## Generalised estimating equations and low back pain - Reply

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## LETTERS

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### Endotoxin: is it an environmental factor in the cause of Parkinson's disease?

Occurrence of Parkinson's disease (PD) has been reported to be associated with environmental factors, notably those associated with employment in the agricultural industry.[1] Some have suggested that the agent associated with agriculture is pesticide exposure, although no specific class of pesticide has been identified.[2]

We suggest that besides pesticides, endotoxin (lipopolysaccaride, LPS) may also be an environmental factor. Endotoxin is a common airborne environmental and occupational contaminate in agricultural[3] and other industries.[4 5]

Endotoxins are part of the outer cell wall of Gram negative bacteria.[6] This agent can elicit a multitude of pathophysiological effects, including inflammation, macrophage activation, fever, and septic shock.[7 8] The blood-brain barrier can become leaky as a result of sepsis,[9] allowing LPS to enter the cerebrospinal fluid.

Experimentally, endotoxin has been shown to cause inflammation in the dopaminergic neurones of the substantia nigra, resulting in pathogenesis of PD.[10 11] LPS stimulate astrocytes and microglia in the CNS to secrete cytokines such as TNF-α, IL-6, and IFN-γ.[10] Microglial activation preceded the apparent neuronal degeneration.[11]

One case study[12] reported that a 22 year old laboratory worker developed Parkinson's syndrome, with bradykinesia, rigidity, tremor, and cogwheel phenomenon, three weeks after accidental exposure to 10 µg *Salmonella minnesota* LPS through an open wound. The LPS caused a chronic inflammation in the nervous system (6600 pg LPS/ml cerebrospinal fluid), which was also characterised by neuralgic pain, polyneuropathy, and encephalopathy, with difficulties in short term memory, learning, and spatial orientation. Damage to the substantia nigra and cerebral cortex was shown by positron emission tomography.

In another case study[13] a laboratory worker would have died without medical help because of severe sepsis after a single injection of 1 mg of *Salmonella minnesota* LPS. This shows the potency of endotoxin in physiological responses.

The case event of PD is supported by animal experimentation.[14] Several animal studies[10 11 14] have shown that LPS causes damage to the substantia nigra, resulting in PD. These animal investigations support the hypothesis that LPS may be one of the environmental factors that trigger PD. A recent study[15] suggests that LPS may be an important contributor to exacerbation of inflammatory disease resulting from particulate matter associated with air pollution. This shows the diverse influences of LPS on physiological systems.

It is suggested that LPS is one of the causes for postencephalitic parkinsonism after encephalitis from Gram negative bacteria. These findings warrant further investigation of this potential environmental factor.

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### Generalised estimating equations and low back pain

We read with interest the article by Hoogendoorn *et al* who examined the use of different approaches to analysing data from their prospective cohort study of work related exposures and the future onset of low back pain.[1]

Exposures and outcomes are time dependent factors as they are subject to change over time. The strength of the relation depends on the assumptions of time dependence (or independence) of exposures and outcomes. The effects of these assumptions can be investigated by adopting different modelling approaches to studies that have collected repeated measures of exposure and outcome data over time.

Hoogendoorn *et al* have adopted such an approach in their study of work related risk factors for low back pain.[1] Information on work related physical and psychosocial factors and low back pain outcome was collected at baseline and in three annual follow ups. They showed an increased risk of low back pain for work related mechanical factors, when using two different generalised estimating equation (GEE) models compared to the standard logistic regression approach.[1] Conversely, for work related psychosocial factors the association with low back pain was weaker when the GEE method was employed. Such an approach is enlightening and we agree that it is important to explore such analytical techniques in the investigation of work related risk factors and musculoskeletal symptoms. Therefore further exploitation of this method of analysis seems appropriate.

