Research Report TM/03/02
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This study aimed to provide an informed opinion on the risks to health of workers' long-term exposure to tunnel dust in the London Underground (LU). Dust mass concentrations and particle numbers were sampled at three stations underground and in train cabs on three lines, selected by trade union representatives. Size and composition of the dust was analysed; likely maximal exposures of staff and passengers were estimated; and toxicity was tested (in comparison with other dusts) using *in vitro* methods. Results were reported to representatives of LU management and unions before this report was finalised.

Results showed that tunnel dust differs markedly from outdoor particles; consequently, risks from outdoor particles are misleading for estimating its health effects. Tunnel dust is coarser, being generated by interaction of brakes, wheels and rails rather than by combustion, with higher mass concentrations (130 – 480µg/m^3 $PM_{2.5}$) and lower particle numbers (14,000-29,000 particles/cm^3). It comprises approximately 90% iron, 1-2% quartz and traces of other metals.

Toxicology showed the dust to have cytotoxic and inflammatory potential at high doses, consistent with its composition largely of iron oxide. The concentrations underground are well below allowable workplace concentrations for iron oxide (we estimated maximal exposures of about 200µg/m^3 over 8 hours; the occupational exposure standard for welding fume, as iron oxide, is 5mg/m^3 over an 8-hour shift) and so are unlikely to represent a significant cumulative risk to health of workers or commuters.

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ASSESSMENT OF HEALTH EFFECTS OF LONG-TERM OCCUPATIONAL EXPOSURE TO TUNNEL DUST IN THE LONDON UNDERGROUND

EXTENDED EXECUTIVE SUMMARY

Introduction

The Institute of Occupational Medicine in Edinburgh (IOM) was approached by London Underground Limited to give an informed and independent opinion about the likely harmfulness of exposure to the concentrations of tunnel dust underground. With colleagues from the Universities of Aberdeen and Edinburgh, we formed a small but highly experienced multi-disciplinary research team. Working in consultation with management and trade union representatives, we designed the present study. This is our final report. It describes what we set out to do, and why; what we found; and what we think it implies for understanding any risks to workers, and to passengers.

Background

Dusts fine enough to be inhaled vary in their potential to damage the health of people exposed to them. One determinant of risk is the actual amount of exposure to the dust. This in turn depends partly on the dust concentration (usually represented as the mass of dust for a given volume of air and expressed in units such as mg/m^3 or $\mu g/m^3$) and partly on how much time a person spends in those conditions.

In this report we consider so-called "respirable dust", that is particles fine enough to be inhaled as far as the furthest, gas exchanging part of the lung known as the acinus. Risk depends also on various characteristics of the respirable dust, and these in turn are related to how that dust was generated and became airborne. Thus, for the same amount of exposure, the kinds of adverse health effects and the risks of them occurring differ according to whether the dust is tobacco smoke (inhaled actively or passively), coal mine dust, fibres, house dust, outdoor air pollution and so on. These differences in the potential to cause damage to health (toxicity) are reflected in different standards for different workplace dusts, and also for outdoor air pollution.

The standard for outdoor particles also takes into account that exposure is more-or-less continuous, rather than during working hours, and that the population-at-risk includes a much greater proportion of those who are vulnerable, e.g. older people, the very young, people with pre-existing serious ill-health compared with a working population. Available data on the age distribution of those who use the London Underground show that the great majority – about 95% – are of adult working age. For other reasons also we think that, in terms of vulnerability to inhaled dusts, regular users of the London Underground are more comparable to a workforce than to the general population.

The main sources of respirable tunnel dust in the London Underground rail system are particles from abrasive forces acting on rails and wheels from traction and braking – these are likely to contain a lot of iron – and particles shed from human beings and their clothes. Both of these kinds of dust would be expected to comprise particles larger than those of which combustion-generated dusts, such as ambient air pollution, are composed. (The main source of particles in ambient air is combustion in vehicles' engines.) In addition, the iron-containing particles will be denser than those in ambient air. Tunnels would be expected to

contain some of the very fine dust generated by traffic outdoors, and drawn in from above ground.

There is a wide community with an interest in understanding whether there are risks to health, of workers and of commuters, from exposure to tunnel dust under ground. Some things are known. For example, tunnel dust contains a proportion of quartz, and previous research has shown that any risk of silicosis, a specific quartz-related lung disease, is likely to be very small indeed. Other things are speculative. For example, in recent years some scientists, and the press, have promoted – or at least not discouraged – the view that tunnel dust is equivalently toxic to outdoor air pollution. This comparison is very highly questionable, given the differences between the two kinds of dust in terms of sources and associated characteristics such as size and composition.

There is a need to assess what is known and what can reasonably be inferred about the risks to health of exposure to tunnel dust and, as appropriate, to make new measurements to assist in that assessment.

Aim and strategy

The overall aim of the present study was to provide an informed expert opinion, based in evidence, on the risks to workers of long-term exposure to tunnel dust. We also aimed to supplement this opinion with suitable commentary on any risks to the health of the travelling public.

After consultation within the industry (management and trade union representatives), we chose to do this by:

a. Taking new measurements to (i) characterise the 'typical' exposures of workers; (ii) characterise better the tunnel dust in terms of the physico-chemical characteristics - notably size distribution – that appear to affect toxicity; and (iii) investigate further how tunnel dust affects cells from the human lung; and then

b. Forming a view on how dangerous (or how safe) exposure to tunnel dust is, compared with other dusts which have been researched much more thoroughly; and so, taking account of actual exposures of workers underground, give guidance on the risks to their health of long-term exposure at work.

Inhalable particles and mechanisms of toxicity

Larger, heavier inhalable particles tend to deposit in the upper airways of the lung and get cleared quite rapidly. Particles that are small enough continue deeper into the acinar region of the lungs, where their deposition depends primarily on physical factors. These smaller particles are a concern for long-term health effects caused by poorly soluble particles, because:

a. the acinar region, though it is well protected by evolved mechanisms against inhaled organisms and toxic substances, may be injured and scarred by heavy and prolonged deposition of particles or gases, and

b. particle clearance from this region – whereby the smaller particles are engulfed by defensive cells (macrophages) that can also summon the assistance of other cells by generating an inflammatory reaction – is much slower than from the bronchial tubes.

The ability of particles to generate such an inflammatory reaction does not mean that lung damage will occur – rather it indicates that they are capable of provoking a defensive reaction by the lung. However, inflammation may lead to harm if it is severe or is at a lower level but prolonged (i.e. chronic), conditions that may be brought about by inhaling very toxic particles, or a very large amount of less toxic particles, especially the smallest ones. Indeed, very small ('ultrafine', <100nm) particles may evade the lung's initial defences and trigger inflammation in the internal (interstitial) tissue of the lung itself, where clearance is even slower. On the other hand, the lung also has effective mechanisms for dealing with inhaled particles and for repairing any damage that may occur, allowing us all to inhale large numbers of particles, some potentially very toxic, throughout a lifetime, usually without showing any easily attributable damage to health.

Particles affect cells directly through contact with them and so the larger the aggregated surface area of the deposited particles, the larger the contact area between particles and cells and consequently the greater the potential for effects on the cells (compared with similar particles of lesser surface area). Also some particles may release, from their surfaces, soluble components such as transition metals that might cause oxidative stress or damage to cells. Thus, though the mass of particulate matter per unit volume of air (expressed as mg/m^3 or $\mu g/m^3$) is the most common exposure metric used for setting dust standards (in workplaces and outdoors), the surface area, or particle number, may be more relevant for assessing harmfulness.

On the basis of this understanding, we have adopted several complementary approaches in order to 'benchmark' the toxicity of tunnel dust relative to other, well-researched ('exemplar') dusts.

a. We have studied the concentrations underground in terms of particle number and size as well as mass;

b. We have examined again the composition of tunnel dust and paid particular attention to how the body deals with exposure to iron – see the next section;

c. We have used two kinds of *in vitro* biological experiments to investigate the potential of inhaled tunnel dust to cause inflammation:
   - We have measured, in 'cell-free' tests, the pure chemical ability of various particles, including tunnel dust, to generate free radicals that may harm or cause stress to cells; and
   - We have measured the effects of tunnel dust and other particles on human lung epithelial cells (cellular tests).

   Note however that these are very limited biological tests because they can represent only very approximately how the lung of a living person responds to inhaled particles, requiring as they do doses several thousand times those to which the lungs of workers may be exposed .

Iron and human health

Iron, the main component of tunnel dust, is an abundant element. It is essential to life and health and is found in the body mainly in blood. Iron is absorbed into the body through the gut. In nature it is combined usually with oxygen as ferric oxide (commonly observed as rust) or as a carbonate or sulphide salt; the oxide is largely metabolically inaccessible. Humans obtain iron from their diet; the daily requirement in adults is about 1-2mg.

