Therefore, welders.

This result remained unchanged after adjustments for smoking. Moreover, with an external reference, the lung cancer SMR of SS welders was low (1.66:1.97-1.91).4

The case-control study by Hull et al in Los Angeles county5 showed ORs of 0.95 (95% CI 0.5-1.8) for SS welders and 1.3 (95% CI 0.6-2.3) for SS welders predominately exposed to chlorinated solvent vapour 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.6

The lung cancer mortality study included 11 092 welders from eight countries.7 This large study provided no consistent difference between MS welders, for which the SMR was 1.78 (95% CI 1.72-2.43), 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 standardized incidence ratio (SIR) of lung cancer was 1.75 (95% CI 1.22-2.42) for MS welders and 1.39 (95% CI 0.74-2.38) for predominantly SS welders.8

In conclusion, it seems to me that, although 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.9 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.10 Further investigations that controlled for smoking, exposure to asbestos, and possible differential healthy worker effects among MS vs SS welders are needed.

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Urinary N-acetyl-β-D-glucosaminidase (NAG) and exposure to inorganic lead

Editor—Urinary activity of N-acetyl-β-D-glucosaminidase (NAG) has been reported to be one of the earliest markers to be increased in workers exposed to lead.1,2 The underlying mechanism for an increase in NAG activity is not fully elucidated. It was suggested that the increase in NAG activity may be due to stimulation of exocytosis.1 In a recent publication3 based on workers from a lead smelting plant, it was further suggested that increased NAG activity among workers exposed to lead was due to coincident “albeit slight cadmium exposure”. Their conclusion was based on the finding that urinary cadmium concentration (CdU) was the only significant variable in explaining the variation in NAG activity through stepwise regression analysis. Furthermore, the correlation coefficient (r) between NAG and CdU, was 0.41 in the non-exposed and 0.35 among exposed workers.4

Our data from exposed workers in a polyvinyl chloride lead stabilizer plant did not support this hypothesis. The increases in NAG, the heat labile isoenzyme (NAG-A), and the heat stable NAG isoenzyme (NAG-B) were all highly associated with the recent change in blood lead (PbB) concentration over the past six months (PbBΔ). Among several exposure indices derived from serial PbB concentrations, PbBΔ was the only significant variable to account for the variation in NAG, NAG-B, and NAG-A.

Furthermore, CdU and blood cadmium (GdB) did not correlate well with NAG and its isoenzymes. When they were forced into the regression model, GdB was in fact negatively correlated (table). Activity of NAG is also increased when other xenobiotics affect the proximal tubules.3,5 It is indeed very unlikely that NAG is a specific marker for cadmium. As inorganic lead is known to cause proximal tubular dysfunction, it is therefore not surprising to see an association between lead exposure and NAG.

Among lead smelters, it is possible that there may be significant concomitant exposure to cadmium. This is evident by the fairly wide range of CdB results (0.3-5 μg/g) and the significantly higher CdB and CdU concentrations among the workers exposed to lead.4 Our cohort of PVC lead stabiliser workers had never been exposed to cadmium. Their geometric mean (range) CdB and CdU were not statistically lower than those of the lead smelters (CdB: 0.56 (0.10 to 1.92) μg/l; CdU 0.41 (0.12 to 2.07) μg/g creatinine). We have also previously reported that NAG activity was better correlated with PbB than with any of the workers exposed to cadmium with a CdB of less than 3 μg/l had increased NAG concentrations.6

It is possible that where there is significant cadmium exposure, cadmium plays a greater part in increasing NAG activity than does lead, and perhaps even masks the contribution of lead. Our data show that lead does increase NAG activity in the absence of cadmium exposure.

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Contribution of PbBΔ, CdB, and CdU to variation in NAG*
The renal dysfunction is associated with tubular injury to the kidney. The tubular injury to the kidney is associated with exposure to Pb and Cd. Indeed, exposure to both metals, albeit at different intensities, usually occurs simultaneously both in industry and in the general population. Buchet et al clearly showed by a two-way analysis of variance that in a group of 62 workers with mixed exposure to Pb and Cd, a moderate exposure to Pb had no direct or synergistic action with Cd on several early markers of nephrotoxicity including NAG and tubular damage. The renal dysfunction associated with Cd exposure only.

N-acetyl-β-D-glucosaminidase activity in urine (NAG) was found to be lower in workers with low levels of Pb than in controls. However, the NAG activity is lower in workers with low levels of Pb than in controls. However, the NAG activity is lower in workers with low levels of Pb than in controls.

Recently, Chia et al have reported on the NAG isoenzyme profile in the urine of workers who manufactured Pb stearate stabilizers. The urinary activity of NAG (total), NAG-A, or NAG-B did not show any relation with current or time-integrated Pb exposure, but were all highly associated with the change of Pb over the past six months (days, weeks, and months). This finding was interpreted as the evidence that increased urinary NAG activity (NAG, NAG-A, NAG-B) could reflect recent changes of Pb exposure. The authors did not support this interpretation by checking whether the change of Pb exposure correlated with the change (A) in NAG activity over the same past six months. Also they did not measure biological markers associated with Pb exposure. The changes in NAG activity were found to be correlated with the concentration of Cd but not with the concentration of Pb. In two of these studies the subjects' Cd exposure did not exceed 2 μg Cd/g creatinine.

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