We have recently conducted a prospective study of new onset low back pain in 1081 newly employed workers from 12 occupational settings.[2] We examined newly employed workers since studies conducted in well established workforces may be influenced by the healthy worker effect, whereby workers may have changed their job or certain aspects of their job as a result of musculoskeletal pain. In brief, at baseline subjects completed a questionnaire, including an assessment of pain status. A preshaded manikin was used to enquire about low back pain, defined as pain between the 12th rib and the gluteal folds, lasting at least 24 hours in the past month. Individuals free from low back pain at baseline were identified and followed up at 12 and 24 months. The detailed

**Table 1** Work related mechanical risk factors and new onset low back pain*

| | Univariate associations | | | | | |
|---|---|---|---|---|---|---|
| | Logistic regression | | GEE model 1 | | GEE model 2 | |
| Exposure | OR | 95% CI | OR | 95% CI | OR | 95% CI |
| *Manual handling activities* | | | | | | |
| Lifting with one hand | | | | | | |
| Never | 1 | Referent | 1 | Referent | 1 | Referent |
| ⩽15 lb | 2.01 | 1.26 to 3.21 | 1.74 | 1.15 to 2.64 | 1.37 | 0.91 to 2.07 |
| >15 lb | 1.92 | 1.05 to 3.51 | 1.75 | 1.01 to 3.03 | 1.62 | 0.98 to 2.67 |
| Lifting with two hands | | | | | | |
| Never | 1 | Referent | 1 | Referent | 1 | Referent |
| ⩽24 lb | 1.68 | 1.04 to 2.71 | 1.63 | 1.06 to 2.51 | 1.34 | 0.87 to 2.06 |
| >24 lb | 2.50 | 1.43 to 4.37 | 2.27 | 1.38 to 3.72 | 1.78 | 1.11 to 2.85 |
| Carrying on one shoulder | | | | | | |
| Never | 1 | Referent | 1 | Referent | 1 | Referent |
| ⩽30 lb | 0.81 | 0.42 to 1.56 | 0.81 | 0.44 to 1.48 | 1.12 | 0.65 to 1.93 |
| >30 lb | 1.46 | 0.71 to 3.04 | 1.39 | 0.71 to 2.75 | 1.28 | 0.67 to 2.44 |
| Lifting at or above shoulder level | | | | | | |
| Never | 1 | Referent | 1 | Referent | 1 | Referent |
| ⩽23 lb | 1.81 | 1.02 to 3.21 | 1.75 | 1.05 to 2.93 | 1.56 | 0.93 to 2.63 |
| >23 lb | 3.48 | 1.71 to 7.08 | 3.17 | 1.66 to 6.06 | 2.12 | 1.18 to 3.81 |
| Pushing | | | | | | |
| Never | 1 | Referent | 1 | Referent | 1 | Referent |
| ⩽65 lb | 1.63 | 0.94 to 2.81 | 1.52 | 0.93 to 2.49 | 1.34 | 0.83 to 2.14 |
| >65 lb | 1.69 | 0.95 to 3.01 | 1.51 | 0.90 to 2.54 | 1.32 | 0.78 to 2.22 |
| Pulling | | | | | | |
| Never | 1 | Referent | 1 | Referent | 1 | Referent |
| ⩽56 lb | 2.01 | 1.06 to 3.79 | 1.95 | 1.10 to 3.46 | 1.53 | 0.90 to 2.57 |
