

PostScript

LETTERS

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Mortality of petroleum refinery workers

We would like to comment on the paper by Satin and colleagues,¹ which reports an update of a mortality investigation on two cohorts of petroleum refinery workers. The authors claim that one of the major aims of their study was the assessment of "health risks relative to more contemporary levels of exposure and work environments". Nevertheless, they explicitly admit that a previous investigation in such cohorts,² using the population of California as referent, found a strong "healthy worker effect" (that is, a significantly lower than expected mortality risk from cardiovascular disease and lung cancer). In our opinion, this observation, along with some other drawbacks, only in part expressly acknowledged in the paper, might have biased most of the results obtained, leading the authors to draw unreliable conclusions. We shall discuss this issue in detail, illustrating the main possible biases and how we believe they should have been interpreted.

Comparison bias

Exposure effects should be assessed in cohort studies by comparing the exposed cohorts with at least an unexposed one, as similar as possible in all relevant aspects.³

The new results by Satin *et al* have confirmed the occurrence of the "healthy worker effect" observed in the previous follow up. Such a finding may indicate a comparison bias concealing the associations, if any, between exposure and health risks.² In fact, occupational cohorts may differ from the general population in many features that have been associated with various risk factors, including socioeconomic status and personal habits.³ The presence of a comparison bias, at least in the Richmond refinery cohort, seems to be suggested by the risk for leukaemia in the subgroup with the shortest duration of employment (≤ 5 years), which is more than four times lower than the referent population (and nearly seven times lower than those of workers who worked the longest—that is, > 30 years). Finally, the lack of data on smoking, whose differential distribution is among

the main factors known to be responsible for the "healthy worker effect", should have suggested a more cautious interpretation of the results of analyses about diseases associated with such a risk factor, especially lung cancer. Owing to the quality of the data analysed, most of these limits are unavoidable. However, in our opinion, the authors should have taken them into account in discussing their results. For instance, the low leukaemia risk observed, in particular, for workers hired after 1949, should not have been considered as evidence of a lack of effect of quite low doses of benzene.

Dilution effect

Petrochemical workers are likely to experience different kinds and levels of exposure by job category. As a consequence, results from an analysis carried out by pooling together different exposure categories may be affected by a dilution effect—that is, an underestimation of the true mortality risk associated to exposure.³ For example, Gennaro and colleagues⁴⁻⁶ highlighted an excess of lung cancer risk among petroleum workers exposed to asbestos in an Italian refinery, which became evident only by using an unexposed job category as an internal referent group. In this investigation, the most heavily exposed group (maintenance workers) was 38% of the whole cohort of employees, similar to the proportion (36%) reported in a previous study on 10 US refineries.⁷ Furthermore, white collar workers constituted 22% and 21% of the workforce among the Italian and US refineries, respectively, suggesting that the composition of this kind of cohort tends to be similar, at least in Western countries. Unfortunately, the quality of the data in their possession prevented the authors from carrying out risk analyses by job category, and they did not discuss the possibility that the inclusion, if it occurred, of a notable proportion of workers scarcely or not at all exposed may have caused a significant lowering of the estimated risks.³

Moreover, the inclusion in the present analysis of workers employed after 31 December 1980, thus inflating the at risk population estimates (person-years), could have further contributed to diluting the possible risks, in addition to preventing a precise comparison with the previous update. In fact, a long lag time is expected between exposure and disease occurrence for most of the cancer sites considered. The mean time of follow up was roughly 33 years for workers hired before 1949 and only 23 for those hired after 1949, but the authors have not provided any information about the group employed since 1981, making it impossible to estimate the true risks associated with prolonged exposures. Comparing the paper by Satin *et al* to the previous follow up,² the number of workers enrolled after 1980 could amount to 3600 (that is, 31% of the subjects hired after 1949) and the corresponding follow up ranges from 1 to 15 years. However, these data do not allow the calculation of either the corresponding person-years at risk or the number of deaths which occurred.

Statistical analysis

The authors have indirectly evaluated the effect of exposures using the period of hiring (before versus after 1949) and the number of

years worked as factors. Due to the lack of more precise measures of the polluting concentrations, such substitute variables are of course necessary, even though in our opinion, the analysis for another cut off after 1949 (for example, 1969 or 1979) might have yielded some additional information about the variation of such risks over time. Furthermore, a possible confounding effect between the period of hiring and the other variables (for example, length of exposure and latency) should have been taken into account, for instance, either by applying a multivariable statistical model, such as the Poisson regression, or by stratified analysis.⁸

Insensitive indicators

Mortality rates may be poor indicators of cancer risk for disease sites with a good prognosis, for example, leukaemia and larynx cancer.³ For this reason, comparisons based on mortality rates might be affected by lack of statistical power. Moreover, the authors admit that the potential inaccuracy of diagnostic information from death certificates may have caused misclassification between asbestosis and pneumoconiosis. However, this inaccuracy might have also affected the risk estimates for specific leukaemia types. While the authors do not report any results for unspecified leukaemia, another investigation on petrochemical workers⁹ found surprisingly much higher risks for both "acute unspecified" and "cell type unspecified" leukaemia than those for each specified cell type, including acute myeloid (AML). In our view, when data come from death certificates, risk estimates for different leukaemia cell types must be interpreted with extreme care. In particular, the authors' claim that "the lack of any increase in AML ... argues against benzene's role in the increase of MM and NHL found here" is not justified.

