CORRESPONDENCE

SAS program for testing the difference between two correlated correlation coefficients

Sir,—Sometimes we need to statistically compare two product-moment correlation coefficients (r) that are correlated—that is, not statistically independent. We might, for example, wish to determine whether the correlation of urinary cadmium (X1) on β2-microglobulin (Y) is statistically different from the correlation of blood cadmium (X2) on β2-microglobulin (Y) in the same group of subjects. In a methodological study to validate two methods, say machine (X1) vs ascultatory (X2), against a reliable invasive method (Y), for measuring blood pressure, we might compare the correlation of X1 on Y with the correlation of X2 on Y.

These correlation coefficients are correlated because they share the common variable Y measured from the same group of subjects, and any test of significance that ignores this non-independence will be inappropriate. Actually the problem has long been recognized and the test for comparing correlated correlation coefficients was first described by Hotelling in 1940.1 Hotelling’s t test has been used for many years and is still being used, even though the method has serious drawbacks.2 Improvements to the Hotelling test have been considered by several persons.

Here I describe an SAS program3 to compare two correlated correlation coefficients. The program uses the statistical procedure given by Meng et al4 and it outputs the Z value (standard normal deviate) and the two sided significance probability pertaining to the statistical difference of the two correlated correlation coefficients. To enhance user friendliness, the program is packaged as an SAS macro named %MCCORR (appendix). The macro requires four user supplied parameters: nx1 (correlation of X1 on Y), nx2 (correlation of X2 on Y), nx1x2 (correlation of X1 on X2), and n (number of observations in the sample). If Hotelling stored the macro with filename and extension as “cncor.m” in C/MY SAS, then the entire SAS program will consist of as few as two statements, one to invoke the macro and the other to supply the four parameters to the macro. Here is an example of a complete SAS program:

```
%Include 'c/MY SAS/CORR.MAC';
%CORR (0.72, 0.55, 0.22, 50);
%CORR (0.25, -0.14, 0.18, 60);
The first test gives Z = 1.4282 with the two sided probability value = 0.15319; the second test gives Z = 2.085 with the two sided probability value = 0.02097.
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Appendix:listing of the SAS codes

```
%Macro ccorr (nx1, nx2, nx1x2, n);
%* stored as ‘CORR.MAC’;
%Options nocenter nodate;
Data null;
zi = 0.5*sqrt((1 + nx1y)/(1 - nx1y));
z2 = 0.5* problem of nuisance parameters.
\( r_{2} = (nx1*nx2 - nx1x2)/(nx1x2 - nx2) \); fstat = (nx1 - 2)*(nx2 - 2)/((nx1 - 1)*(nx2 - 1)); if f > then f = 1;
\( h = (nx1 - 1)/(nx1 - 2) \);
\( \delta f = (nx1 - 2)*(nx2 - 2) \); put @ (Z = Z 8.2 sided p value =
*Note: output is shown on the 'LOG WINDOW'; run;
%Mend ccorr;
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Reproductive risks associated with diving

Sir,—Raymond (1993;50:1055-6) considers the risks to reproduction from contraceptive heat chambers among divers who use hyperbaric chambers. It is worth noting that they may be presumed to be at risk not only from the heat, but also from the pressure. Röcker et al5 reported that the plasma testosterone concentrations of rats exposed to a hyperbaric environment of air were significantly and substantially (about 50%) reduced. Röcker and Haglid6 reported that plasma testosterone in human divers showed it to decrease after diving.

I have hypothesised that the sex ratio (proportion of males) of mammalian (including human) offspring is affected by the hormone concentrations of both parents at the time of conception; high concentrations of testosterone being associated with subsequent births of boys and high concentrations of gonadotrophin with subsequent births of girls.7 This suggestion is supported by the findings of Lyster8 and Röcker9 who reported highly significant low sex ratios in the offspring of Australian abalone divers and Swedish navy divers. It is also supported by the finding of a significantly low sex ratio in the offspring of men who were exposed to the nematode DBCP10: such men have been reported to have high gonadotrophin but normal testosterone concentrations.11

Workers in industrial medicine might consider using the sex ratios of offspring as a criterion of reproductive risk. Unusual sex ratios of offspring are characteristic of a number of diseases—for example, prostatic cancer,12 hepatitis B,13 multiple sclerosis,14 ototoxicosis,15 and non-Hodgkin’s lymphoma.16

Meanwhile it might be prudent to re-examine the testicular function and sex ratios of offspring of further samples of divers.

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Occupational exposure to dust and lung disease among sheet metal workers

Sir,—The study Occupational exposure to dust and lung disease among sheet metal workers by Hunting and Welch (1993;50:452-42) was an ambitious undertaking.

This correspondence considers the modelling and selection techniques employed, the validity of the work history and exposure modelling, the potential impact of possible selection bias, and the appropriateness of the industrial hygiene evaluation on the fibreglass insulation findings.

In terms of the modelling and variable selection techniques, the final analyses of exposure v chronic bronchitis and non-asthma adjusted even though the confounding effects of age and price are ubiquitous and universally recognized in epidemiological research. Age should have been included in the regression equation regardless of statistical significance if such inclusion changes the estimated coefficients of the risk variables by any appreciable degree.17 Without such an adjustment, the statistical significance of the association between chronic bronchitis and high level fibreglass exposure (ripout) may
be entirely due to the association between age and the lifetime odds of having performed ripout.