| >56 lb | 2.96 | 1.68 to 5.23 | 2.72 | 1.63 to 4.52 | 2.05 | 1.23 to 3.42 |
| *Posture* | | | | | | |
| Sitting | | | | | | |
| Do not sit as part of job | 1 | Referent | 1 | Referent | 1 | Referent |
| <2 hours | 0.57 | 0.33 to 1.00 | 0.59 | 0.35 to 0.98 | 0.99 | 0.61 to 1.61 |
| ⩾2 hours | 0.62 | 0.37 to 1.02 | 0.64 | 0.41 to 1.01 | 0.89 | 0.55 to 1.42 |
| Standing | | | | | | |
| Do not stand as part of job | 1 | Referent | 1 | Referent | 1 | Referent |
| <15 minutes | 1.42 | 0.67 to 3.03 | 1.42 | 0.71 to 2.83 | 1.09 | 0.57 to 2.06 |
| 15 minutes to <2 hours | 1.98 | 0.95 to 4.10 | 2.00 | 1.03 to 3.89 | 1.55 | 0.83 to 2.88 |
| ⩾2 hours | 2.37 | 1.09 to 5.13 | 2.27 | 1.12 to 4.59 | 1.75 | 0.90 to 3.40 |
| Drive as part of job | | | | | | |
| No | 1 | Referent | 1 | Referent | 1 | Referent |
| Yes | 1.29 | 0.66 to 2.51 | 1.27 | 0.69 to 2.33 | 1.14 | 0.67 to 1.94 |
| Kneeling | | | | | | |
| Never | 1 | Referent | 1 | Referent | 1 | Referent |
| <15 minutes | 1.02 | 0.61 to 1.70 | 1.00 | 0.63 to 1.60 | 1.40 | 0.91 to 2.16 |
| ⩾15 minutes | 1.85 | 1.04 to 3.26 | 1.79 | 1.07 to 2.97 | 2.05 | 1.28 to 3.28 |
| Squatting | | | | | | |
| Never | 1 | Referent | 1 | Referent | 1 | Referent |
| <15 minutes | 1.40 | 0.86 to 2.27 | 1.37 | 0.88 to 2.11 | 1.11 | 0.73 to 1.70 |
| ⩾15 minutes | 2.19 | 1.19 to 4.04 | 2.12 | 1.22 to 3.67 | 1.81 | 1.08 to 3.05 |
| Bending | | | | | | |
| Never | 1 | Referent | 1 | Referent | 1 | Referent |
| <15 minutes | 0.95 | 0.60 to 1.51 | 1.05 | 0.70 to 1.59 | 1.57 | 1.06 to 2.31 |
| ⩾15 minutes | 1.27 | 0.80 to 2.01 | 1.31 | 0.87 to 1.97 | 1.26 | 0.83 to 1.93 |
| Stretching below knee level | | | | | | |
| Never | 1 | Referent | 1 | Referent | 1 | Referent |
| <15 minutes | 1.42 | 0.93 to 2.17 | 1.43 | 0.97 to 2.10 | 1.34 | 0.92 to 1.94 |
| ⩾15 minutes | 1.28 | 0.67 to 2.45 | 1.22 | 0.67 to 2.22 | 0.91 | 0.50 to 1.68 |
| Working with hands above shoulder | | | | | | |
| Never | 1 | Referent | 1 | Referent | 1 | Referent |
| <15 minutes | 1.40 | 0.86 to 2.27 | 1.40 | 0.90 to 2.18 | 1.60 | 1.06 to 2.42 |
| ⩾15 minutes | 1.49 | 0.91 to 2.45 | 1.43 | 0.91 to 2.24 | 1.56 | 0.99 to 2.46 |
| *Repetitive movements* | | | | | | |
| Repetitive arm/wrist movements | | | | | | |
| Never | 1 | Referent | 1 | Referent | 1 | Referent |
| <2 hours | 1.58 | 0.96 to 2.62 | 1.52 | 0.96 to 2.40 | 1.40 | 0.90 to 2.17 |
| ⩾2 hours | 1.45 | 0.86 to 2.45 | 1.36 | 0.85 to 2.20 | 1.27 | 0.80 to 2.01 |

