While work stress has been linked to coronary heart disease in both retrospective and prospective studies, the underlying biological mechanisms remain unclear. Now, researchers have found that repeated stress at work is an important risk factor for the development of metabolic syndrome.

A study provided evidence for the biologically plausible link between psychosocial stressors from everyday life and heart disease, according to Tarani Chandola, DPhil, senior lecturer in the department of epidemiology and public health, at the University College London. Dr. Chandola and colleagues reported their results in the British Medical Journal.

A prospective cohort study identified the association between work stress and metabolic syndrome. Researchers evaluated 10,308 men and women, aged 35 to 55 years, who employed in 20 London civil service departments at baseline (Whitehall II study). Follow-up was an average of 14 years.

The main exposure measure of the study was work stress based on the iso-strain model. “The iso-strain model is one of several variants of this concept of work stress; this model hypothesizes that socially isolated (no supportive coworkers or supervisors) high job strain carries the highest risk for heart disease,” wrote Dr. Chandola.

The other outcome was the metabolic syndrome based on the National Cholesterol Education Program definition. The concept of metabolic syndrome has recently come under fire — as well as the measure of its individual component. See article, “Metabolic syndrome: Misleading Diagnosis or Valid Condition?,” November/December 2005 issue, pages 13 to 15.

Characteristics of metabolic syndrome include abdominal obesity, atherogenic dyslipidemia, hypertension, insulin resistance and prothrombotic and proinflammatory states. The presence of three or more risk factors indicates the diagnosis.

The investigators measured the accumulation of exposure to work stress by adding together the number of times the participant was exposed to iso-strain. They defined chronic stress as experiencing iso-strain three or more times over the 14-year study.

The investigators also measured the participants’ social position.

A dose-response relation was found between exposure to work stressors over 14 years and the risk of developing metabolic syndrome, independent of other relevant risk factors, Dr. Chandola wrote. Employees who reported chronic work stress were more than twice as likely to have metabolic syndrome than those without work stress (odds ratio adjusted for age and employment grade 2.25, 95% CI 1.31-3.85).

Participants in the lowest employment grades had more than double the risk of developing metabolic syndrome than those in the highest grades. After adjusting for work stress and health behavior, however, the social gradient was no longer significant.

Dr. Chandola and colleagues wrote that prolonged exposure to work stress may affect the autonomic nervous system and neuroendocrine activity directly, contributing to the development of metabolic syndrome. A case-controlled study showed that participants in Whitehall II with metabolic syndrome had raised cortisol and normetanephrine output, as well as reduced heart rate variability.

Psychobiological findings have revealed that heightened stress reactivity and impaired recovery following stress, assessed by blood pressure as well as inflammatory markers predict the 5-year progression of metabolic syndrome. The researchers postulated that chronic stress may reduce biological resilience and disturb homeostasis. It is also possible that altered adrenocortical function may influence hepatic lipoprotein metabolism and insulin sensitivity at target organs.

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