Conclusion

Cohort studies based on mortality data and not including an internal group as a control may be affected by several biases. For this reason, the estimates of association between exposure to toxic chemicals and health risks obtained by these studies should be considered with caution.

Moreover, the observed excess of risk, if any, should not be ignored simply on the basis of the lack of statistical significance. The need for further investigations for a better evaluation of such risk, for instance, through nested case-control studies, should always be suggested.

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Authors' reply

Parodi *et al* raised several comments on our cohort mortality study of petroleum refinery workers in California.¹ Their comments are general in nature and apply to most, if not all, occupational cohort mortality investigations in general and refinery studies in particular, including such studies conducted in the USA, the UK, Canada, and Italy.^{2–7} We have discussed the same issues in our original paper. Below we will reiterate and expand our discussion of these issues in the order raised by Parodi *et al*.

The first comment raised by Parodi *et al* is the potential impact of the healthy worker effect (HWE) in our study. More specifically, Parodi *et al* conjectured that the HWE might have masked an excess of leukaemia, particularly in employees hired after 1949. The HWE is a potential problem common to all cohort studies that use general populations as the basis for comparison. All petroleum cohort studies conducted in the USA, the UK, Canada, and Italy are equally vulnerable. However, when raising the HWE as an issue, one must consider the following points. First, it is generally recognised that the disease most strongly affected by the HWE is cardiovascular disease and that the HWE has little impact on cancer. This view is supported by studies from the USA, Canada, and Europe.^{8–11} Second, the HWE diminishes over time after hire. Monson⁸ estimated that the HWE generally lasted about 15 years. In our study, there was no significant increase of leukaemia among employees 20 or 30 years after hire, regardless of hire date (before or after 1949). Therefore, the lack of a leukaemia excess in our study was not likely due to the HWE. Monson summarised most sensibly the impact of the HWE as follows: "The healthy worker effect is relatively weak in comparison to causal excesses that can be detected in epidemiologic data."

The second comment raised by Parodi *et al* concerns the lack of exposure information in our study that would have allowed us to classify workers by exposure and to conduct more detailed exposure specific analyses. Again, the lack of detailed exposure information is a general problem for all retrospective cohort studies, and our study of California refinery workers is no exception. We acknowledged this limitation in our original paper. A similar

comment regarding the lack of detailed classification of workers by exposure or job activity was raised previously concerning the finding of lung cancer in another study of US petroleum workers, but subsequent detailed analyses by job title revealed no increase of lung cancer for insulators, pipe fitters, electricians, boilermakers, or maintenance workers.¹² The most appropriate approach to deal with specific exposures is to conduct cohort based or nested case-control studies. Such nested case-control studies have been conducted subsequently for a number of cohort studies of petroleum workers in the USA, the UK, and Canada.^{13–16} Detailed exposure information (including quantitative estimates) was collected on individual cases and controls in these investigations. Furthermore, comparisons in these case-control studies are internal, thus avoiding the HWE. Based on nested case-control studies, Rosamilia and colleagues¹³ did not find any relation between lung cancer and asbestos exposure at a US refinery; Wong and colleagues¹⁴ did not find any increase of leukaemia, kidney cancer, or multiple myeloma in relation to gasoline (hence, benzene) exposure among US petroleum workers; Schnatter and colleagues¹⁵ did not find any relation between lymphohaematopoietic malignancies and benzene exposure in Canadian petroleum workers; and Rushton and Romaniuk¹⁶ concluded that there was no evidence of an association between benzene exposure and lymphoid leukaemia, either acute or chronic, among petroleum workers in the UK. Thus, none of the nested case-control studies contradicted the findings of the original cohort studies; nor is it axiomatic that the absence of analyses based on detailed exposure information automatically implies masked health effects.