Humans may also be exposed to iron-containing dust at work, in occupations such as welding and cutting of metal, iron ore mining, fettling and metal-working. The iron in these circumstances is again in the oxidised, ferric form and is relatively insoluble. The lung also has a sophisticated mechanism for preventing absorption of toxic iron and for ensuring that the normal macrophage defences can remove it.

The consequences of inhaling iron differ in different trades, according to its physical form and what other kinds of dust are inhaled along with it. The best-known medical condition associated with iron is haemochromatosis, a rare and inherited condition – there is no evidence that it occurs as a result of absorption of iron through the lungs. For example, no excess risk of haemochromatosis has ever been reported in welders, the occupation with highest exposures to iron-containing dust. The only condition specific to iron inhalation is called siderosis, i.e. an accumulation of iron in macrophages in the lung. It is regarded medically as a "benign pneumoconiosis", that is it causes x-ray shadows without harming the person. The x-ray shadows regress after exposure to iron ceases. Welders have an approximately doubled risk of pneumonia compared to the general population. It is unclear what this is due to, but as iron is known to be a growth factor for bacteria it seems possible that inhalation of the metal as a fume predisposes to pneumonia.

Investigations of the physico-chemical characteristics of tunnel dust

Objective and methods

The main objective of this part of the work was to characterise the physical properties and composition of the airborne dust to which workers were exposed, and to make measurements that would allow estimation of the personal exposure levels of London Underground staff. After discussions, and following a pilot study, three sites were selected to cover a range of depths and circumstances: Holland Park station (Central Line); Hampstead station (Northern Line) and Oxford Circus station (Victoria Line). Measurements were made during January 2003.

Measurements were made on platforms of particle mass and number, using DustTrak and P-Trak instruments. Three other sampling devices were used to collect airborne dust samples for subsequent laboratory analysis. These were a $PM^{1}_{2.5}$ sampler, a PM_{10} sampler and a respirable dust sampler (which approximates to a $PM_{3.5}$ sampler). The sampling pumps and other bulky equipment were located at the end of the platforms. Measurements typically began around 7am and continued until 5pm. Both sampling and laboratory analysis were carried out to a high standard and with suitable quality controls.

In addition, personal sampling in the drivers' cabs was carried out for three days on each line, to assess drivers' exposures. This was done using a second set of DustTrak and P-Trak samplers. Both samplers were positioned inside the cab as close as possible to the driver's breathing zone and the driver was accompanied throughout this time by one of the researchers. The sampling time started with the booking on time for the driver and lasted until the end of the shift. These measurements were continuous except for lunch breaks, which typically lasted between 30 and 60 minutes. The results were adjusted to represent the whole shift, except when stated otherwise. The times when the driver was underground, and in the cab on the surface, were recorded.

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[1] PM = particulate matter, measured by weight, for a given volume of air, e.g. $\mu g/m^3$. The subscript of PM, in this case 2.5 or 10, indicates in micrometers (millionths of a meter) the approximate upper aerodynamic diameter of the particles.

The main results were:

1. *Dust concentrations in stations:* The dust collected on platforms was qualitatively similar at the three stations, with about 80% of the particles having a measured diameter less than 1μm. For iron-containing particles this underestimates aerodynamic diameter. Average $PM_{2.5}$ concentrations ranged from 270-480μg/m^3; average particle numbers ranged from 14,000 to 29,000 particles/cm^3.

2. *Comparisons between concentrations underground and on the surface:* There were very marked differences in the dust measurements underground and on the surface. Above ground, there were high number counts and low gravimetric (mass) concentrations. Underground, the opposite pattern was found.

3. *Concentrations in the cab for the three lines*: Average levels of $PM_{2.5}$ ranged from 130 to 200 μg/m^3; average particle numbers about 20,000 particles/cm^3. These data cover the entire shift, including measurements when the train was underground and on the surface. The number counts reflect principally the exposure when the train is above ground while the mass reflects exposure when the train is underground. Together with other measurements we have been able to conclude that very fine particles from road traffic on the surface contributed most to these particle number counts while (abrasion) dust from the tunnels contributed most to these $PM_{2.5}$ mass concentrations.

4. *Composition of tunnel dust*: Samples from station platforms showed that almost all (typically, about 90%) of the dust in the $PM_{2.5}$ samples was analysed as iron. There were trace amounts of chromium (0.1 – 0.2%), manganese (0.6 – 1%) and copper (0.1 – 1.5%). No zinc was detected on any of the samples (<0.1%). Quartz, analysed in respirable dust, accounted for only 1-2%; these measurements were approximate, being close to the analytical detection limit.

5. *Estimate of personal exposures of LUL workers and commuters:* This involves linking concentration data with the duration of time spent exposed. We focused on the mass concentration of PM rather than on particle number because the number counts were dominated by particles from above ground. Using, on a precautionary basis, the higher values of estimates from the present study, we have estimated that the likely maximum exposures of station staff and drivers are similar over a shift, at approximately 200 μg/m^3, based on an 8-hour average period. Averaged over 24 hours this would correspond to 67 μg/m^3. The duration of exposure of commuters would be less than that of the staff. For someone who spent approximately 2 hours in trains or on station platforms per day, assuming that the average exposure level was similar to the drivers, say at most 200 μg/m^3, then their 24-hour average concentration would be increased by 17 μg/m^3.

Investigations of the biological reactivity of tunnel dust

Objective and methods

The toxicology strategy was aimed at detecting the ability of particles to cause inflammation since inflammation generally underlies the adverse effects of particles. It should be noted however that it is also an essential defensive reaction of the body, without which survival would be impossible. The non-cellular tests (plasmid assay, ESR) and the cell tests (IL-8 protein, LDH) both measure aspects that are precursors of inflammation.

As noted earlier, toxicology tests in cells or in cell-free systems represent an attempt to detect toxicity using simpler and more ethical alternatives to using humans or animal exposures.

These systems cannot hope to replicate the complexity of a human tissue or organ. One result of this is that, in general, far higher doses are required to get a pro-inflammatory effect in a cell test than is required in animals to get an inflammatory response – the dose used in the cell test systems was many thousand of times greater than that calculated for someone in the London Underground. Thus, the tests are best at providing data on whether dusts *can*, at high enough doses, induce some response in cells. Also, they provide some useful data on the comparative toxicity of particles. They are however uninformative on the doses that cause effects in humans.

The tunnel dust studied had been sampled from station platforms – see above. We used PM_{10} sampled from Griffith House. This was sufficient only for the IL-8 test; for other tests we used PM_{10} from Manchester, collected for another study. Samples of titanium dioxide (TiO_2), a well-studied non-toxic dust, and of welding fume were used as control or comparison dusts in a number of toxicity assays. Particle size measurements showed that the tunnel dust samples from the three station platforms had similar size distributions, the TiO_2 comprised slightly finer particles and the welding fume was much finer than any of the other dusts.

The non-cellular and cell assays were carried out using standard methods.

The main results were:

a. *There was evidence of some toxicity from cell studies:* Tunnel dust showed evidence of some toxicity in laboratory cell studies, with very large doses being able to provoke cells to release substances that could cause inflammation in the lung.

b. *There were supporting indications, from the non-cellular studies, that the dust could be toxic:* There was coherence between the results obtained with the different assays, with the free radical chemical assays (non-cellular tests) supporting the findings in the cell assays.

c. *Comparisons with other workplace dusts:* This toxicity is much less than that of quartz but greater than that of the non-toxic dust titanium dioxide when given at comparable doses. It is roughly comparable to that of welding fume.

d. *Comparisons with ambient PM:* Tunnel dust was slightly greater in activity than the PM_{10} samples that were available. However, PM_{10} in different locations and on different days varies markedly in its toxicity; the comparison should be treated cautiously.

e. *Chelation of tunnel dust* (i.e. removing metal ions from 'coating' the surface of the dust), a process that occurs naturally in the lungs of living people, reduced its toxicity to about one-half that of unchelated dust.

f. *Welding fume as a benchmark dust:* The tunnel dusts were similar to welding fume in terms of their high iron content and the role played by the transition metals in the stimulation of IL-8 release. Overall, tunnel dust was closely similar to welding fume in term of toxic potency.

Discussion and conclusions

How can we best 'benchmark' the risks from tunnel dust?

We have considered two principal ways of doing this; by using

i. the risks to the general public of ambient PM_{10} or

ii. the risks to industrial workers exposed to iron-rich dust (iron oxide) of roughly the same size range. Relatively few groups, but large numbers, of workers are exposed to iron oxide. These may be separated into
    - those in whom the iron inhaled is in relatively large particles such as iron mining and fettling and
    - those in whom it is a fume (by definition consisting largely of ultrafine particles), such as welding and burning.

