Passive cigarette smoking increases risk of coronary heart disease

Despite the decline in mortality from coronary heart disease during recent decades, it is still the leading cause of death in the U.S. and other industrialized countries. In 1996 an estimated 476,124 residents of the United States died of coronary heart disease, representing more than one in every five persons who died in that year. In many economically developing countries, mortality from coronary heart disease has increased rapidly and has become the leading cause of death. Exposure to passive smoking is very common in the general population, especially in young and middle-aged men and women. For example, 43% of children aged 2 months to 11 years and 37% of adult non-tobacco users are exposed to passive smoking in the United States. As such, identification of the increased risk of coronary heart disease associated with passive smoking has important public health implications.

The relationship between passive smoking and risk of coronary heart disease has been examined in a recently published meta-analysis of epidemiological studies. A total of 18 epidemiological (10 cohort and eight case-control) studies comprising 6813 coronary heart disease cases was included in this meta-analysis. Overall, passive smoking was associated with a 25% increase in the risk of coronary heart disease among non-smokers (relative risk [RR]: 1.25, 95% confidence interval [95% CI]: 1.17 to 1.32). Furthermore, passive smoking was consistently associated with an increased relative risk of coronary heart disease in cohort studies (RR: 1.21, 95% CI: 1.14–1.30, P<0.001) and in case-control studies (RR: 1.51, 95% CI: 1.26–1.81, P<0.001); in men (RR: 1.22, 95% CI: 1.10–1.35, P<0.001) and in women (RR: 1.24, 1.15–1.34, P<0.001); and in those exposed to passive smoking at home (RR: 1.17, 95% CI: 1.11–1.24, P<0.001) or at work (RR: 1.11, 95% CI: 1.10–1.23, P=0.05). A significant dose-response relationship was identified, with relative risks of 1.00, 1.23, and 1.31 for non-smokers who were not exposed to passive smoking, exposed to 1–19 cigarettes/day, or exposed to ≥20 cigarettes/day, respectively (P=0.005 for linear trend).

These findings are unlikely to be due to misclassification of outcomes. The pooled relative risk from studies in which the end-points were myocardial infarction and/or coronary heart disease death (RR: 1.24, 95% CI: 1.17–1.32, P=0.001) was similar to the pooled relative risk from all studies. Similarly, these findings are unlikely to be due to recall bias, from the desire of the sick patient to identify a cause for their illness. A positive and significant relationship between passive smoking and the risk of coronary heart disease was identified in the 10 prospective cohort studies in which passive smoking exposure was assessed prior to the onset of coronary heart disease.

The results are also unlikely to be due to the confounding effects of lifestyle factors and diet. Several studies have indicated that passive smokers are more likely to consume diets lower in vegetables and fruits and higher in fat content and are less likely to take antioxidant vitamin supplements compared to non-smokers. However, recent clinical trials have indicated that beta-carotene and vitamin E supplementation does not reduce the risk of coronary heart disease in persons without a history of myocardial infarction. In the meta-analysis by He et al., the pooled relative risk of coronary heart disease associated with passive smoking among studies in which important confounding factors for coronary heart disease, such as age, sex, body weight, blood pressure, and serum cholesterol, were adjusted (RR: 1.26, 95% CI: 1.16–1.38, P<0.001) was virtually identical to the pooled relative risk from all studies.

The findings are unlikely to be due to failure to include unpublished studies (publication bias). An exhaustive literature search was conducted to identify unpublished as well as published studies on passive smoking. The pooled relative risk from studies published in peer-review scientific journals (RR: 1.25, 95% CI: 1.17–1.33, P<0.001) was identical to that obtained by pooling the relative risk estimates from all available studies. In addition, correlation analysis on standard error and log relative risk did not support the possibility of publication bias (Kendall tau correlation coefficient=0.24, P=0.16).

The remarkable consistency of the association between passive smoking and risk of coronary heart disease across different populations, study designs, and exposure measurements suggests the presence of a causal relationship. Causality is further supported by the finding of a significant dose–response relationship and biological plausibility for the association. Several potential mechanisms may be involved in increasing the risk of coronary heart disease in persons exposed to passive smoking. The acute effects of exposure to passive smoking on cardiovascular function include an increase in resting heart rate, blood
pressure, and blood levels of carboxyhaemoglobin and carbon monoxide\textsuperscript{[9,10]}. Exposure to passive smoking is also associated with an increased total cholesterol to HDL cholesterol ratio and a decrease in HDL cholesterol levels\textsuperscript{[11]}. Furthermore, passive cigarette smoking has been shown to increase platelet aggregation and cause endothelial cell damage\textsuperscript{[12]}. Abnormal platelet aggregation is an independent risk factor for coronary heart disease. In addition, there is evidence that passive smoking sensitizes circulating neutrophils in humans and may cause their subsequent activation and oxidant-mediated tissue damage, leading to atherosclerosis\textsuperscript{[13]}. These potential biological mechanisms suggest that even light exposure to cigarette smoking may result in coronary heart disease.

The findings in our meta-analysis provide strong evidence that passive cigarette smoking increases the risk of coronary heart disease in non-smokers. Although the increase in relative risk is moderate, these findings have important public health implications. Given the high prevalence of passive cigarette smoking at home and in the workplace and the fact that coronary heart disease is the leading cause of death in many western countries, the excess of coronary heart disease deaths due to passive smoking is substantial. To achieve a meaningful reduction in the societal burden of coronary heart disease, both passive as well as active cigarette smoking must be targeted for reduction.

Many children are exposed to passive smoking on a regular basis at home or in other environments, such as child-care facilities and schools. The health consequences of exposure to passive smoking among children, including the increased risk of cardiovascular disease, should be addressed in future studies. The only safe way to protect non-smokers from exposure to passive smoke is to eliminate this health hazard from public areas, the workplace and the home environment. To accomplish this goal, strong clean indoor air policies and laws should be developed and enacted.

References