Parodi *et al* criticised the inclusion of employees hired after 1980 in our study, and argued that the latency of these workers (15 years maximum) might not have been sufficient, thus "diluting" the risk of prolonged exposures among those hired in or before 1980. We would like to point out that, first, our investigation is not merely an academic exercise but part of an ongoing corporate medical monitoring programme that includes all employees. Second, an analysis stratified by latency was performed (table 3 in our original paper). The groups with 20–29 and 30+ years of latency did not include any employees hired after 1980 and, therefore, could not have been "diluted" by workers hired after 1980. Third, with regard to prolonged exposures, an analysis stratified by duration of employment was also performed (table 2 in our original paper), and the groups with 15–29 and 30+ years of employment would certainly have had prolonged exposures and the results would not have been "diluted" by employees hired after 1980.

With regard to statistical analysis, Parodi *et al* questioned our analysis by period of hire before and after 1949, and suggested cut points of 1969 and 1979. We do not understand the basis of their suggestion. We chose 1949 because of historical exposure patterns. In 1947, the recommended standard for benzene exposure in the USA was reduced from 100 ppm to 50 ppm, which was further reduced to 35 ppm in 1948. Benzene exposure levels in the petroleum industry were significantly reduced after 1949, thus making 1949 a good surrogate measure for exposure.

Finally, Parodi *et al* commented that mortality might not be a good indicator of cancer risk. This general comment, of course, applies to all studies based on mortality. In the USA

there is no national cancer registry, and it is simply not possible to ascertain cancer incidence in a historical cohort study of more than 18 000 workers that goes back to 1950. In their comments, Parodi *et al* were concerned with exposures to asbestos and benzene. The cancers related to these exposures are lung cancer, malignant mesothelioma, and acute myeloid leukaemia. These particular cancers have relatively poor prognosis, particularly in the past, and mortality may not be an unreasonable outcome measure. Parodi *et al* also commented on the diagnostic accuracy of death certificates. Again, this comment applies to all studies based on mortality. It should be noted that diagnostic accuracy varies by disease. For example, lung cancers are seldom misdiagnosed. Although some diagnoses on death certificates may not be as accurate as those based on detailed medical records, relying on death certificates in both the study and reference populations ensures comparability.

Furthermore, our interpretation of the results was based on not only what we observed, but also the findings of related studies. For example, for non-Hodgkin's lymphoma (NHL) and multiple myeloma (MM), in addition to our results, we also relied on hospital based case-controls studies which were included in previous reviews cited in our paper.^{17–18} The diagnoses in these hospital based case-control studies were based on detailed clinical, laboratory, and pathological findings. The conclusion from these hospital based case-control studies is that there is no relation between benzene exposure and NHL or MM. Therefore, our conclusion of MM and NHL based on our study is consistent with other studies in which diagnostic accuracy is not an issue.

In their conclusion, Parodi *et al* cautioned that results should not be ignored simply on the basis of the lack of statistical significance and suggested nested case-control studies be conducted for further investigation. We concur on these two points. In discussing the results of our study, we did not ignore any findings simply because they were not statistically significant. We fully recognised that a result might not be statistically significant because the statistical power of an individual study might not be adequate and that the result must be interpreted in conjunction with other similar studies. As one of the objectives stated in our original paper, we assessed our findings (statistically significant or otherwise) in the context of results of other petroleum studies. To take all studies into consideration, we also relied on several reviews and meta-analyses of studies of petroleum workers around the world.^{12–19} For example, based on a combined database of more than 350 000 petroleum workers in the USA, the UK, Canada, Australia, Finland, Sweden, and Italy, Wong and Raabe¹² reported that consistently not a single study showed an increase of lung cancer; the summary lung cancer standardised mortality ratio was 0.81, with a 95% confidence interval of 0.79 to 0.83 (based on 5695 deaths). As to the suggestion of further investigations using nested case-control studies, such detailed case-control studies have been conducted among petroleum workers in the USA, the UK, and Canada.^{13–16} As discussed above, none of these nested case-control studies contradicted the findings of the original cohort studies.

Therefore, while Parodi *et al* have raised several limitations common to occupational retrospective cohort studies, we believe that we already have discussed these issues in our

original paper and that we have not over interpreted our data.

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Sickness absence due to low back pain

In a recent interesting study published in your journal, Hoogendoorn and colleagues¹ determined that high physical work load and job dissatisfaction increase the risk of sickness absence due to low back pain. I would like to focus on the job satisfaction variable.

It is to be noted that the above study was performed in a prospective fashion with employed workers who had no recent history of low back pain injury. As such, I would like to familiarise the readership with a series of studies performed with chronic low back pain (CLBP) patients treated in a pain facility. The results of the studies described below resonate with Hoogendoorn and colleagues' results¹ and point to the importance of perceived job stress and job dissatisfaction and their importance to job function.