In order that we can 'compare like with like' as far as possible, we have taken account of the following considerations.

a. *The exposure metric (mass concentration or particle number) to be used when benchmarking:* We have drawn attention to the evidence and view that, in principle, particle number or surface area may be a better index than mass concentration for assessing the harmfulness of inhalable dusts. This would however be a difficult way to proceed in practice, because there is as yet limited information from epidemiological studies about health effects and risks in terms of particle number, and practically none in terms of surface area. Also, dust standards (workplace and outdoors) are in mass terms, as is the public debate about the risks of tunnel dust. So, our evaluation is in these terms also. This has bearing on our choice of benchmark dust.

b. *Results of the 'in vitro' biological tests:* The limited tests carried out showed that tunnel dust has some ability to cause inflammation, more so than an inert dust like titanium dioxide, much less so than a highly toxic dust like quartz. The tests showed that the potential of tunnel dust to cause some inflammation is broadly similar to that both of welding fume and ambient PM. However, we have noted limitations to the tests, in terms of quantitative comparisons with other low toxicity dusts; and some difficulties in particular in comparisons with ambient PM, because the latter varies in toxicity. Purely in terms of these tests, there is little to choose between ambient PM and welding fume as benchmark dusts. We therefore consider next the physico-chemical characteristics of the various types of particles.

c. *Nature and size distribution of the dust:* Clearly, these are key aspects of any comparison. Our results have confirmed that the dust underground is principally from abrasion, and comprises almost entirely iron oxide, in particles that are larger than those of ambient pollution or of welding fume. This abrasion dust is mixed through with some penetration of outdoor (ambient) PM, a very different kind of dust – ambient PM is a complex mixture of predominantly very small particles, of carbon, ammonium salts and trace metals, principally from combustion and photochemical atmospheric reactions. Within the overall dust mixture underground, the above ground dust makes a major contribution to particle numbers but only a very small contribution to particle mass concentration.

   In considering then which of the two comparisons is the more appropriate, we note that iron oxide rather than above ground PM is much the more comparable dust, for any comparisons based on mass concentrations – the dusts are similar in terms of:

   - size (and so, in terms of where inflammation is likely to occur) *and*
   - metal (iron) content *and*
   - (we think) in the bioavailability of that iron to cause damage in the lung, implying that the lung will respond in similar ways to the two kinds of dust.

Despite the differences in the nature of the dusts, there might be a case for using ambient PM as a benchmark dust, if particle number rather than mass could be used as the principal means of assessing exposures and risks. In terms of particle number, we note that the dust concentrations underground are much lower than above ground.

d. *Susceptibility of the population exposed:* For a reliable benchmarking of risks, we need not only comparability of the dusts, but also a comparability of the populations exposed and so at risk. We have noted that both the workforce and the travelling public differ markedly from the general population in terms of their likely susceptibility to inhaled dusts. A workforce comparison is more appropriate for our primary purpose, the assessment of risks to drivers and station staff in London Underground. The selected nature of the travelling public underground, compared with the general public, implies that a workforce comparison may also be better in estimating risks to commuters.

We therefore think that the best way to estimate the importance in health terms of exposures to tunnel dust, certainly to workers and probably also to the travelling public, is by reference to any known effects in workers exposed to iron oxide dust of roughly the same size range as tunnel dust. In doing so we note that:

- Most iron-exposed industrial workers are exposed to a more complex mixture of particles than are Underground workers.
- Tunnel dust is more comparable in particle size to that in mining and fettling rather than to the fumes from welding – the smaller size of welding fume would tend to over-state the risks of tunnel dust. However, mining and similarly abrasion-generated iron-containing dusts contain significant amounts of other more toxic substances such as quartz, making them unsatisfactory comparators.

We remain of the opinion that the risks to health of the general population from exposure to ambient PM, and the associated outdoor dust standards, are not a good guide to the risks of tunnel dust or to the standards that need to be maintained underground; and may indeed be seriously misleading in that regard.

What effects might be expected in train drivers and station workers?

In terms of *comparison of exposures*, our results suggest that London Underground workers may be exposed to dust concentrations up to about 200μg/m^3 over a shift, the dust being about 90% iron. In most cases exposures will be less than this. This concentration is less than one twentieth of the allowable limit of 5000 μg/m^3 suggested by the Health and Safety Executive for iron fume, a form of industrial pollution that we believe is likely to be more dangerous than the larger particulate form we have found in the Underground. Even if the HSE Occupational Exposure Standard were too high by a factor of 5 (and in some countries the Standard for welders is 1000μg/m^3), these concentrations would still be well within industrial safety limits.

In terms of *comparison of risks*, in summary our views of the risks to workers are as follows.

a. The physical and chemical characteristics of London Underground dust lead us to the conclusion that some iron may accumulate in the lungs of workers, but in a concentration and form that would not be expected to lead to fibrosis.
b. Similarly, there is no reason to suppose that it could cause emphysema, cancer, asthma or bronchitis.
c. It would not be absorbed into the body in sufficient quantities to accumulate in tissues other than the lung, and would not therefore cause haemochromatosis.

d. It is possible that there is some increase in risk of pneumonia among workers exposed to tunnel dust, by analogy with the increased risk observed among welders. However, we think that the risks are very low, because
    - the exposure of workers underground is clearly lower than that of welders;
    - the dust to which they are exposed is in a larger particulate form; and
    - the limited direct evidence available does not give any evidence of a problem.

Is the general public at risk from dust exposure by travelling on the London Underground?
We do not think that the travelling public is at any serious or substantial risk from tunnel dust inhaled while travelling underground. We have four main reasons for this view.

a. *Daily exposures are not high*: Although, in mass terms, dust concentrations underground are markedly higher than above ground, the relatively short duration of time exposed implies that in general the exposures associated with commuting underground are not high – in mass terms, they are in the same order as a 'typical' day exposed above ground in London. (There will, of course, be variability around this average.)
b. *Tunnel dust particles are relatively large when compared to urban air pollution*: While the effects of larger particles within the PM_{10} size range cannot be ignored, there is a growing body of evidence that a major reason for the observed epidemiological effects of ambient pollution on heart and lung disease is the ultrafine (<100nm) size range of the great majority of urban particles (EPAQS, 2000). A possible reason is that these very small particles deposit more efficiently in the acinus and penetrate into the lung interstitial space where inflammation is more likely to influence adversely the cardiovascular system. These small particles are in lower concentration in the Underground than at the surface in the same area of London.
c. *Iron dust is not especially harmful*: Tunnel dust consists principally of iron, and studies of workers exposed to iron – even as a fume – suggest at most a very limited risk to health.
d. *Population susceptibility:* The data provided by London Underground suggest that in fact the customer population is similar in age distribution to a working population. Moreover, it would be expected that the most vulnerable individuals to air pollution, those with heart and/or lung disease, would be under-represented among customers because of the obvious difficulties involved in such travel for the disabled. It is in our view reasonable to regard LU customers as generally comparable in susceptibility to a healthy workforce – certainly, much more comparable in susceptibility to a working population than to the general population.

Practical implications of the study

It is always wise and prudent to keep the levels of any workplace and ambient dust as low as practicable. There have been successes in London Underground in this regard – for example, the reductions in quartz content of the dust, and the Dust Action Group as a forum within the organisation – and we encourage management and unions in the Underground to continue to work together to find practicable ways of keeping dust levels low.

However we do not think that the risks, such as they are, warrant any special or extraordinary measures to limit exposures either of the workforce or the travelling public. Specifically with respect to the travelling public, including vulnerable groups, we have concluded that the decision to travel above or below ground need not be influenced by consideration of health risks from inhalation.

Reliability of our findings and the need for further research

World-wide, there is still a great deal of active and good research on the relationship between particles and health, e.g.

- what kinds of particles and how much cause adverse health effects;
- what are those effects and what are the risks;
- how do these effects occur;
- what is the role of co-exposure to other pollutants.

Our conclusions are based on argument by analogy, using our best current understanding of particles, their effects, and associated mechanisms of disease. We think that understanding is quite robust, and a good enough basis for policy at present. We recommend however that the conclusions we have reached, and our reasons for reaching them, be reviewed from time to time, in case the wider understanding on which they are based changes in any way that would modify those conclusions.

We have considered what further studies might give new information, specific to workers or the travelling public, that would help appreciably in reducing the remaining uncertainties around tunnel dust and its effects on health; and we have outlined some ideas. However, against the background of what is known already, and our view that the risks are small, we are not recommending as necessary any further studies at this time.

