

Study of Whitehall civil servants explains how stress at work is linked to heart disease

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New research has produced strong evidence of how work stress is linked to the biological mechanisms involved in the onset of heart disease.

Published in Europe's leading cardiology journal, the *European Heart Journal* today (Wednesday 23 January), the research is the first large-scale study to look at the cardiovascular mechanisms of work stress in the population and provides the strongest evidence yet of the way it can lead to coronary heart disease (CHD), either directly, by activating stress pathways controlled by the interaction between the nervous system, the endocrine glands and their hormones (neuroendocrine mechanisms), or indirectly via its association with unhealthy lifestyles.

The research is part of the long-running Whitehall II study, which has been following 10,308 London-based civil servants since 1985, and which is led by Sir Michael Marmot, professor of epidemiology and public health at University College London, UK.

Dr Tarani Chandola, a senior lecturer in UCL's Department of Epidemiology and Public Health, the first author of this EHJ study, said: "Stress at work is associated with an increased risk of coronary heart disease but the mechanisms underlying this association have remained unclear until now.

"This study addressed three questions: 1) Is the accumulation of work stress associated with higher risks of incident CHD and risk factors" 2) Is this association stronger among working-age populations" 3) Does

work stress affect CHD directly through neuroendocrine mechanisms, or indirectly through behavioural risk factors for CHD, or both”

The researchers collected evidence on the incidence of CHD, deaths from CHD, non-fatal myocardial infarctions, angina, heart rate variability, morning rises in the levels of the “stress” hormone cortisol, the metabolic syndrome and behavioural risk factors such as diet, exercise, smoking and drinking.

“During 12 years of follow-up, we found that chronic work stress was associated with CHD and this association was stronger among both men and women aged under 50 – their risk of CHD was an average of 68% more than for people who reported no stress at work. Among people of retirement age (and therefore less likely to be exposed to work stress), the effect on CHD was less strong.”

Dr Chandola said the most important new finding was the evidence linking work stress with the biological mechanisms underlying CHD. The autonomic nervous system (ANS) is the part of the nervous system that regulates involuntary actions, such as the action of the heart, and it has a central role in the neuroendocrine stress responses. The signals that are sent to the heart by the vagus nerve, telling it how to work and controlling the variability of the heart rate, are mediated by the ANS. The researchers found that workers who suffered from greater stress were more likely to have lowered heart rate variability and poor vagal tone. They also found that the hypothalamic-pituitary-adrenal axis (HPA) – a major part of the neuroendocrine system – was disturbed by greater stress, and this was shown by the fact that stressed workers had higher than normal morning levels of cortisol. These results were independent of the workers’ health behaviours.

“Adjusting for health behaviours did not change the association between work stress and low heart rate variability, suggesting a direct effect on

the ANS and neuroendocrine function, rather than indirect effects through health behaviours,” said Dr Chandola. “The effect on the ANS and neuroendocrine function in turn affects the signals to the heart, leading to cardiac instability.”

The researchers also found work stress was associated with poor health behaviours that could lead indirectly to CHD. “There have been relatively few studies that have found an association between work stress and unhealthy behaviours. Work stress is associated with a poorer diet in terms of eating less fruit and vegetables, and less exercise. It has also been linked to problem drinking, although not in this study. In this study, around 32% of the effect of work stress on CHD could be explained by its effect on health behaviours and the metabolic syndrome,” he said.

He concluded: “This study demonstrates that cumulative stress at work can lead to CHD through direct activation of neuroendocrine stress pathways and indirectly through unhealthy behaviours.”

Source: European Society of Cardiology

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