Association between living with others and depressive symptoms in Japanese hospital workers during the COVID-19 pandemic

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This Letter presents the findings of a cross-sectional study on the association between living with others and depressive symptoms among 1228 workers, aged 21–73 years, from a large hospital and its affiliated institute in Tokyo, 66.8% of whom had engaged in some sort of COVID-19-related work. The Introduction, Methods, Results, Discussion, and Tables are presented as an online supplement (Appendix S1).

The COVID-19 pandemic is having a particularly significant psychological impact on health-care workers, with 25% reported to be depressed during the pandemic.1 Health-care workers are not only at higher risk of exposure to SARS-CoV-2 and increased workloads,2 but also social isolation and rejection due to the high probability that they will come into contact with potentially infectious COVID-19 patients.3

Social support has been recognized as a protective factor for mental health among health-care workers during the COVID-19 pandemic.4 However, social restrictions led to reduced access to support from family and friends, and degrade social support systems, which can cause loneliness and depressed mood.5 In particular, individuals who live alone may decrease with increasing number of cohabitants. To our knowledge, this is one of only a few studies to have investigated the association between living with others and depressive symptoms in hospital workers during the COVID-19 pandemic.

Our findings agree with those of a meta-analysis of observational studies among the elderly, which indicated that older people living alone have a higher risk of depression than those living with others.6 In a cross-sectional study conducted in China during the COVID-19 pandemic, medical staff living alone reported significantly higher depressive symptoms than those living with others.7 We confirmed that living with others is associated with the mental health of hospital workers during the COVID-19 pandemic, even after adjustment for sleep and mood-related factors. The present study is limited due to its cross-sectional design and lack of detailed information on family members/cohabitants. Further studies are required to address these issues.

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Disclosure statement
The authors declare no conflicts of interest.

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Sleepiness is considered not to be unidimensional. The International Classification of Sleep Disorders, 3rd edition (ICSD-3) employs two criteria for ‘pathological sleepiness’ for idiopathic hypersomnia: (i) sleep prolongation with a 24-h total sleep time (TST) $\geq$ 660 min, measured either by 24-h polysomnography (24-h PSG) or by wrist-actigraphy-based sleep time averaged for at least 7 days; and high sleep propensity with a mean sleep latency (mSL) of $\leq$ 8 min on the Multiple Sleep Latency Test (MSLT).¹ The MSLT evaluates the tendency to fall asleep during daytime nap opportunities and serves as the gold standard for the diagnosis of central disorders of hypersomnolence. However, recent studies indicate that the MSLT is inadequate to delineate hypersomnia other than narcolepsy type 1.²-⁴ Although several attempts using continuous PSG monitoring have been performed,⁵-⁷ appropriate markers for idiopathic hypersomnia have not been established.⁸ We performed 24-h PSG, standard PSG, and MSLT to understand the difference between the two aspects of sleepiness. This study was approved by the Ethics Committees of the Institute of Neuropsychiatry and Tokyo Metropolitan Institute of Medical Science. All patients gave written informed consent. Forty consecutive patients visiting Seiwa Hospital with suspected idiopathic hypersomnia with long sleep time were evaluated by 3-day sleep studies – unattended 24-h PSG, followed by PSG and MSLT – from January 2017 to June 2019. Clinical and PSG variables from 35 eligible patients were compared to search for markers of pathological sleepiness. We next searched for markers characteristic of patients with sleep prolongation or high sleep propensity. There were no differences in demographic data, self-reported measures, or clinical symptoms except for higher percentage of ‘always unrefreshed nap’ in those with sleep prolongation and higher percentage of ‘experience of sleep attack’ and lower percentage of ‘long nap’ in those with high sleep propensity (Table S1). As expected, we confirmed shorter MSLT mSL in the high-sleep-propensity group and longer 24-h PSG TST in the sleep-prolongation group (Table 1). No conventional PSG variables predicted sleep prolongation. Some sleep variables on 24-h PSG were identified as possible markers for sleep prolongation: shortened REM latency ($P = 0.026$), lower 24-h PSG_N3 (%TST; $P = 0.020$), more non rapid eye movement (NREM)-REM cycle counts ($P = 0.0002$), and shorter NREM-REM cycle duration ($P = 0.046$). Binary logistic regression analyses confirmed that a symptom of ‘always unrefreshed upon waking’ (odds ratio [OR] 44.1, $P = 0.021$), 24-h PSG REM latency (OR 1.009, $P = 0.027$), and 24-h PSG NREM-REM cycle duration (OR 1.07, $P = 0.06$) were independent predictors of pathological sleep prolongation. Similar analyses revealed that a symptom of ‘experience of sleep attack’ was independently associated with high sleep propensity (OR 0.11, $P = 0.025$). (See Table S2. Detailed description for Table 1 and S2 are provided in Supplementary Information.) Twenty-five of the 35 patients fulfilled the ICSD-3 criteria for idiopathic hypersomnia, two with narcolepsy type 2, two with pathological sleepiness without a diagnosis (sleep prolongation with multiple sleep-onset REM periods [SOREMP]), and six with non-hypersomnia. The sensitivity, specificity, and accuracy of two tests for the diagnosis of ICSD-3-defined idiopathic hypersomnia were calculated. Test sensitivity was 12% with MSLT and 92% with 24-h PSG, test specificity was 80% and 60%, and accuracy was 34% and 83%, respectively (Table S3). The low sensitivity and accuracy of MSLT may be partly due to the sampling bias because we performed 24-h PSG only for those with habitually long self-reported sleep time. However, our results indicated that 79% (23/29) of our patients with pathological sleepiness would be overlooked if they were evaluated with MSLT alone, replicating that idiopathic hypersomnia patients often fail to show high sleep propensity.¹, ⁶, ⁹, ¹⁰ Although the presence of multiple SOREMP reflects the pathophysiology of narcolepsy, there is no evidence that their absence is related to the pathophysiology of idiopathic hypersomnia. In this study, four of 27 (14.8%) patients with pathological sleep prolongation showed multiple SOREMP on MSLT. Further studies with larger sample sizes are required to clarify the significance of SOREMP and other REM abnormalities in those with sleep prolongation. (REM abnormality and limitations of this study are described in detail in Supplementary Information.) Our study indicates that the two aspects of sleepiness, sleep prolongation and high sleep propensity, are fundamentally different, and that 24-h PSG should be used as a first-line diagnostic tool for idiopathic hypersomnia with long sleep time.

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