

# Job Stress and Stroke and Coronary Heart Disease

JMAJ 47(5): 222–226, 2004

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**Abstract:** Repetitive or long-lasting effects of work stressors cause a type of exhaustion referred to as “accumulated fatigue,” that may eventually cause ischemic heart disease or stroke. Among the various work stressors to which people may be exposed, long work hours combined with lack of sleep is a major risk factor in our society. Irregular work hours, shift work, frequent work-related trips, working in a cold or noisy environment, and jet lag are also potent risk factors for workers. In addition, the chronic effects of psychological job strain, which can be conceptualized by the job demand-control-support model, are related to cardiovascular disease. In this model, high job demand and low work control accompanied by low social support at work are the most harmful to health. However, the biomedical mechanisms connecting psychological job strain to cardiovascular disease remain to be fully clarified.

**Key words:** Job stress; Cardiovascular disease; Long hour work; Job strain

## The Concept of Work Stress

The Occupational Stress Model<sup>1)</sup> of National Institute of Occupational Safety and Health (NIOSH) is shown in Fig. 1 to facilitate understanding of the concept of work stress and its effect.

Stressors that constantly affect our health exist in all types of work. By interacting with personal factors or non-workplace factors, these work-related stressors induce what are called

‘stress reactions’. There are various stress reactions, including psychological responses (depression and dissatisfaction at work), physiological responses (blood pressure elevation and increased heart rate), and behavioral responses (overeating, overdrinking, smoking, drug use, sickness absence, and accidents). Since the responses are usually acute and transient, health can be restored by taking breaks, resting, sleep, or other adequate approaches.

When exposed to a powerful stressor for a

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This article is a revised English version of a paper originally published in the Journal of the Japan Medical Association (Vol. 128, No. 6, 2002, pages 885–888). The Japanese text is a transcript of a lecture originally aired on May 28, 2002, by the Nihon Shortwave Broadcasting Co., Ltd., in its regular program “Special Course in Medicine”.

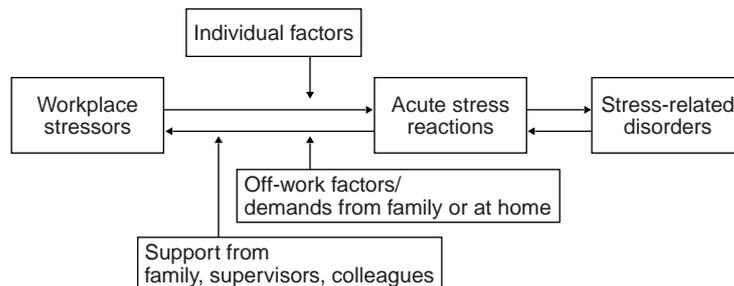


Fig. 1 NIOSH occupational stress model

long period, however, the stress responses increase and become excessive, making it difficult to restore health. This condition is generally called ‘accumulation of fatigue’, and when it persists for a long time, irreversible changes, such as poor physical function and the development of vascular disease, may occur. It may also induce stroke and coronary heart disease in combination with other factors.

Thus, from the standpoint of occupational health, it is important to focus on high-risk, work-related stressors in order to investigate associations between job stress and stroke and coronary heart disease. Stress responses vary with age, gender, personality, behavior and attitude toward work, work skill, underlying diseases, and treatment status. Other factors, such as relationships with supervisors or colleagues at work, regulations and atmosphere in the workplace, daily life outside work, and family relationships and role at home, modify stress responses, and they also need to be examined.

### Work-Related Stressors that Increase the Risk of Stroke and Coronary Heart Disease

Based on the results of previous studies and cases reported in Japan, there are three main work stressors related to stroke and coronary heart disease.

The first stressor is the type of work, which is based on the manner in which the work is performed. The second category is physical, chem-

ical, and ergonomic factors in the workplace, and the third is related to the characteristics of the work that are associated with psychological stress.

