OBJECTIVE: Genetic susceptibility in work-related lung cancer aetiology could have an important public health impact. Few studies have previously evaluated this issue, with inconsistent results. We aimed to investigate interactions between exposure to occupational carcinogens and genetic polymorphisms in lung cancer aetiology, adopting a systematic integrated approach.

METHOD: EAGLE, a population-based case-control study, enrolled 2100 lung cancer cases and 2120 controls (Italy, 2002–2005). Lifetime work histories were collected for 4059 subjects and translated into exposure to six occupational carcinogens (asbestos, silica, polycyclic aromatic hydrocarbons, diesel exhausts, chromium, and nickel) using a job-exposure matrix. We selected 298 tagging single nucleotide polymorphisms (SNPs) for assessment which do or do not take account of latency to estimate numbers exposed to a causative agent using Levin’s formula for PAF, to estimate a workforce turnover factor by age group, which accounts for the age structure of an exposed population. To estimate age-specific RRs from unit relative risks per year of exposure, the link between age and duration of exposure can be modelled using Monte-Carlo methods.

RESULTS: We show the effect of estimating the burden of lung cancer due to occupational exposure to respirable crystalline silica for Britain using PAF estimates which do or do not take age into account. Taking account of age and assuming recruitment between ages 15–44, there were 1188 lung cancer registrations in males in 2010, or 798 without accounting for age, or 636 vs. 804 assuming recruitment between ages 15–24. The extension to using age-specific RRs is demonstrated for occupational asbestos-related lung cancers.

CONCLUSIONS: Given the above results, and although highly dependent on assumptions made about workforce ages, there is clearly a case to be made to estimate PAFs by age.