*Adjusted for gender, age group, and occupation.

questionnaire also gathered information on a number of work related mechanical and psychosocial exposures.

The models used for analysis were identical to those used by Hoogendoorn *et al*.[1] The standard logistic regression model was used to examine the relation between exposures and new onset low back pain at 12 or 24 months. GEE models are used to analyse repeated measures data, by taking the within subject correlation into account, and providing a summary estimate over time. In GEE model 1, the relation between baseline exposures and new onset low back pain at 12 or 24 months was examined. In GEE model 2 that relation was examined for baseline exposures and new onset low back pain at 12 months, and 12 month exposures and new onset low back pain at 24 months.

The two models in which risk factors were assumed to be time independent (standard logistic regression and GEE model 1) produced similar point estimates for developing new onset low back pain (tables 1 and 2), with

**Table 2** Work related psychosocial risk factors and new onset low back pain*

| | Univariate associations | | | | | |
|---|---|---|---|---|---|---|
| | Logistic regression | | GEE model 1 | | GEE model 2 | |
| Exposure | OR | 95% CI | OR | 95% CI | OR | 95% CI |
| *Job demand* | | | | | | |
| Stressful work | | | | | | |
| Never/occasionally | 1 | Referent | 1 | Referent | 1 | Referent |
| At least half of the time | 1.74 | 1.08 to 2.80 | 1.63 | 1.07 to 2.47 | 1.59 | 1.06 to 2.37 |
| Monotonous work | | | | | | |
| Never/occasionally | 1 | Referent | 1 | Referent | 1 | Referent |
| At least half of the time | 1.88 | 1.07 to 3.30 | 1.87 | 1.13 to 3.08 | 1.94 | 1.22 to 3.09 |
| Hectic work | | | | | | |
| Never/occasionally | 1 | Referent | 1 | Referent | 1 | Referent |
| At least half of the time | 1.79 | 1.20 to 2.67 | 1.61 | 1.13 to 2.29 | 1.23 | 0.86 to 1.78 |
| *Job satisfaction* | | | | | | |
| Job satisfaction | | | | | | |
| Not dissatisfied | 1 | Referent | 1 | Referent | 1 | Referent |
| (Very)/dissatisfied | 0.97 | 0.28 to 3.32 | 0.98 | 0.32 to 3.03 | 1.34 | 0.60 to 2.99 |
| *Social support* | | | | | | |
| Support from colleagues | | | | | | |
| Not dissatisfied | 1 | Referent | 1 | Referent | 1 | Referent |
| (Very)/dissatisfied | 2.40 | 0.62 to 9.32 | 2.00 | 0.66 to 6.06 | 1.85 | 0.73 to 4.70 |
| *Control over work* | | | | | | |
| Control over own work | | | | | | |
| At least sometimes | 1 | Referent | 1 | Referent | 1 | Referent |
| (Very)/seldom | 0.95 | 0.51 to 1.79 | 0.90 | 0.50 to 1.61 | 0.74 | 0.37 to 1.45 |
| Learn new things | | | | | | |
| At least sometimes | 1 | Referent | 1 | Referent | 1 | Referent |
| (Very)/seldom | 1.84 | 0.72 to 4.70 | 1.63 | 0.70 to 3.81 | 1.65 | 0.77 to 3.52 |
| *Individual distress (GHQ)* | | | | | | |
| GHQ total | | | | | | |
| 0 | 1 | Referent | 1 | Referent | 1 | Referent |
| 1–2 | 1.08 | 0.70 to 1.68 | 1.09 | 0.73 to 1.62 | 0.97 | 0.65 to 1.46 |
| ⩾3 | 2.51 | 1.52 to 4.13 | 2.26 | 1.47 to 3.48 | 1.44 | 0.94 to 2.20 |

*Adjusted for gender, age group, and occupation.

narrower 95% confidence intervals for GEE model 1. In GEE model 2, where risk factors are assumed to be time dependent, differences were noted for only a small number of variables (carrying on one shoulder, lifting at or above shoulder level, and general health questionnaire score). In addition, the 95% confidence intervals were narrower than those derived from the standard logistic regression and GEE model 1. However, there was no consistent pattern of attenuation or growth noted in either the mechanical or psychosocial risk factors examined (tables 1 and 2).

In summary, we agree that it is important to investigate different statistical techniques in an attempt to determine what effect the assumptions of time dependence (or independence) have on predictors of musculoskeletal pain. However, unlike the study by Hoogendoorn *et al*,[1] our data show that the choice of model has relatively little consistent influence on the magnitude of the results, although GEEs give more accurate estimates.

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## Authors' reply

In response to our paper on a comparison of different approaches to the analysis of data from a prospective cohort study,[1] Harkness *et al* performed a similar analysis on data from their two year prospective cohort study on work related exposures and new onset of low back pain. They agree that it is important to determine the effect of assumptions on time dependence on the observed relations between work related factors and low back pain. They also conclude that their data show that the choice of the model has relatively little influence on the magnitude of the associations.

First, we are pleased with their response to our article, because this is a contribution to the discussion on the design and analysis of repeated measurements studies that we hoped to initiate. Nevertheless, we think that some elements are overlooked in the interpretation of their results.