1. INTRODUCTION AND BACKGROUND

1.1 DUST AND ILL HEALTH

1.1.1 Different dusts have different risks to human health

The term "dust" as used colloquially has a very general meaning. It can be applied to airborne or deposited matter derived from fabrics, materials, domestic animals and our own skin in the house, to particles generated by activities such as mining and grinding in the workplace, and to smoke and fume generated by combustion in the urban environment. In other words, dust derived from domestic activity differs from that derived from vehicle exhausts, which in turn differs from those derived from factory chimneys or mining.

The risks to health of people exposed to these dusts differ accordingly. It is well established that particulate air pollution does indeed have very different effects on the human body depending on its sources and method of generation. Some well-known illustrations make this point.

- Cigarette smoke, inhaled either directly or passively, contains burnt organic matter and a multitude of other chemicals. The more of it people inhale, the greater their risks of lung cancer, emphysema, leukaemia and heart attack, to name but a few of the known consequences.
- Dust in the house contains bits of skin; fibres from fabrics; particles from cooking; mould spores and mite faeces. We all inhale it every day; a few develop allergies and asthma as a result.
- Coal mine dust contains carbon, quartz and various silicates, and miners exposed for years may develop serious lung diseases from its accumulation.
- Urban air pollution is derived in the United Kingdom nowadays largely from combustion in vehicle engines, and its particulate component has been associated with increased risks among vulnerable people, such as those with lung and heart disease, of deterioration in symptoms or even premature death.

Thus the study of dust and its effects on health must occur in the context of the site in which it is found and the sources from which it is derived. While this is intuitively obvious, it becomes less so in two circumstances: Firstly, when the word "pollution" is substituted for "dust", since pollution implies matter generated by human activity with the potential to cause harm, and secondly, when the dust is measured in the same units, for example PM_{10} or $PM_{2.5}$ regardless of its source or nature.

1.1.2 Tunnel dust in the London Underground railway system

The focus of the present study is a particular dust mixture, the respirable dust in the London Underground railway system ('tunnel dust'). A previous review commissioned by London Underground reported concentrations of tunnel dust, measured as the mass of particles less than 2.5μm in aerodynamic diameter ($PM_{2.5}$) considerably in excess of those found above ground (Hawkins, 2001**)**. It is important therefore to assess what are the risks to health of people exposed to such concentrations, notably workers but possibly also commuters. This issue has been considered, on two occasions, by the UK Government Advisory Committee on the Medical Effects of Air Pollutants (COMEAP). On both occasions COMEAP was generally reassuring that the risks were low, noting for example that Underground workers have not in the past shown evidence of excess morbidity and pointing to the different natures of the polluting dusts to which people are exposed above and below ground. However COMEAP also highlighted uncertainties which limited its ability to give a definitive assessment of the risks.

It is against this background that the Institute of Occupational Medicine, working together with colleagues from the Universities of Aberdeen and Edinburgh, was commissioned by London Underground Limited to examine the exposures of employees to dust, and the characteristics of that dust, in the London Underground.

1.2 WAYS OF INVESTIGATING DUST-RELATED RISKS TO HUMAN HEALTH

A great deal of knowledge has accumulated, over many decades of study, of the effects of different kinds of inhalable dusts on the human organism. This knowledge has come from a variety of types of scientific study, each of which gives different but complementary information. In general, it is possible to predict from this accumulated knowledge the likely consequences of exposure to dusts of known characteristics.

1.2.1 Epidemiological studies

Epidemiological studies investigate the associations between the exposures of populations of people and health outcomes. Some of the associated research programmes have been very extensive and detailed. For example, the health effects of exposure to coalmine dust have been studied in more than 50,000 coal miners in the UK, with detailed concurrent measurements and analyses of dust over more than 25 years (e.g. Hurley *et al.*, 1987). Or again, the strongest early evidence linking long-term exposure to ambient particles with reductions in life expectancy (Pope *et al.*, 1995) was based on an American Cancer Society study of more than 500,000 adults in about 150 US cities, followed up for several years. In each case, it was necessary also to take account of other possible causal factors such as smoking habit.

These major research programmes have shown clear relationships between workplace dusts, ambient pollution and various adverse health effects, especially on the cardio-respiratory systems. There are however still many uncertainties and controversies, and ongoing research has the aim of reducing or resolving them.

While such studies only show statistical associations, and quantify the likelihood that those associations might have occurred by chance, they are the most informative in deciding whether a given population is at measurable risk. They have been used extensively in estimating risks to workers in dusty industries and determining appropriate exposure standards to prevent disease occurring. These are also the types of study on which information on the health effects of urban pollution has been based.

1.2.2 Toxicological studies

Toxicological studies aim to investigate the mechanisms whereby harm may occur. They are usually carried out on animals such as rats, which may respond to toxic substances in a way similar to man, or on cells in tissue culture. An important aspect of all such studies is that to obtain effects within the constraints of the experimental situation it is necessary to use much higher doses than would normally be experienced by exposed humans.

A range of toxicological studies may be used, varying in complexity and in closeness to what may happen in humans. Whole animal studies (*in vivo* studies) generally predict reasonably accurately whether humans are at risk, although (because of the difficulties of animal-to-human scaling) they are less useful at quantifying that risk by predicting the toxic dose. The most realistic model of long-term human exposure to an inhaled dust would be a long-term study of inhalation of the relevant dust by experimental animals, for example laboratory rats. This is time-consuming, very expensive and involves killing large numbers of animals. For these reasons, more practicable alternatives are usually employed, and short-term studies where the dust is instilled directly into the animals' windpipes are one alternative.

However, the current move to reduce the use of experimental animals for humane reasons has led to simpler strategies where studies are performed on cultured cells that may be derived from the lungs of animals or humans (*in vitro* studies, a term that also includes non-cellular studies of particle chemistry). These have the advantage that they allow detailed examination of individual mechanisms of response to the dust but the disadvantage that they rarely allow examination of the sequence of events that occur in the living animal. Cell studies are limited to investigating one or a few toxic mechanisms, and will usually only indicate a potential to cause harm. Nevertheless, these studies can define the harmfulness of a substance in broad terms, and they may also give a reasonable indication of a lack of toxicity.

1.2.3 What factors influence the risks of disease?

Together, toxicological and exposure research, along with epidemiology and human experimental studies, have helped establish what aspects of exposure to particles influence the risks of disease. There are uncertainties but the main factors that need to be taken into account are the following:

a. *How much exposure* to dust do people at risk experience in the short term and/or in the long term, e.g. over a working life. In considering this, long-term exposure of individuals is usually separated into two components:
    - *Duration of exposure*, i.e. amount of time spent exposed to the dust of interest; and
    - *Intensity of exposure*, i.e. the concentration of dust to which a person is exposed for any given period of time.

b. *The toxicity of the dust per unit exposure*. On best current thinking, this toxicity is determined by various physico-chemical characteristics of the dust itself, e.g.
    - Its size distribution, and in some circumstances the shape of the particles;
    - The composition of the dust, including its solubility; and
    - Various surface properties of the dust.

    These issues, which influence the potential of the inhaled dust to lead to adverse reactions in the body, and the nature, degree and location of those reactions, are considered in more detail in Chapters 3 and 4, following.

c. *The susceptibility of the exposed population* – the risks of disease depend not only on how much and what kinds of dust people are exposed to, but also on the vulnerability of those who are exposed, for example, the extent of pre-existing heart or lung disease from other causes.

1.2.4 Implications

The extensive existing research base on the effects of dust on health – the detailed knowledge of the risks to health from exposure to particular dusts, and the aspects of exposure that influence those risks – opens up an interesting possibility: it is possible to estimate, at least approximately, the risks to health from a dust of interest, e.g. tunnel dust, provided that we can compare it sufficiently well with other, well-researched, dusts. Such a comparison would involve assessing (i) the characteristics of the dusts that are relevant to its toxicity; (ii) the likely magnitude of exposures of the exposed population; and (iii) the characteristics that influence or indicate the susceptibility of the exposed population. This approach – which is not new in concept – is developed further and applied in the present study.

1.3 SOURCES AND ASSOCIATED CHARACTERISTICS OF TUNNEL DUST

1.3.1 Sources of tunnel dust

The dust we have been asked to investigate in the London Underground system is a form of particulate pollution arising as a result of human activity, that activity occurring mainly underground. The main sources must therefore be there, although a contribution from sources above ground, entrained into the system as air is drawn in from the surface, would be anticipated.

