic response, because pareidolic responses in the scenery version of the test rely on subjects' description. Besides, given the association with deteriorated sleep stability, which may suggest an involvement of brainstem reticular formation regulating arousal/awakening, pareidolia can be observed in other sleep-wake disorders such as narcolepsy. Although the future outcome cannot be concluded from this cross-sectional study, pareidolia could possibly predict the future development of Lewy body diseases, especially for people who experience visual hallucinations earlier. Future prospective studies are warranted to confirm this hypothesis, particularly addressing clinical manifestations of visual hallucinations and brain imaging findings.

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Footnote

Conflicts of Interest: The author has no conflicts of interest to declare.

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