The most important point is that taking into account repeated measurements in their analysis can only have an influence on the magnitude of the associations observed when there is indeed variability over time in the exposure(s) and/or the outcome measure at issue. Harkness *et al* do not report on the variability over time in the work related mechanical and psychosocial factors, nor on the variability of low back pain in their data. This information would have been helpful in the interpretation of the results presented in tables 1 and 2. Harkness *et al* remark that there was no consistent pattern of attenuation or growth noted in the observed associations for either the mechanical or psychosocial risk factors examined. In our opinion, the differences between the associations observed in GEE models 1 and 2 will not necessarily show a consistent pattern as changes over time are not necessarily the same for different exposures.

Harkness *et al* use exactly the same models as we did. Of course, this increases the comparability. However, it is also important to choose an appropriate temporal position for the exposure window relative to the outcome event in the model that includes time dependent measures of the exposure and the outcome (GEE model 2). Harkness *et al* do not argue why they use the same time lag as we did. Since their outcome measure concerns pain in the past month, and not pain in the past 12 months as in our study, use of no time lag might have been another option to consider.

Finally, we do not understand why the authors adjusted for occupation in the analyses. In our opinion, this may introduce overadjustment because subjects from different occupational settings were included to obtain a contrast in the work related exposures studied.

We appreciate the contribution of Harkness *et al* and recognise that there are still many unanswered important questions regarding the data analysis of cohort studies with multiple measurements of work related factors and musculoskeletal symptoms.

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### Reference

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## Availability of thyroid protective lead shields and their use by trainee orthopaedic surgeons

The sensitivity of the thyroid to radiation is well documented, and a preliminary study[1] suggested an increased incidence of thyroid cancer in Australian orthopaedic surgeons. We have conducted a survey to assess the availability and use of thyroid shields by orthopaedic surgical trainees in the UK.

As most *x* ray radiation exposure to the surgeon occurs during surgery for trauma, 50 hospitals with acute orthopaedic services were studied. The survey was conducted by telephone and post. The orthopaedic registrar on-call was asked if a thyroid protective lead shield was available to them when operating with image intensifier, and if they routinely used the shield. If the registar reported that shields were not available, then the hospital's superintendent radiographer was asked if shields were available to the surgeons.

At the 50 hospitals studied, the registrar at 20 hospitals reported that thyroid shields were available. At the other 30 hospitals, the radiographers at eight hospitals confirmed that shields were indeed not available. At the 20 hospitals where the registrars knew shields to be available, only seven registrars used the shield routinely.

A study by Dewey and Incoll,[2] in which orthopaedic trainees followed their usual radiation protection practice and wore a radiation monitor for a three month period while operating, revealed that 50% wore thyroid shields, and that the exposure ranged from 0.01 to 0.4 mSv. This exceeded the dose limits for the general population in two trainees, but all were within current occupational exposure guidelines. Their data also showed that the dose had a close relationship with the number of emergency operations performed. We have also observed a tendency for the more junior surgeons to get closer to the operation site and more directly into the line of the image intensifier.

Our simple study of hospitals in the UK with acute orthopaedic services has shown a low usage of thyroid protection shields by orthopaedic registrars. Only 14% of registrars surveyed use these protective shields routinely when operating with image intensification equipment. The results indicate that a large proportion of registrars do not use shielding although it is available to them. This implies both a rather casual approach to reducing personal occupational risk, and ignorance of the availability or otherwise of thyroid shields.

Our results also show that 16% of hospitals do not provide thyroid protection shields for use by the surgeon. In some hospitals where thyroid shields are available they may not be readily so. We could not assess the level of this accessibility in our study.

The provision and use of protective shields may be viewed as significant health and safety issues. The evidence is that irradiation of the thyroid region should be minimised. Perhaps all hospitals should furnish adequate protective shielding and render it a requirement that they be used all surgeons undertaking procedures with image intensifier.

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# BOOK REVIEWS



## A Citizen's Guide to Air Pollution

Edited by D V Bates and R B Caton (2nd edition; pp 452 pages; $20.00). David Suzuki Foundation

This is the second edition of a book that sets out to tell the intelligent layman, or citizen, about air pollution: its sources, effects and how it may be controlled. It succeeds. Anybody who reads this book will gain a useful and remarkably up to date grasp of the subject matter and should be able to take a confident part in discussions of and decision making in this difficult area.

