
Poor Sleep Linked to Inflammation, a Risk Factor For Heart Disease and Stroke



US researchers found that poor sleep quality is linked to higher levels of inflammation, a known risk factor for heart disease and stroke. Dr Alanna Morris, a cardiology fellow at Emory University School of Medicine in Atlanta, Georgia, presented the findings of the community study at the American Heart Association (AHA) 2010 Scientific Sessions in Chicago. The study is part of the Emory-Morehouse Partnership to Reduce CV Disparities (META-Health), a joint initiative between Emory and Morehouse School of Medicine, also in Atlanta.

Morris said other studies have already shown a link between acute lack of sleep and inflammation markers and changes in blood vessels, but there is not enough research information on the physiological effects of chronic lack of sleep. Morris and colleagues examined data on 525 middle-aged participants of the META-Health study who had filled in the Pittsburgh Sleep Quality Index (PSQI) questionnaire. The PSQI asked the participants detailed questions about sleep duration and sleep quality.

After adjusting the results to take into account potential demographic (age, gender, race) and health (smoking, diabetes, blood pressure, glucose, waist size, blood fat levels) confounders, the researchers found that:

- Participants with poor sleep quality had significantly higher levels of fibrinogen, IL-6, and CRP than participants with good sleep quality.
- Levels of the three inflammatory markers also differed across the three categories of sleep duration.
- Sleep duration of 6 to 8.9 hours was linked to significantly lower levels of mean fibrinogen, median IL-6 and CRP compared to sleep duration of under 6 hours.
- Comparisons between 6 to 8.9 and nine hours or more of sleep duration did not show any statistically significant effects.

The researchers concluded that:

Poor sleep quality, and short sleep durations are associated with higher levels of inflammation. They suggested that improving sleep quality and duration may be an appropriate therapeutic target for reducing cardiovascular disease risks.

Morris said that although the increased levels of C-reactive protein seen in those participants who got little sleep were within the range that health authorities would describe as low to intermediate cardiovascular disease risk, this study looked at community-based participants, as opposed to hospital patients with known cardiovascular disease, "so they have overall lower risk and lower C-reactive protein levels than many of the high risk populations in other studies". Inflammation could be a route through which poor sleep quality increases the risk of heart disease and stroke, said Morris. But she pointed out that it is still not clear whether short sleep duration contributes directly to cardiovascular mortality or whether it is a mediating factor.

Morris and colleagues are also reporting to the conference the results of a separate study that looked at the difference between men and women in terms of links between sleep quality and artery hardening or stiffness, where lack of flexibility in the vessel wall causes high blood pressure and makes the heart work harder. Those results showed that while poor sleep was linked to higher blood pressure in both men and women, the link between poor sleep quality and arterial stiffness was only significant for men.

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