In this paper, we shall only briefly refer to the first and second categories and focus on the third stressor, the characteristics of work that are associated with psychological stress or mental strain, and their effects.

#### 1. Stressors associated with type of work

One of the major work stressors associated with stroke and coronary heart disease is long working hours. The data in Japan show that the risk of acute myocardial infarction for male workers who work more than 11 hours a day (i.e., approximately 60 hours a month over time) is 2.44 higher than for those who work 7–9 hours a day. Working more than 60 hours a week also increases the risk of cerebrovascular and cardiovascular disease.

The effect of long working hours is closely associated with lack of sleep. Lack of sleep is generally thought to increase the reactivity of the circulatory and sympathetic nervous systems. High angina pectoris and myocardial infarction morbidity among persons getting less than 6 hours sleep a day and 2.08 times higher mortality from ischemic heart disease and cerebrovascular disease among those getting less than 4 hours sleep a day than 7 hours have been reported in some studies.

The entire time spent in the workplace, including nap time, break time, and staying at the

office all night, in addition to actual working hours, also needs to be considered as a problem involved in long working hours. Moreover, some studies have found that when work hours are irregular and the changes of starting time are more than 3 hours, the risk of myocardial infarction is 2.53 times higher than when the change in work hours is less than 1 hour. This indicates that irregular work hours may interfere with sleep-wakefulness rhythm and increase the risk of stroke and coronary heart disease. Some negative research results have been reported for associations between cardiovascular disease and shift work, night work, and early work, but many studies have reported a higher risk of cardiovascular disease associated with changes in shift work, ranging from approximately 1.2 to 1.5 higher.

As to other types of work, work with many business trips increases the risk of stroke and coronary heart disease. Breaks and rests, including sleep time, and recovery from fatigue are keys to prevent such disease, especially in work involving business trips to other countries with large time differences.

## 2. Physical, chemical, or ergonomic factors in the workplace

Working in a cold environment induces or aggravates ischemic heart disease and cerebrovascular disease. A temperature decrease of 10 degrees Celsius has been found to be associated with a 13% increase in the incidence of attack in coronary artery, an 11% increase in mortality and incidence of coronary artery disease, and a 26% increase in recurrence of attack in coronary artery.

During chronic exposure to noise in a noisy environment (i.e., more than an 80dB noise level), systolic and diastolic pressure elevation, high hypertension morbidity, a 1.2–1.4 higher times relative risk of hypertension due to noise, and a direct association between noise and ischemic heart disease have been observed.

In a comparative study between commercial pilots and a control group, left ventricular hyper-

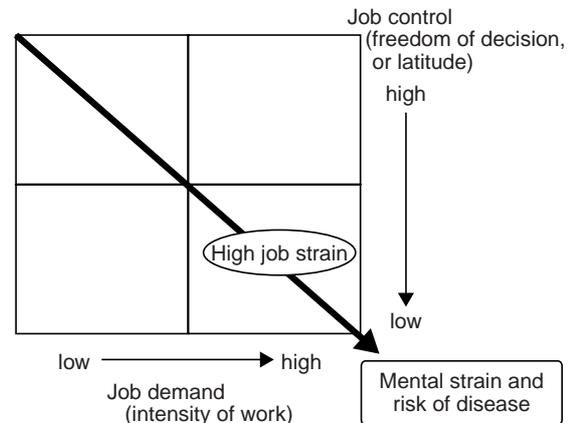


Fig. 2 Job demand-control model

trophy, systolic blood pressure elevation, and high serum cholesterol were more frequently observed in the commercial pilot group, suggesting that shift work system is associated with time difference, and that there is an association between large time difference and stroke and coronary heart disease.

## 3. Work-related psychological stressors

Some work stressors that induce mental strain have sudden powerful effects, or powerful effects for relatively short periods, and others have repeated effects at work every day over long periods. The latter will be examined in this section.

