REVIEW OF:

"CAUSE-SPECIFIC MORTALITY AMONG MALE CHEMICAL WORKERS"
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This is a well-organized and nicely presented investigation of patterns of mortality at the Midland and Bay City, Michigan, locations of Dow Chemical. This company is one of the chemical manufacturing giants, and has made a considerable effort in recent years to improve surveillance of its employees' health, and to maintain records suitable for epidemiological analysis. These efforts have clearly paid off, not only in this paper but in related papers on other Divisions of Dow in Texas and elsewhere.

Dow has often been the center of controversy with respect to environmental hazards. Pollution in Central Michigan has often been attributed to Dow. Dow in fact was reported in Chemical and Engineering News some years ago to have found elevated levels of dioxin in the nearby Tittabawassee River. It then fenced with environmentalists over whether the origin was its own chemical processing plant or "natural" combustion. It is commendable that, in the face of this criticism, its medical department conducts mortality studies of its own employees, who, after all, would be expected to receive higher exposures to various effluents than the surrounding population.

The paper is very nearly publishable in its present form, provided the editor is comfortable with its length; I think it's OK. I have a number of minor but pertinent questions.

What percent of person-years are 'empty' in the sense of pertaining to the very young (who have low death rates)?

What percent of person-years were contributed by very short-term employees (e.g., five years or less), whose death rates are of little relevance to the chemical industry?

How good was environmental control within the plant? Was monitoring ever done? For what chemicals? Are quantitative exposure levels available for any of the workers studied here?

Despite the obvious pitfall of using general population rates for reference, they do it anyway. How would they assess the mortality of the cohort relative to a WORKING population?

In Table VII the authors compare results using the U.S. reference population with what they would get using both a Michigan reference and a reference population consisting of the adjacent seven counties. This is useful and quite interesting; one wishes other investigators

would do similar analyses. Nevertheless, it does not solve a fundamental question. Are observed SMR's high (or low) because there is an effect in the cohort, or because the external reference is abnormally low (or high)? The extent to which SMR's within this table vary between reference populations may reflect nothing more than the variation between those populations themselves. If so, what have we learned about this cohort's experience from the data in Table VII?

I would advise the authors to go back over their tables and recalculate the SMR's. I didn't check all of them, but a few are not consistent with the observed and expected numbers, even allowing for roundoff. For example, in Table VI, under lymphatic and hematopoietic cancer, 190/180.1 is not 110. Under cancer of other and unspecified sites, 101/108.2 is not 140. In table XIII, under cancer of the large intestine 6/2.8 is not 281, and under cancer of the respiratory system 6/3.9 is not 163. There may be others I didn't catch.

Table VIII bothers me a lot, and needs more discussion. The SMR for all causes is significantly higher in hourly workers than in non-exempt salaried workers, which in turn is significantly higher than in exempt workers (judging from 95% confidence limits). A similar observation can be made for all cancers: the lower limit for hourlies exceeds the upper limit for exempts. Is this a legitimate comparison? If so, what

conclusions can one reach about pay status as an 'etiologic factor'? If not, what's the point of presenting confidence limits?

My strongest criticism concerns apparent inconsistencies in statistical results. The authors have used two different statistical methods: modified life table to arrive at SMR's and Hakulinen's (AM J Epid 113:192; 1981) modification of Mantel-Haenszel to compute RR's. Why? Hakulinen gives general advice for using his method. The authors should justify its use for their data, and should state explicitly the circumstances leading to the choice of each method in the Dow data. This becomes quite troublesome in places where the SMR and RR disagree with each other to a substantial extent. For instance, in Table XII, for all-cause mortality in men hired 1940-44, the RR is 1.12, but Obs/Exp = 298/467.3 = 0.64. What's the point of showing both sets of figures if they are so discordant? Which are we to believe?