Correspondence

welders. Therefore, welders some and possible for study by adjustments for smoking. The cause-case study by Hull et al in Los Angeles county showed ORs of 0-9 (95% CI 0-5-1-8) for SS welders and 1-3 (95% CI 0-6-2-3) for SS welders predominately exposed to low-levels of chromium and nickel (manual metal arc welders), whereas the OR was 1-6 (95% CI 0-8-3-1) for MS welders. Adjustments for smoking made little difference.

The European mortality study included 11,092 welders from eight countries. This large study provided no consistent difference between MS welders, for which the SMR was 1-78 (95% CI 1-72-4-34), statistically different and predominantly SS welders whose SMR was 1-23 (95% CI 0-75-1-90). The results of this study for cancer incidence provided the same pattern as those for mortality as the standardised incidence ratios (SIRs) were 1-75 (95% CI 1-22-4-24) for MS welders and 1-39 (95% CI 0-74-2-38) for predominately SS welders.

In conclusion, it seems to me that, although SS welders are potentially exposed to chromium and nickel compounds, epidemiological studies focused on the risk of lung cancer of SS welders do not provide clear evidence to suggest that SS welders are at higher risk of lung cancer than MS welders. Therefore, the cause of the excess lung cancers found among MS and SS welders is an unanswered issue. A recent study by Jockel et al supports the hypothesis that some of the excess risk of lung cancer among welders could be due to exposure to asbestos. Further investigations that controlled for smoking, exposure to asbestos, and possible different healthier worker effects among MS v SS welders, are needed.

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The urinary activity of NAG (total), NAG-A, or NAG-B did not show any relation with current or time-integrated PbB, but were all highly associated with the change of PbB over the past six months (A) or the changes of creatinine excretion (B). The PbB was significantly correlated with the urinary creatinine excretion (B) for all workers. The results clearly show that the change of PbB correlated with the change (A) in NAG activity over the same past six months. Also they did not measure biological values per se at the time of their study, except blood cadmium (CdB) in a subsample of the non-exposed group. It is unclear whether the changes of CdB and CdU in the correlation study with the NAG and NAG-B reported in Chia’s letter are related to the same initial study population and the same period of time. The most disturbing finding of Chia et al is that urinary NAG activity is related to the percentage change in PbB without being correlated to the absolute PbB concentration, which implies that the effects of ΔPbB on NAG activity are independent of the PbB concentrations, is difficult to reconcile with the basic concepts of toxicology. In a study on workers exposed to Pb from a Pb acid battery factory in which all subjects had a CdU below 2 μg Cd/g creatinine (mean 0.36 μg Cd/g creatinine in both the control and exposed groups) we could not find any differences in NAG between the control and exposed groups.1 The mean PbB in the battery workers (510 μg Pb/g) was, however, 50% higher than that of the Pb stabiliser workers examined by Chia et al.3 The hypothesis that NAG represents a specific renal marker associated with an early stage of tubulotoxic action of Pb exposure is also not supported by other independent studies performed in our laboratory and by the findings of other authors.