Psychological stressors in the workplace and their effects have not been fully clarified, because evaluation of work stress is difficult. However, the concept of work stress and evaluation methods have recently become established, and the reliability and validity of the method have been assessed. The number of findings based on these methods has been increasing and we will introduce one of the findings.

Karasek<sup>2)</sup> proposed a 'job demand-control' model (Fig. 2) to evaluate psychological stressors in the workplace. In this model, work-related psychological stresses occur as the result of the interaction between two factors. One factor is workload and allocation of work, or so-called

'job demand', including the concentration and degree of tension required to do the work, in other words, the intensity of the work. The other factor is latitude, or the freedom of execution and decision at work, or what is referred to as 'job control', under which talents and skills can be demonstrated and improved.

According to Karasek's model, psychological work stress is determined by these two factors and the interaction between them. The combination of a high-level of job demand and low-level of job control, that is, an environment of hard work with little freedom, causes the highest psychological stress and high risk of disease. Study of this 'job demand-control' model has mainly been conducted in Europe, and many findings support the correlation between job stress and cardiovascular disease. This model is now one of the best models for evaluating job stress. Although few results have been reported in Japan, a cohort study showed a significantly lower level of job control among workers who were absent from work due to cardiovascular disease. Support by superiors or colleagues, i.e., human relationships in the workplace, as well as job demand and job control, have been found to greatly modify the effects of job stress. Cardiovascular disease mortality and morbidity are high in groups with severe work stress and little support from supervisors or colleagues, especially among manual workers.

In addition, Johnson *et al.* proposed the 'job demand-control-support' model.<sup>3)</sup> In this model, the combination of high-level of job demand, low-level of job control, and low social support is associated with the highest level of mental strain and high risk of disease, such as stroke and coronary heart disease. This model is now widely accepted.

### **Psychological Stress in the Workplace and the Pathogenetic Mechanisms of Stroke and Coronary Heart Disease**

The mechanisms by which work-related psy-

chological strain affects the onset of stroke and coronary heart disease have not been identified, but the following have been suggested. First, work-related psychological strain may increase the cardiovascular risk associated with risk factors such as smoking, drinking, blood pressure, obesity, and low physical activity. The overall trend in previous research has shown relatively negative results for an association between job stress and serum lipids, and no consensus has been reached in regard to smoking. Most studies report negative results for an association between job stress and casual blood pressure, but a significant association has been observed with 24-hour blood pressure. There is a difference in blood pressure at home or in a sleep between workers with high job strain and workers with low strain, which suggests that work stress persists even when away from work. Also, it has been suggested that job stress causes depression, leads to low physical activity, and inhibits the improvement of daily habits and coronary risk factors.

Second, job stress may cause coronary arteriosclerosis by other pathophysiological mechanisms. For example, some studies have shown an association between work stress and fibrinogen and HbA<sub>1c</sub>, which will be clarified in further studies.

Third, at the stage of increasing risk of the onset of disease, psychological stresses in the workplace may trigger the onset of fatal arrhythmia and cardiac infarction. The onset of these diseases is presumably induced by excessive stimulation of the sympathetic nervous system and a delay in recovery from decreased myocardial function. Some studies have shown that working until late at night causes excessive stimulation of the sympathetic nervous system, suppresses the parasympathetic nervous system, and causes blood pressure elevation, especially in hypertensive patients. We expect future studies to identify the effects of work-related psychological stresses in individuals who may have cardiovascular disease.

## Conclusion

Here we shall discuss the association between work stress and stroke and coronary heart disease. Type of work, including work hours, work environment, and work-related psychological stressors based on the characteristics of work have been shown to be associated with the occurrence of stroke and coronary heart disease and the course of the disease after the onset. However, the mechanisms of the associations have not been clarified. Additional findings need to be obtained and assessed to determine the association between work-related psychological stressors and the pathogenetic mechanisms of stroke and coronary heart disease in the workplace.

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