Asbestos exposure (which was modelled as a series of exposures) was treated differently from fibreglass exposure (ever/never had a high exposure) in the multiple logistic regression analyses. The tables and text indicate that a fibreglass exposure index based on either the adjusted years of fibreglass exposure or none/moderate/high fibreglass exposure would not have indicated any association between fibreglass exposure and chronic bronchitis.

The median duration of exposure in the "high level" fibreglass group is zero years, and 75% of this group had less than one year of experience at the "high level". It is not biologically plausible that such a fleeting exposure is responsible for symptoms of chronic bronchitis.

Work history and exposure modelling are not adequately considered. It is questioned whether the exposure models are truly able to distinguish qualitatively and/or quantitatively between the exposures of asbestos, welding, and fibreglass, given the high degree of correlation among them. No attempt was made to validate the self-reported exposure models (which are open to recall bias) nor to validate the models of fibreglass and asbestos exposure. More should have been done to validate the exposure modelling assumptions because the paper's conclusions are based on these assumptions (see industrial hygiene comments later).

The overall design of the survey raises important questions about the potential influence of various factors on the study, including the representativeness of the results and the validity of generalising these results beyond the sample.

The survey results are based on less than 40% of those eligible and invited to participate. It relied on data from a previous medical screening in which only 47% of those invited agreed to participate (12,454 of 26,329 sheet metal workers). Of this, 407 (41%) of the workers were selected from United States Sheet Metal and Air Conditioning National Association locals in the southeast sun belt and west coast states. Only 333 (82%) of these 407 completed the interviews.

Unanswered, yet most important questions remain. How did survey eligibility criteria affect results? Are those health related selection factors that influenced eligibility—for example, worked in the sheet metal shop for at least 70% of his working career, did removal for at least 40% of his working career, or welding for less than 20% of his working career? What sort of self selection bias can be overcome to determine if a group that saw asbestos ripout was actually more likely to have high exposure to asbestos and/or fibreglass? What is the true percentage of the male population with significant fibreglass exposure or respirable glass fibres?

The fibre concentrations reported by Balzer, Copper, and Fowler, as well as being total fibre counts, did not differ significantly between glass and fibreglass models. Further, the airborne fibre diameters were well above the respirable range, suggesting that respirable fibre exposure would be lower.

Using NIOSH 7400A analytical methods, airborne dust concentrations were based on concentrations from a wide variety of fabrication and installation operations including pipe insulation, range assembly, duct assembly, duct board installation, water heater assembly, and flex duct assembly ranged from 0.009 fibres/ml (duct board assembly) to 0.007 fibres/ml (general fabrication) for all fibres and 0.002 (duct board installation) to 0.071 fibres/ml (general fabrication) for glass fibres. In no instance did the 95th percentile individual concentration exceed 0.12 fibres/ml. These respirable fibreglass exposure concentrations are similar to average concentrations recently noted in an industrial wool manufacture (all fibres, 0.03 fibres/ml)* and all fibres, 0.025 fibres/ml. Because of these low uniform exposure values, it is not reasonable to divide the sheet metal workers' exposures into high, medium, and low groups.

It then follows that it is difficult to attribute the apparent excess of chronic bronchitis to overexposure to fibreglass. The authors have then faced with the same issues which confronted and confounded Engholm, and Von Schmallese1 and Engholm et al.* Based on the data presented, the paper's conclusion that high intensity exposure to fibreglass causes chronic bronchitis is unwarranted.

**Authors' reply**

Korzen makes a number of criticisms of our study's finding that sheet metal workers with chronic bronchitis were 2.2 times as likely to have performed tasks involving high level fibreglass exposure (that is, ripout of fibreglass materials). We would like to take this opportunity to clarify our methods and rationale.

Korzen has a concern about selection bias. He correctly notes that only 47% of invited sheet metal workers participated in the initial medical examinations from which our sample for interview was drawn. For this study, 407 workers were selected from among those 12,454 initially examined, and 333 (82%) completed a telephone interview. Forty of the 74 non-participants were decreased or otherwise lost to follow up; of those actually contacted, 90.7% completed an interview.

To look indirectly at possible selection bias, we compared baseline (medical examination) characteristics of participants and non-participants from this study; the prevalence of chronic bronchitis was 15% in both groups. Notably, the non-participants (rather than the participants) had spent significantly more time doing installation, repair and ripout work, which generally involved more dust exposure than shop work. Thus it is unlikely that the association between chronic bronchitis and ripout exposures would be biased by participation.

Korzen also questions whether our selection criteria may have biased the results. We selected workers who reported at the baseline medical examination doing primarily shop work (>70% of career) or doing ripout for >40% of their careers. These selection criteria were established to obtain a range of asbestos and fibreglass exposure among participants, with shop workers having more fibreglass and less asbestos exposure, and other workers having a variety of exposures, including high level exposures to both substances. We excluded workers who reported welding more than 20% of the time, in order to exclude this exposure as a major confounder. We do not believe that there would have been exposure and health selection factors simultaneously operating among the workers we would have excluded, for example, how much of such a selection bias could occur would be if workers with lung disease switched from job site installation work to (often) less demanding shop work as they developed symptoms. These workers, however, would not have worked at least 70% of their careers in the shop, and thus would not have been included in this study. We...