ABSTRACT

Cardiovascular disease (CVD) includes numerous heart and circulatory system disorders and is a leading cause of death worldwide. In the United States, over 80% of all deaths are related to some form of disease. While the majority of research on the health effects of diesel exhaust (DE) has focused on the impact of DE on respiratory end-points, there is growing body of research on the cardiovascular effects of DE that could have significant public health implications. DE is highly toxic and can cause oxidative stress, inflammation, angiopathy, thrombogenic, and ischemic effects of inhaled DE, albeit for older-generation models diesel engines and at concentrations that are much higher (~300 µg/m3) than typical levels in or even occupational levels (Kipen et al., 2009).

Results from animal studies provide insight into the potential mechanisms underlying DE and support the need for more human studies. Some studies have found increased risk of cardiovascular diseases in workers exposed to DE, while others found no significant difference between the exposed and unexposed groups. Epidemiological studies, which use occupationally exposed populations to which workers are exposed, often have long latency periods, and workers may have multiple exposures. Overall, the DE epidemiology studies did not show a consistent effect. In addition, a meta-analysis of epidemiological studies concluded that in the current state of the art, there is not sufficient evidence to support a causal relationship of DE with cardiovascular disease.

OBJECTIVE

To determine the strength of the evidence of an association between CVD and DE exposure and to provide a review of cardiovascular epidemiology studies of DE and to make action statements. In addition, we evaluated previous reviews of evidence generated by national and international public health organizations. Since most of these reviews had been published prior to 2006, our focus on the original literature was on studies published after that date. Only those studies were evaluated using WHO methodology, with more weight given to studies of larger size.

RESULTS

Human and Animal Toxicology Studies

Human and animal toxicology studies provide new evidence of lung inflammatory, thrombogenic, and ischemic effects of inhaled DE, absent for older-generation diesel engines and as a consequence of more than 100% increase in lung-related death in one occupational cohort. Kipen et al. (2009).

Animal studies provide insight into the potential mechanisms underlying DE and support the need for more human studies. Some studies have found increased risk of cardiovascular diseases in workers exposed to DE, while others found no significant difference between the observed and expected disease rates. In a meta-analysis of epidemiological studies, conclusions that allow reliable prediction of adverse health effects at DE-exposure levels.

Epidemiology Studies

Epidemiology of cardiovascular epidemiology studies is complicated due to continuing exposure regional and occupational sources, gasoline exhaust, and tobacco. Overall, there is a focus on occupational epidemiology studies, where exposures are relatively well controlled.

PUBLIC HEALTH AGENCY REVIEWS

Environmental Health Criteria 177 (1996). No consistent effects of DE on cardiovascular diseases has been identified in short studies of workers with potential exposure to diesel exhaust.

Noncancer Health Effects of Diesel Exhaust (IPCS, 1996): No consistent excess of mortality from all causes in DE-exposed workers.wró

Noncancer Health Effects of Diesel Exhaust (US EPA, 2002): Epidemiological literature of DE and cardiovascular disease is consistent with the hypothesis that DE exposure may be a contributing factor to cardiovascular disease risk in DE-exposed workers.

Recent animal studies provide insight into the potential mechanisms underlying DE and support the need for more human studies. Some studies have found increased risk of cardiovascular diseases in workers exposed to DE, while others found no significant difference between the observed and expected disease rates. In a meta-analysis of epidemiological studies, conclusions that allow reliable prediction of adverse health effects at DE-exposure levels.

CONCLUSION

Despite the lack of consistent evidence of cardiovascular disease attributable to DE exposure, the evidence suggests that DE exposure may be a contributing factor to cardiovascular disease risk in DE-exposed workers. Further epidemiological studies are needed to confirm these findings and to better understand the mechanisms underlying the observed associations.

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