The possible sources of polluting dusts underground are as follows:

- Abrasive forces acting on rails and wheels from traction and braking
- Combustion of carbon brushes from sparking
- Volatilisation of oil used for lubrication
- Particles shed by human beings and their clothes
- Biological particles from animals, fungi and bacteria
- Residual particles from previous tunnelling
- Particles generated by repair activity, cutting and burning.
- Particles generated above ground and entrained into the system

Of these, a number are shared with the above ground urban environment and are likely to be present underground in lower concentration – biological particles and urban traffic-related pollutants. Particles shed by humans are also present above ground but likely to be in higher concentration underground because of the greater concentration of people.

Aside from these, particles generated by repair activity are likely to make a relatively minor contribution, confined to the site of activity. Combustion of electrodes and volatilisation of oil are also likely to occur only to a very minor extent, and make a small contribution. The presence of residual particles from tunnelling is likely in recently driven tunnels only, since the corresponding dust from older tunnels will have largely been removed by the diluting effect of years of air changes. Any remaining dust will reflect the mineralogy of the strata through which the tunnel was driven.

1.3.2 Associated characteristics of tunnel dust

It may thus be speculated that the main sources of dust underground are the actions of wheels on rails and of brakes on wheels together with the presence of large numbers of human beings. Abrasion-generated and human activity-generated particles are mainly in the size range above about half to one micrometer, being represented particularly in pollution measured as PM_{10}, to a lesser extent in $PM_{2.5}$, and hardly at all when pollution is measured as particle numbers below 100nm. It follows from this that the dust underground, as measured by weight, is likely to be of relatively large size compared to that above ground. Furthermore, in contrasting these two major sources, dust generated by abrasion would be expected to contain a high proportion of iron while that generated by human movement would be predominantly derived from clothing. Thus, when the dust is measured by weight, this mass would be expected to represent largely the heavier, iron-containing particles.

Relatively large and heavy particles fall from the air more rapidly than lighter smaller particles. Having fallen, however, they may be re-suspended by the action of wind. Thus the regular passage of trains and large numbers of people through the underground system would be expected to keep particles in suspension during the hours of activity, with falls in the night-time. Similarly, the contribution of smaller, combustion-generated particles from vehicle activity above ground would be likely to increase somewhat as pollution builds up in the busy periods and to decline overnight.

1.3.3 Contrast with urban particulate air pollution

In contrast to the situation underground, in the streets of London the single most important man-made contribution to particulate pollution is from the internal combustion engines, now most notably those of diesel vehicles. These sources produce a very fine (often called "ultrafine") particulate dust, mostly smaller than 100nm in aerodynamic diameter. Other domestic and industrial combustion sources, and atmospheric photochemical activity, add to this. There is in addition a contribution from larger particles, generated by abrasion of roads and tyres and by wind activity on soil and water surfaces.

The consequence of this in terms of measurement of pollution is that equal weights of dust in the two circumstances, above and below ground, are likely to comprise dusts of very different size and composition. A milligram of city street dust would contain huge numbers of very small particles while a similar weight of dust underground would be likely to contain relatively smaller numbers of larger, iron-containing particles. Thus, in order to comment on possible relative toxicity, it is necessary to measure both particle mass and particle numbers and also to compare the content (composition) of the underground dust to that above ground.

1.3.4 Inhalability and deposition of dust

The same physical principles that determine the rate at which particles in the air fall to the ground determine where they can get to in the lung. It is usual for scientists in this field to refer to inhalable and respirable dust. The former includes all particles that when breathed in pass beyond the larynx (voice box). The latter are those that reach the delicate structures in the furthest part of the lung where exchange of gases occurs, the acinus. For the purposes of this report, all the particles considered are potentially respirable. They differ in their sizes, however, and this determines what proportion, having reached the acinus, is actually deposited there. Of the smallest particles, below about 100nm, about half are deposited. Above that size, up to about 7μm, about a third are deposited. It should be noted that here, size refers not to actual diameter as measured by a ruler but to a relative diameter in relation to that of a particle of the density of water that determines the rate at which the particle falls. This is termed "aerodynamic diameter". Thus a particle of iron measured by ruler may have a diameter of, say, 1μm but would have the falling speed of a much larger particle and would thus be less likely to reach the acinus as it would be more likely to fall on the airways on the way down the lung. This rather complex matter will be addressed later when we discuss the particle size distribution of underground dusts.

1.4 PREVIOUS STUDIES OF DUST IN UNDERGROUND RAILWAYS

1.4.1 Dust concentrations

Examination of the concentrations of dust in underground railways appears to have been confined to the cities of London and Stockholm; the results are consistent with the general considerations described above. In London, a series of occupational hygiene studies by London Underground dates back to 1979 (reported in Hawkins 2001), but peer-reviewed publications are few. Since the report published by the Health and Safety Executive (HSE, 1982) it has been apparent that relatively high dust concentrations occur underground, compared with typical urban concentrations of PM, and that iron comprises the main component, quartz concentrations being around 1 to 10%. Respirable dust concentrations (approximately PM_4) measured by London Underground have ranged between 100μg/m^3 and 3.6mg/m^3. Priest *et al* (1998) have reported concentrations of PM_9 ranging between 0.5 and 1.12mg/m^3, with a size range between about 0.5 and 9μm diameter. Pfeifer and colleagues (1999), investigating metal exposures among London commuters, noted higher concentrations among those using the Underground. Adams and colleagues (2001) have compared exposures to $PM_{2.5}$ of people in different London transport microenvironments, and found

underground exposures to have been some three to eight times higher than those above ground.

In the Stockholm underground railway respirable dust concentrations are also high compared with urban concentrations of PM. Johansson and Johansson (2002) have reported that concentrations of PM_{10} and $PM_{2.5}$ averaged respectively 470 and 260μg/m^3 between 7am and 7pm on weekdays. These levels fell at weekends and were closely related to the number of trains passing through the station being studied. Watering of the tunnel walls and track made little difference to average concentrations. These authors also summarised the results of some earlier studies in the Stockholm underground that had shown generally similar results. As in the Pfeifer study, the starting point for this survey had been the finding that office workers who commuted by underground showed higher blood concentrations of manganese than taxi drivers, strongly suggesting a metallic source of the dust inhaled underground.

Overall these studies are consistent in showing that absolute concentrations of dust underground are greater than those above ground and that the dust underground is likely to be metallic, and so consistent with dust generated mainly by friction between brakes, wheels and rails rather than by combustion.

1.4.2 Toxicity

There appears to have been only one report of an experimental study of the toxicity of underground dust, that by Cullen *et al* (1995). The focus of the study was the toxicity of quartz in tunnel dust and, in particular, whether that toxicity was modified (reduced or increased) by the presence of iron. (It should be noted that, since this study, there has been a successful attempt by LU to reduce the levels of quartz in the air of the LU – see Chapter 5). The study was carried out on a sample of respirable dust (less than about 4μm diameter) collected at a London Underground station. The dust contained 35% iron and 7.8% quartz. The researchers combined an investigation of the effects of the dust instilled into the lungs of rats with experiments to study dust toxicity to lung cells (macrophages) on the laboratory bench.

- The instillation (*in vivo*) studies, using of necessity high doses of dust, showed a mild inflammatory reaction to underground dust without evidence of silicotic change. This was markedly less than the response to a corresponding dose of pure quartz and also less than the response to a dose of quartz mixed with iron particles in the same proportion as in underground dust.

- The laboratory bench (*in vitro*) studies showed underground dust to be less toxic than quartz in similar doses, except at a very high dose.

The authors concluded that the dust examined was considerably less toxic than the same dose of pure quartz and that this was probably due to the amelioration of the effect of the contained quartz by the iron content of the particles. Limited human data support this view. Specifically, Carlton (1994) has reported a study of 100 track re-conditioners exposed to quartz-containing dust from drilling work, but did not show evidence of silicosis. Nevertheless Cullen *et al* stated that the dust was not completely harmless, as in very high doses it had the capacity to cause inflammation in the lungs.

1.4.3 Summary

In summary, there is consistent evidence that individuals working in the environment of the London Underground railway are likely to be exposed to higher absolute dust mass concentrations than those working above ground. Compared with exposure on the surface, underground exposures are likely to be to a dust of higher density and larger particle size, but

with a contribution from ultrafine particles entrained from above ground. The likely cumulative exposures of workers are not known. The dust appears to have some toxicity, less than that of quartz, but more than that of a completely inert dust. No large studies of effects of such exposures on humans have been reported, although London Underground has found no evidence of silicosis in selected relatively highly exposed workers.

1.5 SUSCEPTIBILITY OF THE EXPOSED POPULATION

Having considered the characteristics of the dust, we now turn to the characteristics of the exposed populations. In considering effects on health of exposure to tunnel dust, we need to consider two populations at risk: the workforce, and the travelling public. There are differences between these two groups in terms both of exposure and possibly of susceptibility to any adverse effects of exposure to tunnel dust. In addition, there are differences in likely susceptibility between both of these exposed groups and the general public resident in London; i.e. those exposed to outdoor air pollution in the city.

