Manganese and Parkinson's Disease

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Employment as a Welder and Parkinson's Disease among Heavy Equipment Manufacturing Workers

A relatively powerful study of the potential link between welding work and cause or aggravation of Parkinson's Disease and diseases of a similar nature. It provides strong evidence that a history of welding work is not significantly associated with these outcomes.

Exposure to manganese e.g. as a result of inhaling welding fumes, can lead to symptoms which imitate Parkinson's disease (PD); the condition is known as manganism. PD is progressive but manganism is thought to be completely reversible, CT scans show different pathologies and, treatments for the two conditions differ. However, the similarity of symptoms leads to speculation that exposure to manganese can cause PD or lead to acceleration and/or aggravation of PD.

Several epidemiological studies have, on balance, not been supportive of this speculation.

The present case control study was designed to examine the possible link between having worked as a welder, and later suffering from PD. The study population was drawn from one heavy equipment manufacturing company which, from 1998 to 2004, ran its own no-fault but not compulsory medical insurance programme. Controls were matched for age, race, gender and production plant; a ratio of 10 controls to each case. Cases were those with any GP diagnosis of PD, Parkinsonism, or a related neurological disorder during the study period. Welding history was determined from employment records.

Given the uncertainty of diagnosis, two groups were analysed:

- Group 1; those with PD or secondary Parkinsonism and,
- Group 2; all those in Group 1 and, those with "other degenerative diseases of the basal ganglia" and "essential or other specific forms of tremor".

In this way those who did not receive a diagnosis of PD, but perhaps should have, would not inadvertently be excluded from the test of the hypothesis. Recurrence of a diagnosis from before the study period was included as a new case if it occurred during those six years.

The proportions of the workforce enrolled in the medical insurance programme were not reported. The authors assert that there was no relevant distribution bias, but data were not provided.

27% of the study population had been welders.

Out of a population of 12,595 there were 28 new cases and 42 long running cases. A population incidence rate of 10 cases of PD per 100,000 and a prevalence rate of 300 per 100,000 would predict fewer than 10 new cases and fewer than 30 long-running cases. The cases were dominated by PD and, "essential or other specific forms of tremor".

There were no statistically significant associations between welding and any of the above case definitions.

Comment
The results do not support welding-related causation, acceleration or aggravation hypotheses. This could mean that exposure to manganese is not related to causation of PD, but as there was no exposure assessment, this is an extrapolation.

6% of the calculations of the Odds Ratio produced a statistically significant result; 5% would be predicted by random effects alone.

Strengths of the study:
Random selection of matched controls, case ascertainment was made from live diagnosis (not death certificates), statistical power, employment record coverage.

Weaknesses:
The likelihood of seeking medical attention would be related to the degree to which a condition interfered with work. Essential tremor would be a handicap for a welder but other occupations could be more severely affected. Unstated analysis of medical care provider bias, actual exposure to manganese

was not available, no information on smoking history, no stated analysis of employment termination bias.

Conclusion:
In our view the study is of sufficient quality to strongly refute the proposal that welding work is a risk factor for cause or acceleration of PD.