Pollution

Outdoor air pollution and DNA damage

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Commentary on the paper by Tovalin *et al* (see page 230)

Although working outdoors has frequently been considered more healthful than working indoors, a growing literature suggests that outdoor air exposures increase the risk for a variety of diseases, such as asthma, heart disease, and lung cancer.[1 2] Consistent with these epidemiological studies are reports using the ^{32}P-post-labelling assay for stable DNA adducts showing that outdoor air causes DNA damage,[3] which is a prerequisite for most mutation and cancer. Supporting these observations are hundreds of studies using primarily the *Salmonella* mutagenicity assay showing that the particulate and volatile fractions of outdoor air are mutagenic.[4]

As reviewed by Tovalin and colleagues[5] in this issue of *OEM*, a few studies have used the single cell gel electrophoresis (comet) assay to assess DNA damage associated with outdoor air pollution. However, unlike many of the studies using the ^{32}P-postlabelling assay, studies using the comet assay have not included the personal monitoring of pollutants in order to link the DNA damage to specific components of outdoor air. With one exception, these studies have generally examined only those tissues exposed directly to air (epithelia from the nose, tear ducts, or buccal region), and they have not compared DNA damage in people working outdoors to those working indoors.

By incorporating personal monitors into their study, Tovalin and colleagues[5] have begun to provide a link between the exposure assessment of outdoor air pollutants and systemic DNA damage as measured by the comet assay associated with outdoor air pollution. The authors show that relative to indoor workers, outdoor workers have higher levels of systemic DNA damage that is associated with levels of particulate matter (PM) and 1-ethyl-2-methyl benzene (as measured by personal monitors) and ozone (estimated by modelling).

Gaseous and particulate emissions from sources such as power stations, various industries, and vehicles, along with their atmospheric transformation products, cause damage to public health and to the environment.[1 2] In particular, recent prospective studies have confirmed previous findings[1 2] by showing that exposure to inhaled (PM_{10}), fine ($PM_{2.5}$), and ultra-fine ($PM_{0.1}$) particles in outdoor air is associated with an increased risk for lung cancer.[6 7] Although the levels of ozone in outdoor air also have been associated with an increased risk in lung cancer and DNA damage, the causal nature of this relationship is unclear.[8–10]

Tovalin and colleagues provide mechanistic support for these epidemiological observations by showing that the DNA damage in outdoor workers was associated with $PM_{2.5}$. This is the first clear demonstration that particles of this size in outdoor air are associated with DNA damage in humans as measured by the comet assay. This emphasises the importance of small particles in outdoor air as the cause of fatal diseases. Small particles are more mutagenic than large particles, and air with a higher concentration of small particles is more mutagenic than is air with larger particles.[4]

Tovalin and colleagues also found an increased level of alkali labile sites in outdoor workers relative to indoor workers. This type of DNA damage is likely due to oxidative stress, consistent with the observed association between DNA damage, $PM_{2.5}$, and ozone. Mechanistic studies of DNA damage using the comet assay in cultured human fibroblasts exposed to urban dust particles indicate that the genotoxicity of PM is due to a combination of DNA adduct-forming polyaromatic compounds, oxidising agents, as well as the insoluble particle core itself.[11]

The advantages and limitations of the comet assay for human biomonitoring have been discussed.[12] Supporting the use of the assay are the results obtained in the present study, which are consistent with other measures of DNA damage, most notably the ^{32}P-postlabelling assay for stable DNA adducts and assays for haemoglobin adducts.[3] The use of these and other biomarkers of DNA damage and mutation in studies of outdoor air pollution is critical to understanding the mechanisms by which polluted air leads to human disease.[3] A recent statistical analysis of published biomarker studies showed that biomarkers had smaller variance ratios than did air measurements, suggesting that biomarkers would provide a less biasing surrogate for exposure than would typical measurements of chemicals in the air.[13]

The work of Tovalin and colleagues adds to the growing body of data indicating that genotoxic substances in outdoor air result in systemic DNA damage, some of which is likely due to oxidative stress, and highlights the value of biomarkers for assessing responses and exposure to outdoor air pollution. Because the work of Tovalin and colleagues is a small pilot study, the results need to be interpreted cautiously. Nonetheless, further work in this important area should be encouraged and supported.

