

# PostScript

## LETTERS

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### Personal and static sample measurements are related

The paper from Harrison and his co-workers<sup>1</sup> and the subsequent correspondence<sup>2,3</sup> has reignited a debate about the relation between personal and static sample measurements that started more than 40 years ago. In 1957 the personal sampling pump had just been invented by Jerry Sherwood and Don Greenhalgh from the UK Atomic Energy Authority.<sup>4</sup> They compared their new personal sampler with the conventional static sampler and showed that personal exposures were generally higher than those made at a fixed location. This classic paper has recently been reproduced in the electronic edition of the *Annals of Occupational Hygiene* together with a commentary on its significance to the science of human exposure assessment.<sup>5</sup> In this commentary, information concerning personal and static measurement results from papers published in that journal over the past 10 years was reviewed. This showed, as Lange asserts in his letter to this journal,<sup>3</sup> that personal samples are "generally higher in concentration than static samples". In this analysis more than 80% of the personal

measurements exceeded the corresponding static sample concentration. The median ratio between personal and static concentrations was 1.5, although the individual data points ranged from 0.4 to 10. It is reasonable to expect that in general, personal exposure would be greater than static samples if on average workers spend a proportion of their time close to sources of the hazardous substance.

Lange poses the question "are personal and static samples related?".<sup>3</sup> The answer must be "yes", but perhaps a more pertinent question is: how can the relation between personal and static samples be useful in epidemiological studies or risk evaluations? A simple conceptual model, shown in fig 1, is sufficient to convince us that there must be a relation between personal and static monitoring data.

Here the air compartments represent the local external environment, room air (where static samples are obtained), breathing zone air (where personal air samples are collected), and the inhalation of contaminants into the nose or mouth. A key assumption here is that the contaminant is thoroughly mixed throughout each compartment. In addition, in the model we have the potential for airborne contaminants to adsorb or sediment onto room surfaces and for this contamination to become resuspended in the air. In general there is the potential for contaminants to be exchanged to and from each compartment; for example, as air flows from the breathing zone to the body of the room, it is replaced by air from the room. The purpose of showing this model is to illustrate the complexity of the processes relating room and breathing zone air concentrations; this is almost certainly the main reason why there is such a wide range in the ratio of personal and static air concentrations. Key factors in determining the relation between personal and static measurements in any situation will include the volume of the room, the quantity of general ventilation, the time the person spends in the proximity of sources of hazardous substances (that is, with a source in their breathing zone), the presence of other internal or environmental sources of the contaminant, and others. In most circumstances, without knowing something

about each of these factors it is impossible to predict what the relation between personal and static concentrations might be.

There is one class of situations where room samples and personal samples are likely to be very similar. Using a simple mathematical model, Cherrie<sup>6</sup> showed that in small poorly ventilated rooms it makes little difference whether the concentration is measured in the breathing zone compartment or in the room compartment because the contaminant quickly mixes throughout both spaces. In most domestic situations it is likely that this is the case since the rooms are generally small and the ventilation rate is likely to be low, probably less than one air-change per hour. Therefore, I think almost uniquely, in epidemiological studies where we want to assess the exposure of people in houses it probably does not make a lot of difference whether we use samplers located in the room or samplers located in the person's breathing zone. This will mostly not be the case in occupational epidemiological studies, where spaces are typically larger and ventilation rates greater.

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### Lifetime risk of silicosis death for quartz exposed workers among German population

In a recent article of *Occupational and Environmental Medicine*,<sup>1</sup> Manneje *et al* presented quantitative evidence of an exposure-response relation between occupational exposure to crystalline silica and silicosis mortality in a carefully designed pooled analysis. This paper impressively showed that simple silicosis, one of the oldest occupational diseases, is still a relevant occupational health problem nowadays which may have a negative effect on the life expectancy of silica exposed workers. The quantitative evidence (exposure-response relation) presented in this paper provided a sophisticated basis for quantitative assessment of the absolute risks of silicosis deaths for workers exposed to different level of crystalline silica.

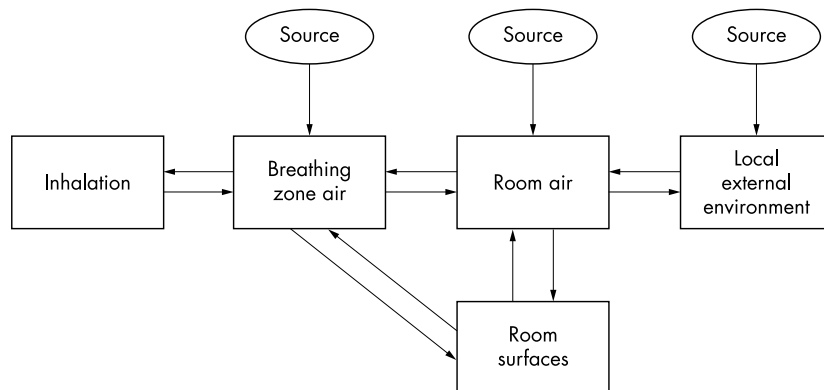


Figure 1 A conceptual model of inhalation exposure.

**Table 1** Estimated lifetime risk of silicosis death for German workers exposed to crystalline silica for 45 years

Exposure level	Estimated lifetime risk* (per 1000)	
	Latency not considered	Latency considered for 28 years
0.05 mg/m <sup>3</sup>	5.3	0.7
0.1 mg/m <sup>3</sup>	11.4	1.6

\*Estimation was conducted by using the exposure-response relation given by Mannetje *et al* and life table of German population in the year 1995.