As a general rule, workforces exclude children, the elderly and many of those with chronic disability, although they are likely to include some with latent disease, for example asymptomatic coronary artery disease. Moreover, workers who develop serious illness tend to retire from the workforce. This means that a workforce tends to be fitter on average than the general population. It may also mean that workers are on average less likely to be adversely affected by environmental factors than the general population. In addition, for toxic substances present in the ambient air, the duration of exposure of the general population is theoretically from birth to death, whereas workers' time at work, and so their exposure to industrial pollutants, is for a shorter duration, with fewer years of exposure, and fewer hours per week exposed even in those working years. For these and other reasons, it is usual when setting standards for exposure to toxic substances to have a lower level for the general population than for groups of industrial workers – see also Section 1.6.2, later.

Table 1.1. Age distribution of London Underground customers (figures provided by London Underground from their rolling survey programme (RODS) designed to capture information about journeys on the network)

	Number of customers	% of total
Under 16	7970	0.24
16-19	111369	3.40
20-24	562527	17.17
25-34	985689	30.09
35-44	734155	22.41
45-59	765062	23.35
60-64	42565	1.30
65-70	34726	1.06
Over 70	31857	0.97

In terms of exposure and likely susceptibility the travelling public differs from the London Underground workforce but it also differs from the general public.

- Regarding exposure, clearly the duration of exposure of the travelling public is in general much less than that of station employees and train drivers. This is considered in more detail in Section 5.5, later.
- However regarding susceptibility, as indicated by its age distribution, the travelling population is not representative of the general population and is much closer to that of a workforce (Table 1.1). It seems probable that children and the elderly are markedly under-represented among commuters. The vast majority of passengers are aged 20-59; less than 2.5% are either under 16 or over 65 years of age.

Moreover, among commuters, individuals with serious heart and lung illness are effectively excluded from the Underground because of the problems such people have with mobility. It is thus likely that the selection pressures operating on a workforce and making it healthier than the general population are little different from those that determine who travels in the Underground. This is not to say that children, the elderly and the disabled do not travel on the Underground; they do but are under-represented compared to the general population.

1.6 POSSIBLE EFFECTS ON HEALTH OF WORKERS AND COMMUTERS

1.6.1 Silica and silicosis

At the time of the Cullen *et al* (1995) report, the concern was that exposure of workers to a dust known to contain quartz could lead, over years of exposure, to the development of silicosis. This is a condition of nodular lung fibrosis that may lead to impairment of function and breathlessness, and in rare cases may prove fatal. The effects of silicosis have been extensively reviewed (see e.g. Seaton 2003).

Exposures above about 1mg/m^3 may be associated with the more severe and disabling forms of the disease (Seaton and Cherrie, 1995). Under the Health and Safety at Work Act's Regulations for the control of substances hazardous to health (COSHH Regulations) there is a Maximum Exposure Limit (MEL) for respirable crystalline silica (RCS) of 0.3 mg.m^{-3} (8-hour time weighted average [TWA]) (HSE, 2002). Long-term exposure to concentrations around and above the occupational exposure standard may lead to dust accumulation in the lungs and pulmonary lymph nodes with associated scar formation. A detailed examination of exposure-response relationships has recently been published (Buchanan *et al* 2003) indicating that the risks of exposure to some kinds of quartz are higher than had previously been thought. Partly as a result, the HSE has recently issued further guidance. In a Chemical Hazard Alert Notice, pointing out that the legal requirement for substances with MELs is to control exposure as far below the MEL as is reasonably practicable, the HSE has advised that "employers should aim to control exposures to 0.1 mg.m^{-3} (8-hour TWA) or below" (HSE, 2003).

Quartz varies in its toxicity; potency to cause damage is greatest with pure quartz mineral, when quartz is combined with very little other matter or when it has been freshly fractured and has active surface properties. Many workers, however, are exposed to quartz mixed with other minerals, usually silicates or carbon. In general such mixtures are less toxic than would be predicted from the concentration of quartz in them. It is believed that this is because the other minerals occlude the surface of the quartz crystal, preventing it from damaging the lung's defensive cells, the macrophages. For example, coal miners do not appear to develop signs of silicosis unless the quartz content of the coal dust they have inhaled is greater than 10%. The study by Buchanan *et al* referred to above was of coal miners exposed to unusually high concentrations of freshly-fractured quartz; the risks there were untypical of coal mining generally.

We have noted that the previous toxicity studies of tunnel dust (Cullen *et al*, 1995) were intended to investigate whether the presence of iron in the dust inhaled by workers in the London Underground reduced the toxicity of the quartz in the dust, perhaps by affecting its surface activity. This proved to be the case, suggesting that silicosis would be an unlikely outcome in the workers, an assessment supported by the earlier and relatively small-scale study of exposed workers by Carlton (1994).

However, at that time it had not been established what doses of dust would have been inhaled by workers underground, and dose is clearly as influential as toxicity in determining the risks from exposure to any dust. Also, concentrations of quartz and associated exposures of

individuals may have changed over time, and so consideration of the risks from exposure to quartz has remained a relevant issue.

1.6.2 Comparisons with the cardio-respiratory effects of outdoor particles

More recently, Priest and colleagues (1998) have suggested that for regulatory purposes, and in terms of hazard to health, underground dust should be regarded as similar to ambient urban PM_{10}. Noting that dust levels underground were "considerably higher than ambient PM_{10} levels and the Government's air quality standard of 50μg/m^3" they inferred that passengers would obtain a high proportion of their daily exposure to PM_{10} while travelling in the Underground. They concluded that risks of such outcomes as heart attack and hospitalisation for lung disease might be correspondingly increased to an extent that requires preventive action.

This argument ascribes to tunnel dust the adverse health effects that are well-established as associated with, and most probably caused by, exposure of the general population to ambient particles. In effect, it treats each μg/m^3 of tunnel dust as having the same toxicity as a μg/m^3 of ambient PM_{10}. Indeed, Priest *et al* (1998) suggested that the iron in the tunnel dust might add to its toxicity compared with ambient PM.

The reasons for the epidemiological associations between exposure to ambient, vehicle-derived particles and effects on the heart and lung are not fully understood, and are likely to be complex. However, the weight of evidence suggests that the toxic effect is likely to be determined to a large extent by the numbers of particles in the aerosol and by their surface area, rather than their mass. If this is true, it means that there is not a sound basis for treating as comparable the toxicity per μg/m^3 PM_{10} of the larger and heavier abrasion-generated tunnel dust and the relatively fine combustion-generated ambient particles. Such a comparison ignores the obvious differences in sources and composition between tunnel dust and ambient PM – differences that are almost certainly reflected in differences in particle size distributions and other characteristics that are relevant to the toxicity of dusts. It should be noted, for example, that a housewife working at home and in the kitchen may be exposed to mg/m^3 concentrations of PM_{10} but nobody would consider that as comparable to outdoor, traffic-generated PM_{10}.

For these reasons, the conclusions of Priest and colleagues were criticised by the Department of Health's Expert Committee on the Medical Effects of Air Pollutants (COMEAP 2002). Nevertheless they have attracted the attention of the media and have caused some alarm among workers and passengers.

The characteristics of dusts that may influence their toxicity are reviewed in some detail later (Chapter 3), with special reference to the role of iron (Chapter 4). However, we note now that support for the view that workplace dusts should not be viewed as similarly hazardous (per μg/m^3) as ambient PM, comes from many workplace studies. For example coal miners, who had been exposed to respirable coal dust concentrations in the order of 2000 to 10,000μg/m^3 on most days of their working lives, did not have an increased risk of death from lung cancer (Miller *et al.*, 1997) or from internal non-accidental causes generally (Miller and Jacobsen, 1985).

Many other groups of workers have regular exposures to mixed dust concentrations in the range 500 to 1000μg/m^3, and the Health and Safety Executive sets Occupational Exposure Standards that take account of the relatively less toxic propensity of abrasion-generated dusts. Some examples of workplace respirable dust standards are shown in the table.

Dust	8-hour exposure standard ($\mu g/m^3$)*	Main toxic effect
Chromium compounds	500	lung cancer
Quartz	300	silicosis
Grains	10,000	asthma
Hardwood	5,000	asthma
Flour	10,000	asthma
Coal	2,000	pneumoconiosis
Graphite	4,000	pneumoconiosis
Manganese	5,000	pneumonia, parkinsonism
Barium sulphate	4,000	benign pneumoconiosis
PVC	4,000	benign pneumoconiosis
Welding fume (iron oxide)	5,000	siderosis
Wool	10,000	bronchitis

* These are normally expressed as mg/m^3, but here we express them as $\mu g/m^3$ in order to facilitate comparison with the ambient air quality standard of $50\mu g/m^3$.

