Letters

Should office workers spend fewer hours at their computer?

In the April issue, IJmker et al suggest that reducing work time is an answer to the discomfort and pain that computer users often get (Occup Environ Med 2007;64:211–22). However, if the discomfort and pain is the result of a combination of muscle tension and time, as comments in the section on biological plausibility seem to suggest, is this the only possible strategy?

Limiting the painful effects of computer use (clearly worthwhile) by time alone may be superfluous and counterproductive. Many anecdotal reports from computer users indicate that having to limit the time at the keyboard, or being suddenly frozen out, can be frustrating.

Is there an alternative? A better solution could be to build a device into the mouse that integrates the following: the length of time for which the mouse is moved; the precision in the movements made; the distance over which it is moved; the acceleration of the mouse; the force with which it is gripped; and the rate of clicking.

Research would be needed to find out if tension in the arm muscles is reflected somehow in mouse movements and grip, and if a suitable weighting for each factor can be found. The display of feedback about performance would be an interesting challenge for a student of information design. Software is currently available that interrupts users by measuring the keystroke rate and the time spent keying and combining these in some way. The authors’ findings, indicating that mouse use is more associated with discomfort than keyboard use, suggest the device described could be more beneficial.

In the absence of such a device, does the suggestion to limit time have validity? As noted above, some approaches to achieving this can have disadvantages.

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Competing interests: None declared.

Occup Environ Med 2008;65:147

Authors’ response

Mr Darby raises some interesting points regarding the prevention of discomfort and pain among computer users. The main topic is whether the suggestion to limit computer time has validity in preventing hand-arm-wrist and neck-shoulder symptoms.

We agree with Darby that reducing the duration of computer use without breaks may not be the solution for all workers. At present, there is uncertainty regarding the positive effects of breaks in preventing hand-arm-wrist and neck-shoulder symptoms, because high quality intervention studies are lacking and the results from the available evidence are inconsistent.1 2 In addition, a study by Veiersted3 among assembly line workers might fit into the idea that rest breaks might not be productive in preventing symptoms for some workers. He found that during enforced machine stops future patients showed higher muscle tension compared with employees who remained healthy.

In addition, Blangsted and co-workers4 showed that increasing the duration of rest breaks does not per se cause muscle relaxation. High quality studies are needed in this field.

Darby also notes that reducing time alone may be “superficial”, given the supposed underlying mechanism (sustained muscle activation). Darby notes a number of factors that might relate to muscle tension during mouse use. We agree on these factors. However, in our opinion there are other factors to be considered as well, if we assume that sustained muscle activation plays a leading role in the development of symptoms. Factors to be added to the list might be cognitive demands5 and psychosocial demands (such as time pressure, see Visser et al). Despite the indications from experimental lab studies, longitudinal field studies examining the additive and/or multiplicative effect of these factors, in combination with the duration of mouse use per se, are lacking. Currently several longitudinal studies are in progress that are using software to register computer use and associated factors. We hope that these studies will provide more insight into these issues.

Contrary to what Darby states, modern break-reminder software already registers mouse use. This information is integrated with information on keyboard use into algorithms to provide feedback to the user to take a break from computer work. However this kind of software does not register all the factors listed by Darby. For example, the gripforce while pointing the mouse is not registered.

Finally, we would like to note that the review included a limited number of studies, which where all based on self-reported duration of computer use. Future studies, especially those with objective recordings of computer use, might change our conclusions. Also, the pathophysiological link we made between mouse use, sustained muscle activation and symptoms has been criticised.7

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Occup Environ Med 2008;65:147

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Trichloroethylene exposure and non-Hodgkin’s lymphoma: supportive evidence

We appreciate the interest in our recent meta-analysis of occupational trichloroethylene (TCE) exposure and non-Hodgkin’s lymphoma (NHL) (Occup Environ Med 2007;64:332). Three criticisms were mentioned as being “serious limitations”: (1) the alternative descriptions of the Group I occupational cohort studies (multiple industry vs aerospace, incidence vs mortality and Europe vs US studies) should have been characterised and discussed a priori; (2) that mortality and incidence data should not be combined in a meta-analysis; and (3) our interpretation that the epidemiological data are not supportive of a causal relation is wrong and it is suggested that our analysis provides more evidence of a causal effect between TCE exposure and NHL. These criticisms, however, are not serious limitations and have little relevance in interpreting the meta-analysis findings. We also disagree that our meta-analysis provides further support for a causal association.

The author would have preferred that we consider interpretation of potential sources of heterogeneity a priori. The evaluation of heterogeneity across individual studies is a critical component of any meta-analysis and has not been addressed in previous studies.1 2

We did consider different approaches to stratification a priori, but preferred to discuss the interpretation of this after we had analysed these (and other) potential sources of heterogeneity. Before conducting a quantitative analysis of heterogeneity, interpretation of potential findings would be mere speculation.

