Brain, Behavior, and Immunity 56 (2016) 413

Contents lists available at ScienceDirect

Brain, Behavior, and Immunity

journal homepage: www.elsevier.com/locate/ybrbi

Letter to the Editor

Toward a mechanistic understanding of psychosocial factors in telomere degradation

We welcome Tempaku et al.'s insights regarding sleep deprivation as a systemic stressor and as a contributor to telomere degradation. Here, we provide some concrete recommendations to inform future research designs that could begin to parse the complex mechanisms by which sleep and similar factors could affect the stress-telomere length relationship.

The causal dynamic of sleep deprivation with stress, telomere length, and age is likely quite complex. For example, sleep deprivation and stress may be mutually causal: sleep deprivation appears to cause systemic stress (as Tempaku et al. describe); in addition, stress also causes sleep disturbances (Cano et al., 2008). We caution, therefore, that sleep deprivation cannot be modeled as a simple confounder. Instead, sleep deprivation and stress may act as mutual mediators in a potential stress-telomere length pathway. Such complex mechanisms are not limited to sleep deprivation: as mentioned in our meta-analysis, psychiatric comorbidities (such as clinical depression) and behavioral factors (such as smoking and physical activity) may act both as confounders and as mediators.

Parsing such mechanistic possibilities is integral to an improved scientific and practical understanding of telomere degradation, but will be possible only if future research moves beyond currently predominating cross-sectional designs. Therefore, we emphatically echo Tempaku et al.'s (2015) call for longitudinal studies using repeated measurement of stress, sleep deprivation, and variables relevant to other suspected causal pathways. For example, although current cross-sectional data (such as those analyzed in our meta-analysis) do not permit valid statistical estimation of mediation effects of sleep deprivation, longitudinal designs would enable such modeling (Vanderweele, 2015).

Additionally, using meta-analytic methods to address mechanistic roles of sleep and similar factors is contingent upon more robust data sharing in this field at the level of individual subject data. In our meta-analysis, we were able to acquire only aggregate effect sizes, rather than raw data, from most eligible studies, largely precluding analysis of mechanistic, rather than associative, hypotheses. Within limitations of confidentiality, a progressive shift toward routine data sharing via platforms such as the Open Science Framework would open the door to a more nuanced causal understanding of psychosocial determinants of telomere biology. Such shifts toward public data sharing are underway throughout many social science fields (Nosek et al., 2015; Eich, 2014).

In summary, to enable principled modeling of effects of sleep deprivation and other similarly complex variables, we urge our colleagues in the field to begin moving toward longitudinal designs and sharing data at the individual subject level.

References

Cano, G., Mochizuki, T., Saper, C.B., 2008. Neural circuitry of stress-induced insomnia in rats. J. Neurosci. 28 (40), 10167–10184.

Eich, E., 2014. Business not as usual. Psychol. Sci. 25 (1), 3–6.

Nosek, B.A., Alter, G., Banks, G.C., Borsboom, D., Bowman, S.D., Breckler, S.J., et al, 2015. Promoting an open research culture: author guidelines for journals could help to promote transparency, openness, and reproducibility. Science (New York, NY) 348 (6242), 1422.

Tempaku, P.F., Mazzotti, D.R., Tufik, S., 2015. Telomere length as a marker of sleep loss and sleep disturbances: a potential link between sleep and cellular senescence. Sleep Med. 16 (5), 559–563.

Vanderweele, T., 2015. Explanation in Causal Inference: Methods for Mediation and Interaction. Oxford University Press.

Maya B. Mathur *
Quantitative Sciences Unit, Stanford University, Palo Alto, CA, USA
Department of Biostatistics, Harvard University, Boston, MA, USA
* Address: Quantitative Sciences Unit, c/o Inna Sayfer, Stanford University, 1070 Arastradero Rd., Palo Alto, CA, 94305, USA.
E-mail address: mmathur@stanford.edu

Elissa Epel
Department of Psychiatry, University of California, San Francisco, CA, USA

Shelley Kind
Department of Psychology, Suffolk University, Boston, MA, USA

Manisha Desai
Quantitative Sciences Unit, Stanford University, Palo Alto, CA, USA

Christine G. Parks
Epidemiology Branch, National Institute of Environmental Health Sciences, NIH, Research Triangle Park, NC, USA

Dale P. Sandler
Epidemiology Branch, National Institute of Environmental Health Sciences, NIH, Research Triangle Park, NC, USA

Nayer Khazeni
Division of Pulmonary and Critical Care Medicine, Stanford University, Stanford, CA, USA
Center for Health Policy and Center for Primary Care and Outcomes Research, Stanford University, Stanford, CA, USA

Available online 23 April 2016

http://dx.doi.org/10.1016/j.bbi.2016.04.011
0889-1591/