All these substances have known harmful effects on exposed workers; the different standards reflect differing toxicities. None approaches the ambient standard of $50\mu g/m^3$. However, account should be taken of the fact that workers, at maximum, may be exposed to these concentrations for 8 hours daily over a working lifetime whereas the general population is exposed to ambient pollution potentially for a whole lifetime. This fact was taken account of when the government's Expert Panel on Air Quality Standards recommended the $50\mu g/m^3$ standard for PM_{10} (EPAQS, 2001)

1.7 THE NEED FOR FURTHER INVESTIGATION AND ASSESSMENT

This overview has shown that there are several reasons why it is useful to re-assess the possible harmfulness of tunnel dust. All these reasons are concerned, one way or another, with making as informed as practicable an assessment of the risks to workers, and to the travelling public, of exposure to tunnel dust underground, given that the usual basis for comparison – the risks to the general public of ambient PM – is highly questionable. In addition, it is still relevant to see if there are specific risks to workers from long-term exposure to the silica fraction of the dust.

2. AIM AND SUMMARY PLAN OF WORK

2.1 AIM

The overall aim of the present study is to provide an informed expert opinion, based in evidence, on the risks to workers of long-term exposure to tunnel dust. The study's focus on the workforce follows from their higher exposures and was agreed with London Underground's Dust Action Group, comprising representatives of management and of unions. However we also aim to supplement this opinion on the risks to workers with suitable commentary on any risks to the health of the travelling public.

2.2 STRATEGY FOR MEETING THESE AIMS

2.2.1 Possible strategies

We considered two strategies:

1. ***Estimate directly the risks to health of long-term exposure to tunnel dust, by carrying out one or more large-scale new studies***. These might examine the health of LUL workers in relation to estimates of the exposures they had experienced, and taking account also of other factors such as smoking habit that may influence the risks of lung disease and that, if they were ignored, might distort any evidence of a relationship between exposure to tunnel dust and lung disease (health). As well as medical surveys of a large group of workers, such a study would also require recording of work histories, establishing the conditions of individuals' exposures throughout their working life at LUL, and in other dusty industries, as well as (where practicable) gathering information about confounding factors such as smoking habit.

2. ***Estimate indirectly the risks to health***, by

    i. Characterising the 'typical' exposures of workers, based on current working practices.
    ii. Characterising better the tunnel dust in LU, in terms of:
        - the physico-chemical characteristics (notably size distribution and composition) that current research suggests affect toxicity and,
        - by some direct investigation of biological responsiveness – the quickest way (though not the most reliable) is to examine how cells from the human lung respond to the dust;
    iii. Based on what is known about tunnel dust, including in particular these new investigations, 'benchmarking' how dangerous (or how safe) exposure to tunnel dust is, compared with other dusts which have been researched much more thoroughly;
    iv. Using research on these other dusts to give guidance on the risks to health of tunnel dust, based on comparisons of:
        - the toxicity of tunnel dust and the comparisons dusts, including dust characteristics which affect that toxicity;
        - the likely exposures of workers in LUL and
        - the likely susceptibility of the workforce and of the travelling public.

2.2.2 Strategy recommended and agreed

The authors of this report strongly recommended the second option (strategy 2), of estimating the risks indirectly, and this is what was adopted. There are several reasons.

- It is the only strategy which would give well-informed risk estimates reasonably soon – direct research would take several years to complete.
- Also, although there are uncertainties associated with both approaches, we thought that the indirect approach was more likely to give better estimates of exposure-related risks, provided that we could benchmark tunnel dust against at least one other well-researched dust. This is because the scale of any studies that could be carried out directly is limited by the size of the exposed workforce.

The authors therefore decided, after full discussions of these options with management and trade union representatives, to estimate risks indirectly, using a plan of work along the lines described above. This report describes what we did and gives our findings and conclusions.

3. PARTICLE CHARACTERISTICS AND MECHANISMS OF TOXICITY: A BRIEF OVERVIEW

This brief overview is supplemented by a more detailed exposition in the paper by Donaldson and Tran (2002). Later, in Section 6.1, the general ideas summarised here are discussed in explicit relation to the toxicity tests used in the present study.

3.1 THE LUNG, DEPOSITION AND FATE OF INHALED PARTICLES

The lung is a system of ever-narrowing tubes that terminate in the alveolar region, where gas exchange occurs. Conventionally the lung may be regarded as being divided into three compartments: the upper airways (nose, throat and larynx), the tracheo-bronchial region (windpipe and bronchial tubes), and the acinar region which is the terminal airways and their associated alveoli (the tiny tubes and the fine air spaces into which they lead).

Inhaled particles, depending on their size and density, may deposit in the upper airways, the tracheo-bronchial region or the central parts of the acini (centri-acinar region of the lung) – the terminal bronchioles, alveolar ducts and proximal alveoli (Donaldson *et al*, 2002). If they deposit in the upper airways or the airways of the bronchial tubes, then clearance by the 'mucociliary escalator' is relatively rapid, with most of the particles being swept upwards and out of the lungs to be swallowed, within 24 hours.

Particles that are aerodynamically small enough continue deeper into the lungs before depositing and a proportion of them settle in the acinus. Here clearance – mediated by macrophage cells which ingest the particles and move up to be swept out of the lungs to the gut by the mucociliary escalator – is much slower. In the order of 65 days is needed before half of the particles are removed in rats, and even longer in humans with unimpaired clearance (Tran *et al*, 1999) – longer still, of course, in humans whose clearance has become impaired by lung disease. There is therefore special interest in the behaviour of particles and cells beyond the airways towards the centri-acinar region of the lung, since particles deposited there remain longer and are in contact with an especially delicate region of the lung that is prone to injury. Particles that deposit in this region are the major concern for long-term health effects caused by poorly soluble particles (Brody *et al*, 1984).

Having said this, the primary function of the lung is to allow the transfer of oxygen from air into the blood stream and of carbon dioxide in the opposite direction and this necessitates that the blood and the air are brought into close proximity, a potentially dangerous situation. The mammalian lung has therefore evolved robust systems to defend the acinar structures, which are constantly threatened from the time of birth by deposition of inhaled particles, particularly dust, bacteria and viruses. As indicated above, larger particles are removed from the airways by their capture in airway mucus. Smaller particles are engulfed by defensive cells, macrophages, that can both remove the particles and also summon the assistance of other cells by generating an inflammatory reaction. It should be noted that the ability of particles to generate such a reaction does not necessarily mean that lung damage will occur – rather it indicates that they are capable of provoking a defensive reaction. However, protracted (chronic) low level inflammation or large scale (acute) inflammation can result in permanent change in lung structure such as fibrosis.

These defences may be overcome in a number of ways. First, particles such as quartz, asbestos and some microbes, may simply prove too toxic for them to cope with. Secondly, the sheer number of particles may be too large for the macrophages to clear. Thirdly, the size of the particles may be so small that they evade phagocytosis and interact with the epithelial cells in prolonged fashion even passing through the epithelial cell layer to set up

inflammation, not in the airspaces, but in the internal (interstitial) tissue of the lung itself. This process of "interstitialisation" is one factor that has been evoked to explain the harmful effects of the ultrafine particles characteristic of urban air (Seaton *et al*, 1995; MacNee and Donaldson, 1999). Removal of particles from this interstitial space is slower and depends on transport through the lung's internal system of lymph vessels to glands (lymph nodes) where the immune defences are concentrated. Finally, even if inflammation leads to lung damage, there are systems for repair by regeneration of the lining cells of the air spaces. Thus, most of us are able to cope with inhalation of large numbers of particles, some potentially very toxic, for all of our lives without showing any measurable damage to our lungs. Indeed, the successful survival of the human species and of other higher animals has depended on the ability of the lung to resist attack by particles.

3.2 THE ROLE OF INFLAMMATION

Inflammation is a programmed sequence of events that has evolved to defend against infection and damage and involves the accumulation of white blood cells at the site to fight microbes and 'heal' damaged tissue. Although it is an important defence mechanism in the body, inflammation may lead to harm if it is very severe or is at a lower level but prolonged over years, i.e. chronic.

Inflammation is the type of response seen with most harmful particle exposures and is directly linked to a number of adverse health outcomes. In the case of PM_{10} the inflammation caused by particles has the greatest effect in those with existing ill health. It may worsen existing airway disease such as asthma and bronchitis, provoking acute attacks and leading to increases in medication use and, on occasion, to unplanned hospital admissions. The local lung inflammation may also have an effect in worsening heart disease, culminating in deaths and hospitalisations. It should however be stressed that the risks of these severe effects are very low and that healthy people are unlikely to notice any effects of ambient air pollution.