With respect to combining incidence and mortality data, we agree that incidence data are generally preferable if available. In our meta-analysis of subgroups of studies, we stratified studies on this characteristic and, in fact, the analysis of mortality studies was more homogeneous (table 2). More importantly, if TCE exposure were causally associated with NHL, we would expect both incidence and mortality rates to reflect this.
Finally, with respect to causal interpretation of the available data, it is our opinion that causal inference needs to be made based on a comprehensive evaluation of the data. Such an evaluation is not limited to the calculation of relative risk estimates and confidence intervals (or p values), but also includes evaluation of exposure-response consistency, and other factors, in addition to potential sources of bias.1-4

Results were inconsistent across the different groups of studies (for example, considering Group I, Group II and the case-control studies), available data in the Group I studies did not indicate exposure response trends, there were significant limitations with respect to exposure classification across all studies, and there was variation in findings across subgroups within the Group I cohort studies. These observations considered together led us to conclude that epidemiological data of occupational TCE exposure and NHL were not consistent with a causal association.

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Occupational medicine and asthma

The article by Francis et al (Occup Environ Med 2007;64:361–5) is interesting and makes an important contribution to clinical practice. It is a pity, however, that a consensus on definition did not precede the production of evidence-based guidelines1 to make sure that the relevance of the evidence was consistent with the emerging consensus.

I note that the consensus panel comprised a group of experts in occupational respiratory diseases based in major hospitals. As far as I can tell only two of the panel are accredited specialists in occupational medicine. While the individual credentials of the panel members are impeccable, full participation by practising occupational physicians rooted in the workplaces where exposure occurs would have given better balance to the panel as a whole.

I am left wondering if the absence of practising occupational physicians is an unfortunate oversight, a reflection of how uncommon occupational asthma is in the everyday practice of occupational medicine, or a tendency by doctors to focus our attention on the most serious presentations of respiratory and other diseases.

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Occup Environ Med 2008;65:148

Definition of occupational asthma

Francis and her colleagues (Occup Environ Med 2007;64:361–5) have completed an interesting and useful piece of work in relation to a definition of and diagnostic resources required for occupational asthma. It is, in my view, however important to identify, in full, the context of such definitions if they are not to be used for unintended purposes or, perhaps, inappropriately. Is it possible to suggest that “occupational asthma” used in terms of identifying work factors important to be controlled in that individual’s protection could be different from a definition of occupational asthma in terms of regulatory or litigation issues where, perhaps, part of the definition could be in relation to a breach of a statutory duty of care? I, at least, do not believe that these two issues are the same in relation to asthma and to a number of other health conditions that have some work relation. I wonder if the authors would agree that the deliberations in terms of scope might have been different if their starting definition had included considerations of employers’ liability as well as a clinical basis?

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Occup Environ Med 2008;65:148

Authors’ response

We thank Dr Preece for his comments. We believe that he is justified in questioning the make-up of the panel and that this has a significant bias for tertiary assessment of occupational lung disease. However, our aim in performing this process was to get this group of experts to agree on “definitions” with a view to unifying the label at this later clinical stage of the process.1 In addition, we hoped that the requirements of a specialist occupational lung disease assessment service would form a basis for developing a “standards of care” document for those giving expert medical advice in this specialist field. We felt that it was appropriate to carry out the process from this standpoint, as if this body could not agree on what constitutes occupational asthma or work aggravated asthma in principal, how could we work towards an agreed process for evaluation at all levels?

A similar exercise done in parallel using occupational physicians would be a very interesting and valuable one. It is likely that the view of workplace-based physicians will be different because of local variations in perspectives. We reiterate that it was never intended for this to be within the scope of this project. In carrying out the process in this way we certainly did not wish to alienate our occupational physician colleagues and in no way intend to imply that their viewpoint is not valued. We thought it was valuable to share the perspectives from the specialist occupational lung physician, in publishing our findings.2

Finally, we note the comment regarding the evidence-based guidelines and while there is some practical relevance of the consensus to the guidelines and joint interested parties on the two documents, it was not practical to include the consensus in the guidelines. Doing so would have caused a significant delay in publishing the guidelines that was not merited as, after all, consensus is the least important and lowest level of evidence base.

We thank Dr Kalman for his views and understand his issues with regard to context, a point similar to that made by Dr Preece. We performed this study specifically to deal with producing the definition on which specialist centres should base their assessment of cases of possible work-related asthma. The “facilities required” component was specifically aimed at identifying standards of care for this process. While the definitions are of relevance to the workplace and legal situations, we thought that a firm basis for discussion and minimum criteria required to assess difficult cases appropriately was an important issue and the sole basis for this study. The requirement of a breach of statutory duty of care does not affect the clinical diagnosis or management of a worker in the workplace. We think that a medical diagnosis (such as occupational asthma) should be based on what is wrong with the patient and what is the cause.

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Occup Environ Med 2008;65:148

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Occup Environ Med 2008 65: 147-148
doi: 10.1136/oem.2007.035212

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