The book comprises 11 chapters, arranged fairly conventionally, beginning with the history of air pollution and proceeding via effects on health and the statistical issues raised by studies in that area to effects on vegetation, decision making, and air quality management. A chapter on indoor air pollution is added and a useful linking chapter closes the book. Twelve authors contribute, though in many chapters the original author, David Bates, is a co-author. This has ensured a consistently lucid style.

What, then, of the details? This is not a book that presents all the evidence: it is selective and avowedly so. In these days of systematic review, such a selective approach is often frowned upon. In the section on health effects the selection seems to me very fairly balanced, though the authors do, perhaps, make little of negative studies and do not discuss how such should be interpreted. The thorny issue of publication bias is not touched on. More importantly, the authors do not tell us how important they think the findings reported are or whether they agree with the original investigators' interpretations. This would be useful to the lay reader; such a reader needs assistance from an experienced author.

The chapter "Statistical Issues and Causality" is important and elegant. The primary author, James Zidek, is a professor of statistics: he is ably aided by David Bates. The chapter discusses errors, paradoxes, and misinterpretations of data: the discussion could be useful to any research worker and I have not come across such a clear exposition elsewhere. This is not to say that all the chapter is easy reading. How familiar are you with Popper's notion of intersubjectivity? The authors assert that this, superficially, means that if given all available knowledge, a conclusion is consensually reached by a community possessing that knowledge, then that conclusion would have the status of a fact relative to the knowledge available and the community formulating the conclusion. A reference to the work by Zidek is given. I turned to "The Logic of Scientific Discovery". Zidek and Bates may well be correct but I was left worried about the difference between objectively verifiable propositions and widely held views. In the air pollution field it is certainly true (and verifiable) that widely held views are taken as facts: this needs further thought and discussion. The use of words to describe ideas is a field of study in itself and I cannot resist dealing with what I think is a persistent error of interpretation. The authors draw attention to a statement made in the UK (referring to the question of causality in the context of time-series studies) "that it would be imprudent not to conclude that the association was causal" and describe this as "an interesting use of a double negative". The authors seem to think that the wording implied doubt. They are correct but fail to see that the wording implies less doubt than the alternative: it would be prudent to conclude that the association was causal. The wording was carefully chosen for its force.

In the section dealing with standard setting the authors extol the approach used by the US EPA. This is beyond all doubt exhaustive and open, but is also expensive and few countries other than the USA would wish to adopt such an approach. The climate of litigation in the USA demands such an approach; that climate is regrettable. Valuable insights into cost-benefit analysis are provided, though this is an area in which US policy development is not as clear as it might be and difficult issues remain unresolved. One area that could have stood a longer discussion is that of whether standards should protect individuals or whether they should apply to cities. This is especially important now that time-series studies are being so widely used.

In conclusion, this is an outstanding book that provides a great deal of well assimilated information and raises issues of great importance. The authors and publishers should be congratulated for producing such a useful book at such low cost. It should be read by all interested in air pollution: laymen and those with a professional interest.

**R L Maynard**

## Statistics in Clinical Practice

David Coggon (2nd edition; pp 120; £14.95) 2002. BMJ Books. ISBN 0 7279 1609 2

It is more difficult to write a good short book than a good long book! Textbooks of statistics tend to vary between easy introductions that never seem to tell you how to do the calculations and large ones that tell most of us too

much. The desire, common among mathematicians, that we should understand the origins of all the methods, makes the bigger books hard going and, in practice, many research workers use computer software packages to do the analyses. How long must it be since anybody calculated a standard deviation for themselves using the "short cut formula"? Of course, unthinking application of packages is dangerous. Even when properly applied by experts, problems will arise: this year's discovery of the errors in the Generalised Additive Models much used in air pollution work proves the point.

David Coggon is both a distinguished medical epidemiologist and a mathematician. He has provided a book that is easy to read and yet rigorous in the way in which concepts are developed. Very few formulae are included: the computer program approach is accepted. The rigour is most clearly seen in his analysis of bivariate data: 10 combinations (5 types of variable, arranged in pairs: 5!/2!(5–2)!) and in the chapters dealing with statistical modelling and with statistical power and sampling. This latter chapter has the clearest definition of statistical power, in terms of the probability of a type 2 error, that I have seen. A little more discussion of the relative importance of avoiding type 1 and type 2 errors would have been useful. Correlation coefficients are discussed in useful detail, though I could not find a definition of the much quoted statistic $r^2$: the coefficient of determination.

