exposure and ovarian cancer in a case-control study of women from the Central Valley of California, U.S.
Methods: A population-based case control study of epithelial ovarian cancer is being conducted in the Central Valley of California. Cases were identified through rapid case ascertainment from two population based tumor registries. Controls were selected using random digit dialing techniques. As part of a telephone survey, women were asked about perineal use of talcum powder as well as frequency and length of use. Age-adjusted odds ratios and 95% confidence intervals were calculated using the Mantel-Haenzel technique.
Results: Analysis of the preliminary dataset which includes 214 cases and 862 controls, showed an adjusted OR of 1.2 (0.87, 1.7) for ever use of talcum powder. Among the frequency of use categories, women who reported use 4-7 times per week had a significantly increased risk of 1.6 (1.0-2.5), but there was no clear dose-response relationship with frequency of use. Although the risks were slightly increased for duration of use for 5-9 years (OR=1.6; 0.76-3.5) and 10+ years (OR=1.3; 0.91-1.9), the relationship is not significant or consistent.
Conclusion: Based on these data, there appears to be a modestly increased risk of ovarian cancer among some women who report use of talcum powder 4-7 times per week in the perineal area. However the analysis by frequency and duration of use yielded no consistent dose-response patterns. More work is needed on this topic, including assessment of risk comparing older users whose talc may have had asbestos contamination with younger subjects.

37 Comparative risks: the risk of renal disease versus other outcomes due to silica exposure

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There is now a sizeable body of literature which is reasonably consistent in showing that renal disease is associated with silica exposure. There is some suggestion that the mechanism involves auto-immunity, although direct silica toxicity to the kidney is also possible. In the United States the background (non-exposed) lifetime male risk of end-stage renal disease (ESRD) incidence is 2%. For a worker exposed for 45 years at a current recommended limit of 0.10 mg/m^3, the lifetime risk of ESRD is 19.1% (95% CI 2.5%, 82.0%), based on the only quantitative study to date (18 observed cases). For renal disease mortality (underlying cause), the lifetime risk of death at the same level of exposure is 2.1% (2.9%-10.0%), above a background risk of 0.3%, based on the only quantitative study (51 deaths). For death from any cause in the presence of renal disease (multiple cause), the lifetime risk is 7.2% (3.2%-15.6%), above a background risk of 1.5% (one study, 204 deaths).
These risks should be compared with the risk of other outcomes associated with silica exposure. The risk of silicosis on chest X-ray (1/1 or above) is 55%-92% in four studies with followup after employment (1583 cases) , but the risk of death from silicosis is 1.3% (170 deaths). There is no background risk for either silicosis incidence or death. The risk of from lung cancer due a 45 year exposure to 0.1 mg/m^3 is increased from a background rate of 5.3% to 7.0% (5.5%-8.9%) (1072 cases, 10 pooled studies).
Despite the relative sparsity of data for renal disease, these data indicate that the excess risk of death from renal disease (1.8%) is similar to the excess risk from lung cancer (1.7%), and the absolute risk of death from silicosis (1.3%), and that renal disease mortality should be of equal concern to regulators as lung cancer and silicosis mortality. Furthermore, in many cases renal disease is a major contributory cause, and the excess risk of death from any cause in the presence of renal disease is 5.7%.
All these risks exceed what the US OSHA considers acceptable, ie., an excess risk of 0.1%. Permissible exposure limits would need to be reduced ten-fold, to 0.01 mg/m^3, to approximately attain acceptable excess mortality risks. Morbidity risks from renal disease and silicosis (diagnosed via chest X-ray) are much higher than mortality risks. End-stage renal disease, however, is a very severe disease, while silicosis diagnosed via X-ray opacities may be sub-clinical.

38 Induction of acute phase systemic reactions and ischemic heart disease due to inhalation of fine dust

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From epidemiological studies there is increasing evidence that inhalation of fine dust in both environmental a[illegible] setting may enhance the risk of ischemic hea[illegible] posed populations. There is even some recen[illegible] workers who are mainly exposed to silica-dus[illegible] creased risk of ischemic heart disease. A hypothesis has been launched that inhalation of very fine dust from urban air pollution may induce inflammatory reactions in the lungs with release of mediators which may in turn influence blood coagulability und thus lead to IHD. Linking occupational exposures to inhaled particles to IHD has later extended this hypothesis. In more detail, the proposed pathogenetic pathway for this possible relationship is that inhaled irritants induces an increased production of interleukin-6 (IL-6) from cells in the bronchial mucousa which in turn stimulates the production of other acute phase proteins including fibrinogen from hepatocytes. As there is an established association between high plasma fibrinogen levels and the occurrence of coronary heart disease, it might well be that the proposed pathway can be part of the pathogenic explanation as to why workers in several occupations with exposure to fine dust and other lung irritants have an increased risk of IHD. In an attempt to test the hypothesis that an increased risk of IHD could be explained by dust inhalation with subsequent inflammatory reactions in the bronchial mucousa, IL-6 and fibrinogen were measured before and after exposure in 8 volunteers who were exposed for 4 hours to a median of 19.3 mg/m^3 of organic dust inside a piggery. The results showed an increase in the blood concentrations of both IL-6 and fibrinogen 6 and 24 hours after the exposure bad started. In a study of stainless steel welders and grinders we were not able to find any significant changes in their blood levels of IL-6

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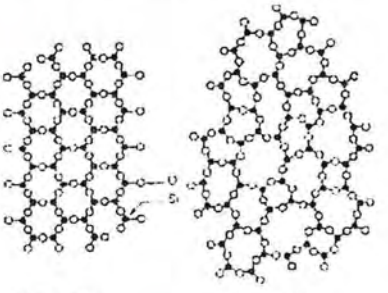

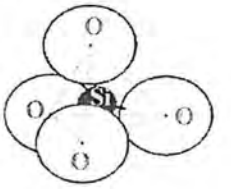

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