In a series of four papers, Fishbain and colleagues have attempted to determine if pre-injury job satisfaction impacts on "intent" to return to work to the pre-injury job after pain facility treatment. In the first report, Fishbain and colleagues² showed that chronic pain patients not intending to return to work after pain facility treatment were more likely to complain of job dissatisfaction. In the second report from this group, Rosomoff and colleagues³ showed that an association between non-intent to return to work after pain facility treatment and pre-injury job dissatisfaction was similarly found across Workers' Compensation and non-Workers' Compensation chronic pain patients. In the third report, Fishbain and colleagues⁴ looked at actual return to work after pain facility treatment in relation to these variables. They found that actual return to work was predicted at one month "by intent", perceived job stress, and job like (job dissatisfaction plus other variables). At 36 months, return to work was predicted by "intent" and by perceived job stress plus other variables. In the final study, Fishbain and colleagues⁵ attempted to predict "intent" to return to work after pain facility treatment in relation to actual return to work. "Intent" was predicted by perceived pre-injury job stress plus other variables. In addition, those chronic pain patients who intended to return and did not, were predicted by whether there was a job to go back to. Furthermore, chronic pain patients not intending to go back to work to the pre-injury job initially, but doing so later, were predicted by having a job to go back to.

Overall, this series of studies points to a strong relation between pre-injury work variables such as job dissatisfaction and "intent" to return to that job after treatment. In addition, these studies indirectly support the findings of Hoogendoorn and colleagues.¹ It seems that in trying to understand the low back pain injury and recovery process, it is important to take into account work related perceptions such as those of perceived job dissatisfaction and job stress.

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NOTICES

First World Congress on Work-Related and Environmental Allergy (1st WOREAL), and Fourth International Symposium on Irritant Contact Dermatitis (ICD), Helsinki, Finland, 9–12 July 2003

Congress on Work-Related and Environmental Allergy

- Work related and environmental aspects of respiratory and skin allergy
- Specific issues related to pathophysiology and skin allergy
- Management and prevention of allergy

Irritant Contact Dermatitis Symposium

- Occupational irritant dermatitis
- Prevention of irritant dermatitis
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- Irritant dermatitis from cosmetics

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- Satellite Symposia, 9 July 2003
- Allergy School, 9–10 July 2003
- 7th International NIVA Course on Work-Related Respiratory Hypersensitivity, 11–15 July 2003

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Assessment of Psychological Factors at Work
3–6 March 2003, Geilo Hotel, Geilo, Norway

Evaluation and Good Occupational Health Practice
23–27 March 2003, The Fell Hotel, Saariselkä (Lapland), Finland

Principles of Etiologic/Etiodiagnostic Research
11–16 May 2003, Hanasaari Cultural Center, Espoo (Helsinki), Finland

Toxicokinetic and Toxicodynamic Modeling in Occupational Health
15–19 June 2003, Red Cross Educational Training Center, Gripsholm, Sweden

Work-related Respiratory Hypersensitivity
10–15 July 2003, Marina Congress Center, Helsinki South Harbour, and The Sunborn Yacht Hotel, Naantali, Finland

Bullying and Harassment at Work
11–15 August 2003, Hotel Eckerö, Åland, Finland

Good Management Practice—Interaction of Environment, Safety and Quality
31 August–4 September 2003, Hotel Levittunturi, Sirkka (Lapland), Finland

Workplace Health Promotion—Practice and Evaluation
The first part 15–17 September 2003, Hotel Eckerö, Åland, Finland and the second part 19–21 January 2004, The Nordic School of Public Health, Gothenburg, Sweden

Indoor Air Quality Problems—Link between Indoor Pollution, Psychological Factors and Complaints
22–26 September 2003, Vilvorde Course Center, Vilvorde (Copenhagen), Denmark

Occupational Health Risk Assessment and Management
6–10 October 2003, Medical Academy of Latvia, Riga, Latvia

Introduction to Occupational Epidemiology
23–29 October 2003, Hotel Gentofte (Copenhagen), Denmark

Work-related Musculoskeletal Disorders: Current Research Trends
1–7 November 2003, The Sunborn Yacht Hotel, Naantali, Finland

Tools for the application of European Directives on health at the workplace—The example of chemical risk; Athens, Greece, 19–21 May 2003

Companies have to comply with national regulations stemming from European Direc-

tives on the prevention of occupational risks, including those resulting from exposure to hazardous chemicals. The symposium will provide a forum for a review of the problems encountered at the workplace.

The symposium is intended for a wide range of people: occupational physicians, people in charge of occupational health and safety, those responsible for implementing legislation or for advising companies on occupational risk prevention, representatives of the social partners and social security organisations, and specialists in air monitoring, biomonitoring, and epidemiology, etc.

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- Chemical risk prevention tools.
- Outlook and new challenges for prevention.

Contributions will be given in English, French, German, and Greek, with simultaneous translation. Posters will be in English.

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