of working conditions on cardiovascular health. This bias would be larger among women, as a considerable proportion has not worked regularly outside the home, and minor among men, as almost all men are (or have been) working.

CONCLUSION
The calculations presented in this paper give a provisional impression of the quantitative impact of working conditions on cardiovascular diseases. The impact appears to be rather large. We feel that work environment factors ought to play a bigger role in cardiovascular epidemiological research in the future and also that the cardiovascular diseases should be acknowledged as work related diseases in the same way as cancer, reproductive failures, lung diseases, and many others. It is evident that more—and better—research is needed, but this fact ought not be used as an excuse to postpone preventive programmes aiming at improving the work environment.

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11 Miettinen OS. Proportion of disease caused or prevented by a given exposure, trait or intervention. Am J Epidemiol 1974; 99: 323–32.

Commentary
The effects of occupation on the incidence of cardiovascular disease have been seriously underreported in published work. Public health epidemiologists tend to concentrate almost exclusively on life style factors while occupational health professionals prefer to report the influence of work on the incidence of cancer, reproductive outcome, or musculoskeletal disorders. The imbalance is partly restored by this thought provoking paper of Olsen and Kristensen.

The standard texts in occupational medicine give little space to cardiovascular disease despite the fact that it is a major contributor to Western mortality and morbidity. Two recent and thorough reviews by, incidentally, one of the authors of the current paper, cite a number of chemical and non-chemical workplace factors which can (or might) influence cardiovascular disease.1,2 Of the chemical factors, carbon disulphide and nitroglycerine/nitroglycerol are well recognised causes, although recent studies suggest that carbon disulphide may act more through a reversible, direct cardiotoxic or thrombotic influence than through previously assumed atherogenic effect.3 Nitroglycerine can cause both acute and chronic effects and is well known for the disturbing “Monday morning death” syndrome. Some chemicals, such as methylene chloride, cause carboxyhaemoglobinemia while other chlorinated hydrocarbons appear to cause cardiac arrhythmias. Inconclusive effects, possibly in some cases secondary to hypertension, have been ascribed to lead, cadmium, arsenic, and antimony. Recent reports suggest that epichlorohydrin4 and hydro sulphide5 might be added to the list. Peripheral vascular effects certainly arise from workplace exposure to cold, vibration and vinyl chloride monomer.

While chemical exposures are relatively rare, the influence of putative circumstances such as work “stressors”, shift work, and sedentary occupations has received less attention but might be of greater preventive health importance. The difficulty has always been how to tease out these influences from life style when reviewing the mass of health data related to working populations. Olsen and Kristensen have attempted to do just that.

Using a scoring system for the quality of epidemiological studies, they calculated crude relative risks using quality in combination with quantity and consistency of findings in published reports. Combining this with a rough guide to the
prevalence of probable risk factors—such as noise, sedentary occupation, shift work, monotonous high paced work etc, they come to the conclusion that workplace related cardiovascular mortality is a sizeable proportion of the total. For males, they reckon that 16% of premature cardiovascular deaths could be avoided by “workplace interventions”. The figure is 22% for females and rises to over 50% for both sexes if sedentary occupation is taken into account. Monotonous high paced work and shift work account for most of the aetiological fraction.

Olsen and Kristensen can be criticised for a number of the assumptions they make in their paper—particularly the assertion that independent risk factors will act multiplicatively. The mere fact, however, that they have attempted to assess occupational influences on cardiovascular disease is to be commended. If all their calculations were correct—and they admit that they are crude approximations—then preventive medicine practitioners had better start looking seriously at cardiovascular workplace factors. At the very least, this paper should start an interesting debate. At best it could be the first step in diminishing cardiovascular risk factors which are more readily altered than those associated with genes or life style.

J M Harrington
Institute of Occupational Health
University of Birmingham