generally saw a stronger effect for squamous- and small cell lung carcinomas than for adenocarcinomas. Smoking and simultaneous exposure to other occupational exposures exerted a minor confounding effect on the risk estimates. The effect modifications with smoking tended to be supra-additive.

Conclusions SYNERGY adds valuable knowledge to the field of occupational cancer epidemiology, and underlines the importance to collect data on histology, and lifelong information on occupational exposures and smoking.

Method As an alternative to mean and maximum blood lead levels, we carried out an exposure assessment that assigned workers to high, medium or low exposure to lead. We additionally assessed whether workers would be exposed to an important level of relevant co-carcinogens.

Results 3466 deaths were observed among 7770 men and 1352 women. The SMRs for all causes (109, 95% CI 105–112) and all malignant neoplasms (113,107–120) were significantly raised. SMRs for oesophageal, stomach, bladder, brain and kidney cancer and non-malignant kidney disease were not raised, but were raised for lung cancer (142,129–157). The SMR for circulatory diseases (105,99–100) was of borderline significance. No trends were observed for mean or maximum blood lead level or assessed lead exposure for any of the cancers of a priori interest, but a significant association was found for circulatory diseases (ischaemic heart disease) with mean and maximum blood lead level.

Conclusions The excess of lung cancer is possibly to be due to tobacco smoking. This study provides strong evidence to support an association between increased lead exposure and increased risk of ischaemic heart disease mortality. The study is, however, limited by the lack of complete occupational histories for the included participants.

Method We conducted a meta-regression of lung cancer mortality and cumulative exposure to elemental carbon (EC), a proxy measure of DEE, based on relative risk (RR) estimates reported by three large occupational cohort studies. Based on the derived risk function, we calculated ELRs for several lifetime occupational and environmental exposure scenarios, and also calculated the fractions of annual lung cancer deaths attributable to DEE.

Results We estimated a lnRR of 0.00098 (95% CI: 0.00055, 0.0014) for lung cancer mortality with each 1-µg/m^3 3-year increase in cumulative EC. Estimated numbers of excess lung cancer deaths through age 80 for lifetime occupational exposures of 1, 10, and 25 µg/m^3 EC were 17, 200, and 689 per 10 000, respectively. For lifetime environmental exposure to 0.8 µg/m^3 EC, we estimated 21 excess lung cancer deaths per 10 000. Based on broad assumptions regarding past exposures we estimate that approximately 6% of annual lung cancer deaths may be due to DEE exposure.

Conclusions Combined data from three US occupational cohort studies suggest that DEE at levels common in the workplace and in outdoor air appear to pose substantial excess lifetime risks of lung cancer, above usually acceptable limits in the US and Europe, which are generally set at 1/1000 and 1/100 000 based on lifetime exposure for the occupational and general population, respectively.