Poor Sleep Health Could Contribute to Inflammatory Disease

Reports new study in Biological Psychiatry

Philadelphia, PA, July 6, 2016 – A new meta-analysis in Biological Psychiatry reports that sleep disturbances and long sleep duration are associated with increases in markers of inflammation.

“It is important to highlight that both too much and too little sleep appears to be associated with inflammation, a process that contributes to depression as well as many medical illnesses,” said Dr. John Krystal, Editor of Biological Psychiatry.

Insufficient sleep is considered a public health epidemic by the Centers for Disease Control and Prevention. Common sleep disturbances, such as insomnia, have been associated with increased risk of inflammatory disease and mortality.

Substances that increase in response to inflammation and circulate in the blood stream, such as C-reactive protein (CRP) and interleukin-6 (IL-6), predict adverse health conditions including cardiovascular events, hypertension, and type 2 diabetes. Many studies have investigated the mechanism behind the association between sleep health and immunity, but variations between studies have made it difficult to understand the effects.

In a recent article, Michael Irwin, Richard Olmstead and Judith Carroll, all of the Cousins Center for Psychoneuroimmunology, UCLA Semel Institute for Neuroscience, University of California, Los Angeles, systematically reviewed existing studies for associations between sleep and inflammatory markers. The meta-analysis examined 72 different articles, which included over 50,000 participants from population-based and clinical studies. The researchers looked at CRP, IL-6, and tumor necrosis factor α (TNFα) as indicators of inflammation.

People with a normal sleep duration get 7–8 hours of shut-eye per night. The analysis showed that sleep disturbance (poor sleep quality or complaints of insomnia) and long sleep duration (more than 8 hours) were associated with increased levels of CRP and IL-6. Shorter sleep duration was associated with increased levels of CRP. No associations were found with TNFα.

According to Irwin, sleep disturbance or insomnia should be regarded as behavioral risk factors for inflammation, similar to the adverse effects of high fat diet or sedentary behavior. Treatments targeting sleep behavior could be a strategy for reversing the inflammation and reducing risk of inflammatory illnesses.

“Together with diet and physical activity, sleep health represents a third component in the promotion of health-span,” said Irwin.
Notes for editors

Copies of this paper are available to credentialed journalists upon request; please contact Rhiannon Bugno at +1 214 648 0880 or biol.psych@utsouthwestern.edu. Journalists wishing to interview the authors may contact Michael Irwin at mirwin1@ucla.edu.

The authors’ affiliations, and disclosures of financial and conflicts of interests are available in the article.

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The journal publishes novel results of original research which represent an important new lead or significant impact on the field, particularly those addressing genetic and environmental risk factors, neural circuitry and neurochemistry, and important new therapeutic approaches. Reviews and commentaries that focus on topics of current research and interest are also encouraged.

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