

Letters

to the
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Editor

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Vinyl Chloride and Its Polymers

Letter-to-the-Editor. — This is in reference to the article by Tabershaw and Gaffey: Mortality Study of Workers in the Manufacture of Vinyl Chloride and its Polymers, JOM 16, August 1974.

I find this article to be quite interesting as well as intriguing. Although it appears that the methodology employed could withstand a great deal of scrutiny, I am not at all convinced regarding the interpretation of the results.

For example, sentence one of paragraph two of the abstract is a statement of fact, i.e. as long as readers do not get confused regarding a "study population" and the "population at risk"; the latter being the one on which inferences are made and presumably the population of importance — in most epidemiological efforts. Very interesting, but what can we make of the statement? Not much, really, for the real inference to the "population at risk" can be found under (2) in the 3rd paragraph of the abstract. The last sentence of the abstract suggests that vinyl chloride may be associated with cancer of multiple sites. Nonsense; no such a suggestion exists. In the same light, vinyl chloride may not be associated with cancer of multiple sites. These high SMR's may well be a consequence of sampling variability and in fact, the authors were unable to infer that significant excesses occur in the risk population.

The discussion on page 513 presents rationale in support of the conclusions

found in the abstract. While the authors intuitive judgment may well be correct, the results are inconclusive. Using statistical justification as support for their conclusions is most certainly invalid.

Robert B. Reger
Chief, Statistics Branch
NIOSH-ALFORD
Morgantown, WV 26505

Reply to Robert B. Reger's Letter

Reger's first comment on our paper makes a distinction between "population exposed to risk" and "study population," with which we agree. In every epidemiological study of which we are aware, the study population is, usually by design, a subgroup of the total population exposed to risk. Unless that subgroup is chosen by some kind of probability sampling, which is rarely the case, generalization from the study population to the population at risk is not a purely statistical process. It is

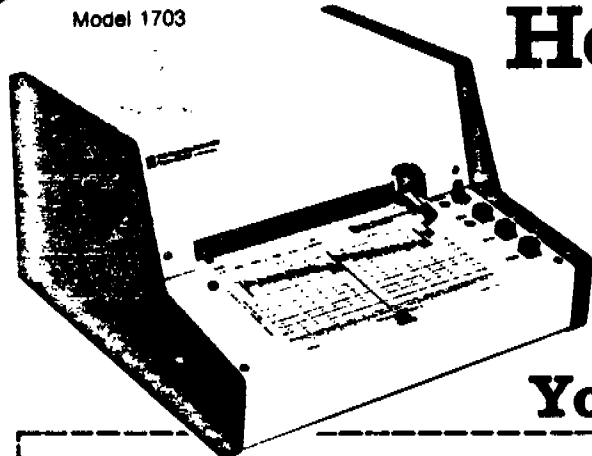
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mediated by a certain amount of common sense, and the existence of other data bearing on the plausibility of the generalization. Our investigation shares this common defect.

The second comment attacks our conclusions on the ground that the SMR's on which they are based are not statistically significant. Of the several responses which might be made to this point, we believe the following two are most relevant.

The first is that the evidence at hand in this case is a complex of facts; (a) malignant neoplasms do in fact occur more often than expected, while other

causes of death, with trivial exceptions, do not; (b) the deaths from malignancies which cause this excess are generally in the high exposure groups, and the more stringent the criterion for high exposure the greater the concentration of excess deaths; (c) for one site, digestive cancers, the observations agree with what is known from other evidence. We can measure the statistical significance of each SMR, but we know of no way to put a numerical measure on the significance of the above pattern.

The second is that the interpretation of a non-significant, as opposed to a significant, SMR is not as simple as Reger

implies. There is ample evidence that most employed groups have a lower mortality than the general population, so that there is a question as to what the "expected" SMR should really be. There is no problem when the SMR is significantly greater than 100, but when it is not, one enters muddy waters, statistically speaking.

We still believe that we are justified in what we consider to be a temperate conclusion from data which are admittedly less than overwhelming.

Irving R. Tabershaw, M.D.
William R. Garvey, Ph.D.

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