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Sickness absence

Ill health, social protection, labour relations, and sickness absence

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Commentary on the paper by Virtanen *et al* (*Occup Environ Med*, March 2006)*

Health and work have a bidirectional relationship. Hazardous work can produce negative effects on health, in terms of injury and disease. It is less evident that ill health, due to the work environment or not, can produce negative effects on work in terms of absenteeism and low productivity. Sickness absence is an expression of this complex relation. In the first place sickness absence is necessary for the recovery of ill workers,[1] as part of the medical treatment, and as such it is certified by a physician. In a second and complementary perspective, sickness absence is a consequence of ill health on work in terms of absence from work. Indeed, it has been considered as an indicator to measure the working population's health status,[2] and also it may be considered as an indicator to measure the functioning of the companies.[3] In other words, sickness absence might be considered as something else beyond simple ill health.[4]

Taking the natural history of diseases as an analogy, a natural history of sickness absence illustrates that ill health (for example, a common cold) could develop into a sickness absence spell or not. There is solid evidence that whether this happens or not, depends on the working conditions of the job (for example, high physical demands), and on personal characteristics of the worker (for example, older age). However, what is very often forgotten is the role played by a third determinant, namely the social protection system, for example, percentage of benefits covered. For instance, a 50 year old manual worker who has a common cold and has social protection benefits that only cover 60% of the salary and do not pay the first three days of sick leave, probably he/she will not take a sickness absence spell. In addition to ill health, and after assessing their working and personal conditions, ill workers will consider what their social benefits are in the case of taking sick leave. It is likely that many workers take all these considerations into account before going to the doctor and asking for a sick leave certificate.

This third determinant (social protection system) is critical to explain the progress from ill health to sickness absence, and is often omitted in our analyses, and interpretations.[5 6] In the current economic period, public policies from governments act essentially on this determinant to try to control the budget of their social protection system, reducing the benefits of workers with sickness absence. At the same time public policies do not take into account the workers who work while ill. This situation, which has been called ''presenteeism'',[7] could increase the risk of more severe diseases.[8]

In the March issue of this journal, Virtanen and colleagues,[9] from the ''Finnish Public Sector Cohort Study'', provide valuable evidence about the complex association between health and work, specifically the role of the labour relations. Among public sector workers, a high rate of sickness absenteeism significantly increases the risk of job termination and unemployment in temporary employment. Conversely, permanent workers with high rates of sickness absence appear to be protected against job termination and unemployment by job security and disability pensions. Ill workers have to introduce a new variable in their algorithm before going to the doctor and asking for a sick leave certificate: sickness absence may affect their employment status. Despite temporary workers having less sickness absence than permanent workers,[10] sickness absence in temporary workers seems to be a predictor of job termination (mainly in women) and unemployment. As Virtanen and colleagues have found, sickness absence may be one of the factors associated with the potential non-renewal of a temporary job contract, and consequently could be an additional cause of presenteeism. This association is probably worse among private sector workers.

This situation is clearly unjust and can lead to a vicious circle. It has been clearly established that sickness absence is a valid predictor of health.[11] A temporary worker with high sickness absence has a high probability of being condemned to a poor career with periods of temporary work and periods of unemployment. This hypothesis is consistent with recent evidence which shows that some forms of temporary contracts are related to some social indicators such as remaining single and having fewer children when married or cohabiting.[12]

In conclusion, in an ever more globalised economy with an increasing number of temporary workers, governments must reinforce their social protection systems instead of reducing them, if one of the aims of our societies is to avoid increasing marginalised people. As Virtanen and colleagues have shown, flexible employment is an obvious public health problem, which should be included in our scientific and professional agendas.

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