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Health effects of ambient combustion-related fine and ultrafine particulate air pollution: recent epidemiological evidence

There is ample evidence that breathing combustion-related fine particulate air pollution contributes to cardiopulmonary disease and mortality. Epidemiologic evidence suggests that short-term exposure exacerbates existing cardiovascular and pulmonary disease and increases the risk of becoming symptomatic, requiring medical attention, or even dying. Long-term repeated exposures increase the risk of chronic pulmonary and cardiovascular disease. Recent research has attempted to evaluate potential mechanistic pathways that link exposure to particulate air pollution and cardiopulmonary disease and mortality. This research suggests that general mechanistic pathways probably include pulmonary and systemic oxidative stress and inflammation, enhanced initiation and progression of atherosclerosis, and altered cardiac autonomic function. Recent research has also attempted to evaluate the constituents or characteristics of particulate air pollution most responsible for the health effects. Epidemiologic studies have generally implicated combustion-related fine particles as being largely responsible. Ambient air in urban and industrial environments is constantly being polluted by fresh emissions of ultrafine particles primarily from combustion-related sources. These primary particles, however, have a very short life (minutes to hours) and rapidly grow (through coagulation and/or condensation) to form complex aggregates. Epidemiology is limited in its ability to elucidate the role of ultrafine (or nanoparticles) versus other fine particles (in the accumulation mode) because of the complex and ever changing nature of these particles. Nevertheless, there is clear evidence that both fine and ultrafine particulate air pollution can have adverse effects on human health.

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