

Commentary

Sensitivity to light in bipolar disorder: implications for research and clinical practice: commentary, Terao

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Keywords

Light; bipolar disorder; depression; bright light therapy; dark therapy.

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Response

Dr Roguski and colleagues' editorial¹ raises important questions about sensitivity to light in bipolar disorder, but there are several confusions. First, the authors claimed that indirect clinical evidence supporting light hypersensitivity in bipolar disorder comes from the recognized efficacy of chronotherapies such as bright-light therapy for bipolar depression and blue-light blocking glasses (or 'dark therapies') for mania. Simply speaking, however, bright-light therapy increases the amount of light and dark therapy decreases the amount of light, both of which are independent of sensitivity to light in bipolar disorder. Even if bipolar patients have normal sensitivity to light, they can obtain benefits from both treatments, which does not support the light hypersensitivity hypothesis in bipolar disorder.

Second, Dr Roguski and colleagues¹ provided experimental evidence that melatonin suppression did not differ between bipolar disorder and control groups,² and insisted that pathways from intrinsically photosensitive retinal ganglion cells (ipRGC) to the suprachiasmatic nucleus (SCN) for melatonin synthesis are intact and sufficient for entraining circadian rhythms, and that light is in itself capable of influencing mood states. However, there is another projection from ipRGC to the perihabenular nucleus (PHb), which is completely independent of SCN and drives the light-mediated mood alterations.³ That is, ipRGCs that project to the SCN mediate the effects of light on circadian rhythms and learning, whereas mood regulation by light requires an SCN-independent pathway linking ipRGCs to PHb.³ The involvement of the PHb in bipolar disorder may be associated with sensitivity to light, which should be clarified in further studies.

Third, Dr Roguski and colleagues¹ indicated a possibility that light is in itself capable of influencing mood states. Hirakawa and colleagues^{4,5} conducted a randomised controlled trial with a 4-week period of bright light (10 000 lx) exposure or dim light (50 lx) exposure in healthy participants⁴ and in patients with mood disorders.⁵ Both studies showed a significantly increased volume of the head of the left dentate gyrus in the bright light exposure group but not in the dim light exposure group.^{4,5} Particularly in patients with mood disorder, there was a positive correlation between the percentage change in the volume of the whole left dentate gyrus and the percentage change in the scores of the mood visual analogue scale.⁵ These findings suggest a possibility of neurogenesis induced by bright light in the left dentate gyrus. Although this possibility is yet to be determined and should be cautiously investigated in further studies, if present, it may be applied not only to individuals with mood disorders including bipolar disorder, but also to the general population. Notably, the amount of light may be inversely associated with suicide in the general population.⁶

Fourth, a 2-year follow-up study⁷ consisting of 172 bipolar patients (35% of which were people with bipolar I disorder) shows that manic/hypomanic episode relapses were significantly higher when the average nighttime illuminance was ≥ 3 lux than when it was < 3 lux, whereas the association between nighttime light and depressive episode relapses was not significant. These findings suggest that hypersensitivity to light may be associated with manic/hypomanic relapses of bipolar disorder, supporting the hypersensitivity hypothesis in bipolar disorder.

Finally, I agree with the authors' conclusion that a light hypersensitivity perspective of bipolar disorder is an exciting focus for future research, although sensitivity to light and the amount of light should be differentiated.

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Declaration of interest

None.

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