In this manuscript, the authors exemplarily quantified the risk of silicosis deaths for workers exposed to crystalline silica at exposure levels of 0.1 and 0.05 mg/m<sup>3</sup> for 45 years. The estimated lifetime risks of silicosis death were 13 and 6 per 1000, respectively. The authors concluded that, due to exposure misclassification and possible under-report of silicosis deaths, the lifetime risks of silicosis deaths may be underestimated.

Based on the estimated exposure-response relation provided by Mannetje *et al*,<sup>1</sup> we recalculated the lifetime risk of silicosis deaths for quartz exposed workers by using the life table of the German population in the year 1995. The results of our calculation are presented in table 1. If latency of silicosis death is not considered in our calculation, we get nearly the same results as those given by Mannetje *et al* (11.4 and 5.3 per 1000 at exposure levels of 0.1 and 0.05 mg/m<sup>3</sup> for 45 years, respectively). However, Mannetje *et al* reported in their manuscript that workers who died of silicosis had a median duration of exposure of 28 years and only 9% of silicosis death occurred within one year after leaving the job. The median latency of silicosis death may, therefore, account for at least 28 years. If we consider this latency period (28 years) in our calculation, we get a lifetime risk of silicosis death of 1.6 and 0.7 per 1000, respectively. These values are about seven times lower than our previous estimation without considering the latency period. We believe that these values are more likely to be overestimated, since the assumption of a 28 year latency is the most conservative assumption (period after leaving jobs was not accounted into the 28 year latency period). Furthermore, this latency was estimated from workers exposed to a much higher level of quartz (median cumulative exposure of 7.15 mg/m<sup>3</sup>-year) than the maximal possible exposure level in our calculation (maximal cumulative exposure of 4.5 mg/m<sup>3</sup>-year). Therefore, longer latency period should be considered in a more realistic assessment of lifetime risk of silicosis deaths. We wonder whether Mannetje *et al* have considered latency period in their calculation of lifetime risk of silicosis deaths.

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## Reference

- 1 Mannetje A, Steenland K, Attfield M, *et al*. Exposure-response analysis and risk assessment for silica and silicosis mortality in a pooled analysis of six cohorts. *Occup Environ Med* 2002;59:723-8.

## BOOK REVIEW

### The Illustrated Guide to Functional Anatomy of the Musculoskeletal System

Rene Cailliet (pp 310). American Medical Association Press, USA, 2004. \$69.95, ISBN 1-57947-408-X

This new book from Rene Cailliet is primarily intended for medical clinicians and medical trainees; it is designed to detail the normal functional anatomy of the musculoskeletal system, with a view to practitioners eliciting an understanding of how various musculoskeletal impairments might occur, and developing suitable diagnostic regimes and therapeutic approaches. Dr Cailliet has written a number of excellent texts over the years and most clinicians will be aware of his books on topics such as disorders of the musculoskeletal system, low back pain, and hand pain and impairment. Overall this current book is another good addition to this collection.

Following a fairly full introductory chapter on the concepts of functional anatomy, the book is broken down in a regional approach with separate chapters detailing functional anatomy in the lumbosacral spine, cervical spine, shoulder, elbow (plus wrist, hand, and fingers), knee, hip joint, and foot and ankle. In each case the text is well written and informative and largely logical in format; however, there is a tendency to over explain some aspects of the anatomy, while other aspects are quite sparse in detail, even though they are equally important areas. In the case of the lower limb, for example, there is no mention of the limb rotation that occurs during development, which allows an appreciation of flexion and extension at the ankle joint and thus the position of the flexor and extensor muscles. Being a functional anatomy text, it is also a little disappointing that in some cases explanations of muscle attachments are rather simplified and there is no real mention of anatomical variation. Therefore I would suggest that some readers may need to refer to other texts, including anatomy atlases, to support and enhance the functional descriptions given.

As the title of the book suggests, this is an illustrative guide, and as such it contains a multitude of clear and uncluttered figures that graphically illustrate the anatomical function explained within the text. Although these figures are in the majority of cases effective and of good quality, the lack of full colour, I feel, is a missed opportunity.

Although presumably to keep the costs of the book down, the use of only two-colour artwork will probably result in not inspiring all its potential readers (especially medical students to which the book is also aimed), and also means that the illustrations are not always as easy to interpret as they could be. The figure legends are in most cases self explanatory, with all abbreviations defined. Where legends are less clear, reference back to the main text should elucidate full understanding.

The inclusion of extensive reference sections at the end of each chapter is to be welcomed, although for many readers the clear and authoritative nature of the text means that further reading may not be required. The inclusion of a short glossary at the back of the book in future editions would prove useful to readers unfamiliar with some of the terms used.

Overall, this book is an authoritative and a generally well written text, and despite my limited concerns over the illustrations I would recommend this as a useful and informative addition to most libraries and clinical practices.

D J R Evans

## NOTICE

**International Conference on Occupational Health Services 2005, 25-27 January 2005, Marina Congress Center, Helsinki, Finland**

### Main themes

- Current status of occupational health services
- Evidence base in occupational health service practice
- Health and economic impact
- Vulnerable groups and high risk sectors
- Human resources in occupational health services.

### Who should attend?

Researchers, OHS practitioners and other health professionals, legislators, administrators, medical and other inspectors of labour.

### Call for abstracts

Participants are welcome to present free communications on the topics of the conference. The deadline for abstract submission is 30 September 2004.

### Further information

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