indicative of exposure to gaseous components (e.g., aldehydes, NO_x, SO_x, CO) of diesel exhaust.
Conclusion This study highlights both the historical context and the current status of exposure sampling for diesel exhaust in the occupational setting. Development of exposure sampling plans in the workplace should take into account both the timeframe of exposure (e.g., acute or chronic) and the toxicological endpoints of concern (e.g., acute irritation or chronic ailment). Furthermore, the individual assessing exposure should be aware of, and account for the differences between traditional diesel engine exhaust (TDE) and new technology diesel engine exhaust (NTDE).

1712b OVERVIEW OF DIESEL ENGINE EXHAUST ISSUES

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The mutagenicity of organic solvent extracts of diesel exhaust extracts was first noted over 60 years ago. Epidemiology and toxicology studies have resulted in classification of diesel exhaust by the International Agency for Research on Cancer (IARC) as a known carcinogen and by United States Environmental Protection Agency (USEPA) as likely to cause cancer; however, there is continued debate and quantitative risk has not been established by USEPA or environmental regulatory bodies in other countries. The major changes in diesel exhaust emissions make New Technology Diesel Exhaust (NTDE) distinct from the historic epidemiologic studies and recent animal bioassays question the relevance of studies of traditional diesel exhaust for risk assessment of NTDE.

1712c OCCUPATIONAL DIESEL EXHAUST EXPOSURE IN RELATION TO LUNG CANCER AND ISCHAEMIC HEART DISEASE MORTALITY

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Introduction General population studies of air pollution suggest that particles from diesel exhaust emissions are a potential risk factor for cardiovascular disease, while diesel exhaust is also classified as a known human carcinogen. We used data from the Diesel Exhaust in Miners Study to assess excess risk of lung cancer and ischaemic heart disease mortality associated with occupational diesel exhaust exposure.
Methods Analyses were performed in a cohort of non-metal miners in the US (n=12,315), who were exposed to diesel exhaust. We applied the parametric g-formula to assess how hypothetical interventions setting respirable elemental carbon (surrogate for diesel) exposure limits would have impacted lifetime risk of lung cancer, and ischaemic heart disease mortality, while adjusting for time-varying employment status.
Results Lung cancer and ischaemic heart disease mortality risk decreased in association with interventions on respirable elemental carbon, with risk ratios of 0.73 (95% confidence interval CI: 0.44 to 1.07) and 0.86 (95% CI: 0.66 to 1.12) respectively, when comparing an intervention setting respirable elemental carbon exposure to zero to the observed natural course.
Conclusion Our findings suggest excess risk of lung cancer and ischaemic heart disease mortality associated with diesel exhaust exposure in this occupational setting and that interventions on exposure would have resulted in reduced risk for both outcomes.

1190 HIGH TEMPERATURE INSULATION WOOLS: STUDY OF CYTOTOXIC, GENOTOXIC/OXIDATIVE AND INFLAMMATORY EFFECTS OF POLYCRYSTALLINE WOOLS COMPARED WITH REFRACTORY CERAMIC FIBRES

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Introduction Refractory ceramic fibres (RCF) and polycrystalline wools (PCW) constitute a family of fibres known as High Temperature Insulation Wools used in industrial applications above 800°C. Current European Classification, Labelling and Packaging of substances and mixtures classifies RCF as Category 1B ('Substances presumed to have carcinogenic potential for humans'). Regarding PCW toxicity, no studies are available. We aimed to evaluate and compare cytotoxic, genotoxic-oxidative and inflammatory effects of alumina-silicate RCF and PCW on human alveolar (A549) cells.
Methods SEM analysis was performed to characterise fibre dimensions. We exposed for 24 hour the cells to five different concentrations (2–100 µg/ml) of tested fibres to evaluate viability reduction by MTT and Trypan blue assays, membrane damage by LDH release, direct/oxidative DNA damage by Fpg comet assay and IL-6, IL-8 and TNFα cytokine release by ELISA.
Results SEM analysis found a length-weighted geometric mean fibre diameter (D^{LG}) of 2.1 µm with 68% of respirable fibres for RCF and a D^{LG} of 4.2 µm and 20% of respirable fibres for PCW. Moreover the content of fibres with $d<3$ µm and $l>20$ µm was 44% in RCF and 11% in PCW. We found lack of viability reduction for both fibres and membrane damage induction only for RCF at 100 µg/ml. Both the fibres induced dose-dependent DNA damage that, however, was higher for RCF reaching 5.7 fold of control vs 3.8 of PCW. Oxidative effects were induced only by RCF at the lowest concentrations. Regarding inflammatory effects, both the fibres induced only slight increase of IL-6 release at 100 µg/ml.
Conclusion The study confirms the genotoxic/oxidative potential of RCF with thinner D^{LG}, higher percentage of respirable and longer fibres than PCW and shows genotoxicity also for PCW, suggesting also for this fibre with similar chemical composition and low biosolubility, the need of further studies to confirm such results also on other cells.

116 SPECIFIC BIOMARKERS FOR THE EXPOSURE TO ORGANOPHOSPHATE AND CARBAMATE PESTICIDES

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Introduction Organophosphate pesticides (OPP) and carbamates are still counted among the most prominent agents used for crops protection. Up to date the determination of dialkyl