Each of the nine short chapters ends with a set of questions. These are very well designed to test how well the reader has understood the chapter. Answers, rather more explanatory than usual in statistics books, are provided. Reading the questions and answers is a short cut to knowledge! Many years ago, *School Algebra by Hall* (568 pages), was the standard O level text: even more useful was Grenvilles *Key to Hall's School Algebra* (699 pages) which took apart every problem set by Hall and explained how to solve them. Oh, for such a book on statistics: perhaps the author should write one!

In conclusion, David Coggon has written probably the best short introduction to practical statistics. All doctors interested in research should read it.

**R L Maynard**

## Doctors and Patients. An Anthology

Edited by Cecil Helman (pp 162; £19.95, paperback) 2003. Radcliffe Medical Press. ISBN 1 85775 9931

How do doctors see their patients and how do patients see their doctors? This book sets out to explore these questions via the works of well known medical authors including Conan Doyle, Cronin, Somerset Maugham, Chekhov, and Oliver Sacks and by those of patients including the authors Clive Sinclair, Ruth Picardie, and Renate Rubinstein. Fictional accounts are mixed with direct testimony. Much can be learnt from these accounts. And how attitudes to patients have changed! Conan Doyle's *Tales of Adventure and Medical Life* are well known but not easy reading today. At first glance the paternalism is cloying and repulsive—on a second glance the concern and care for the whole patient is obvious. Conan Doyle was writing in an age of therapeutic impotence and was familiar with the slow decline and inevitable death that characterised so many diseases. He was also familiar with surgical heroics: less admired in this radio- and chemotherapeutic era. "There is nothing surgical which Hargrave has not the skill and audacity to do". A higher standard of writing is set by Somerset Maugham. His short story *Sanatorium* is as good an example of this literary form as you are likely to find outside Kipling. The isolation from the world and the growing gap between patients and relatives presages Solzhenitsyn's *The Cancer Ward* and yet ends on a note of hope.Cancer figures strongly in this book and the true accounts from the patients' standpoint are rewarding. Ruth Picardie's two pages (extracted from *Before I Say Goodbye*) moved me to tears: read it and weep.

The editor, a doctor, social anthropologist, and author, has contributed an outstanding introduction: all doctors and medical students should read this. He addresses the most difficult issues: telling bad news, the disillusionment of doctors and patients, how to handle uncertainty. He quotes Johnson in support of being told the truth (he could have quoted JBS Haldane too) and gives us some splendid quotations: "If you can't be a king, be a doctor" (Indian proverb), "one has a greater sense of intellectual degradation after an interview with a doctor, than from any other human experience" (Alice James), as well as the better known ones from Hippocrates and Hutchinson.

Books of short stories make good bedside table books. This is one that any doctor would benefit from reading—and it is an ideal gift for a student or young doctor. It puts the whole patient first—isn't that what medicine is all about? Buy it.

**R L Maynard**

# NOTICES

## NIVA Training Programme 2003: Advanced Courses in Occupational Health and Safety

NIVA Training Programme 2003 offers 12 advanced courses on current themes of work life. Further information is available from the NIVA Office:

NIVA Nordic Institute for Advanced Training in Occupational Health
Topeliuksenkatu 41 a A
FIN-00250 Helsinki
Finland
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Fax: +358 9 4747 2497, +358 9 2414 634
Email: niva@ttl.fi
Website: www.niva.org

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 Levitunturi, 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

## The Scottish Group of the Society of Occupational Medicine, Meetings 29 May and 12 September 2003

The Scottish Group of the Society of Occupational Medicine is organising the following meetings in 2003:

- Spring meeting: Past, Present, and Future; 29 May 2003, The Royal College of Physicians and Surgeons, 242 St Vincent Street, Glasgow
- Autumn meeting: Health and the Wider Environment—a symposium to mark the retirement of Anthony Seaton; 12 September 2003, The Royal College of Physicians, Queen Street, Edinburgh

Registration
For further details and registration form, please contact:

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# Generalised estimating equations and low back pain

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