In occupational settings, as discussed above (Section 1.5), the workforce typically is healthier than the general population, even in comparison to those of the same age, because people need to be reasonably healthy to secure and maintain employment. Nevertheless, concerns may justifiably arise when dust exposures at work are simultaneously *high*, (i.e. the dust is at high concentrations and people are exposed for long periods, possibly throughout a long working life) and *to particles* such as asbestos or quartz *that are relatively toxic* and are not generally encountered by the public, except at very low concentrations.

Under these circumstances, long-term and high exposures can result in inflammation that leads to scar formation, i.e. fibrosis and, for some pollutants (e.g. asbestos), to cancer.

3.3 PARTICLE CHARACTERISTICS AND MECHANISMS OF TOXICITY

3.3.1 Target cells

Two cell types can be seen as being especially important in the lung response to particles: (i) the epithelial cells, which line the lung acinus, and (ii) the alveolar macrophages that protect the acinus by scavenging and defending against bacteria.

- As particles are deposited in the lungs they first come into contact with lung lining fluid. This is a very thin layer of lipid-rich fluid that covers the epithelial cells. As mentioned below, this fluid may be important in chemical reactions that iron undergoes when an iron-rich particle lands there.
- Particles then encounter the epithelial cell layer. The contact between particles and epithelial cells occurs at the particle surface and so the outcome of the interaction depends on the surface characteristics of the particle.

- Macrophages then migrate to the particles and phagocytose ('swallow') them. The particle may affect the macrophage during phagocytosis and during residence inside it.

3.3.2 Release of transition metals

Some particles may release, from their surfaces, soluble components such as transition metals (technically, metallic ions that may exist in different valency states) that might damage cells or cause oxidative stress (Jimenez *et al*, 2000). Transition metals can generate the highly oxidative hydroxyl free radicals by a process of cyclical chemical reactions known as redox cycling (Donaldson *et al*, 2002). Release of transition metals has been postulated to play a role in the pro-inflammatory effects of PM_{10} (Gilmour *et al*, 1996), asbestos (Fubini *et al* 1995) and quartz (Castranova *et al*, 1997).

3.3.3 Low surface reactivity particles

Particles affect cells directly through contact with them and so the larger the aggregated surface area of the deposited particles, the larger the contact area between particles and cells and consequently the greater the effects of particles on the cells (compared with similar particles of lesser surface area). There is a clear relationship between surface area and pro-inflammatory effects *in vivo* (Duffin *et al*, 2002) and *in vitro* (Faux *et al*, 2003). Contact between particles and epithelial cells triggers release of chemotactic proteins called chemokines (such as IL-8) (Gilmour *et al*, 2003) which attract leukocytes to the site of deposition; i.e. cause inflammation. Particles may also activate soluble chemotactic components in the lung lining fluid (Warheit *et al*, 1985).

3.3.4 High reactivity particles

Some particles have much greater, or smaller, effects than others. For a given surface area, for example, quartz has a much more intense pro-inflammatory effect than does titanium dioxide (TiO_2) (Duffin *et al*, 2002). These effects are considered to be a consequence of the reactions between the quartz surface and cell membranes, resulting from the generation of free radicals at the quartz surface.

3.4 IMPACT ON THE DOSE METRIC – THE IMPORTANCE OF PARTICLE SURFACE

As noted earlier, the mass of particulate matter per unit volume of air (expressed as mg/m^3 or $\mu g/m^3$) is the most common exposure metric used for regulatory purposes. Clearly, however, particle mass is not the metric of choice if the component that mediates the response is the total surface area, the reactive surface area, or a metal component released from the surface. Therefore, in terms of quantifying risk, the mass dose metric may be limited in its usefulness.

This central role of the surface of particles means that better characterisation of the surface is one key component of future improved risk assessment.

3.5 CONCLUSIONS ON PARTICLES AND INFLAMMATION, AND IMPLICATIONS FOR MEASUREMENTS MADE IN THE PRESENT STUDY

We have noted that the effects of exposure to dust differ according to (i) the amount of dust exposure, which in turn depends on the concentration of the dust (whether measured by mass or number or surface area) and the length of time exposed; (ii) the toxicity of the dust, i.e. its potential to cause inflammation, and where that inflammation occurs, both of which are related to the size and shape of its particles and to its composition and surface properties; and (iii) the susceptibility of the population exposed.

Clearly it will be helpful if we can 'benchmark' the toxicity of tunnel dust relative to other, well-researched ('exemplar') dusts. At the least, we need to be able to say whether tunnel dust is similar to a high toxicity dust like quartz, a low-toxicity dust like TiO_2, or some dust of intermediate toxicity. To do this, we have adopted several complementary approaches. These are related as follows to the conclusions from the above brief review of particle toxicity.

First, we have noted the importance of considering more than one measure of the concentration of particulate matter (PM). There are no currently well-established methods of characterising particle surface area directly. In this study, we have focussed therefore on both particle mass (e.g. in units such as $\mu g/m^3$) and particle number as giving two different characterisations, both of which are relevant to a dust's potential to cause damage – not only its potential to cause inflammation, but also where that inflammation is likely to occur.

Secondly, although we recognise also the importance of the physico-chemistry of the particle surface we have not attempted a detailed characterisation of the surface properties of tunnel dust. That is, because even if such a characterisation were practicable – and this is a subject of active research currently – there is not yet a basis in evidence for inferring from its surface properties the likely toxicity of tunnel dust compared with other, 'exemplar', dusts. Instead we have aimed to establish the inflammatory potential of tunnel dust in two complementary ways.

a. We have examined again the composition of tunnel dust and confirmed – see Chapter 5, later – that the main component is iron. Consequently, we have paid particular attention to studies of the effects on health of long-term exposure to iron. The role of iron is reviewed in Chapter 4.

b. We have noted that the dominant mechanistic hypothesis for the cellular action of PM_{10} continues to be oxidative stress and that epithelial cells lining the lung acinus play a key role in the initiation and progression of particle-induced pulmonary injury (Donaldson et al , 2003) . Our working hypothesis is that interactions between particles and epithelial cells cause oxidative stress, leading to a cascade of events that underlie the pro-inflammatory effects associated with inhaled particles. These events culminate in the expression of chemokines such as the IL-8 that attract neutrophils (PMN) and alveolar macrophages (AM) to the site of dust deposition. This accumulation of neutrophils and macrophages in the lungs is the hallmark of inflammation in the whole animal *in vivo*.

Against this background, we have used two kinds of *in vitro* experiments to investigate the potential of inhaled tunnel dust to cause inflammation.

- We have measured the pure chemical ability of the particles to generate harmful free radicals, in the absence of cells ('cell-free' tests).
- We have measured the effects of tunnel dust and other particles on human lung epithelial cells, as represented by A549 cells (cellular tests).

Details of methods and results are given in Chapter 6.

4. IRON AND HUMAN HEALTH

4.1 THE METABOLIC ROLE OF IRON

Since the evidence to date indicates that iron is the main component of the dust present in the London Underground, it is appropriate to consider the relevance of this metal to human health. Iron is essential to life and health and is found in the body mainly in blood, in haemoglobin and as an iron-protein complex, ferritin. The former is the agent in red blood cells responsible for carrying oxygen while in haemoglobin iron is combined with a protein that transports it to the places in the body where it is used or stored. The iron stores are in the mononuclear/phagocytic cell system. A similar chemical to haemoglobin, myoglobin, also contains iron; it is found in muscle. Iron is also present in other body tissues as enzymes and in mononuclear cells in small amounts. The total amount of iron in the body is remarkably small, around 3 to 4 grams.

Iron is absorbed into the body through the upper intestine, the mechanism being regulated in order that absorption is increased when the body's requirements are greater. This process is aided by exposure of food in the stomach to hydrochloric acid. Once taken in, iron is conserved in the body and recycled from old cells (red blood cells live for about 120 days) into the bone marrow for incorporation into new ones. There is no active mechanism for removal; loss of iron from the body occurs with bleeding and from shedding of cells from skin and bowel, so little usually needs to be replaced. The daily requirement in men is rather less than 1mg, whereas in menstruating women it is over double this and may be higher depending on the amount of blood lost.

4.2 THE AVAILABILITY OF IRON IN THE ENVIRONMENT

Iron is the fourth most abundant element in the earth's crust, after carbon, silicon and oxygen. The element exists in two states, divalent ferrous and trivalent ferric iron. Although in ionic form it is a reactive chemical (see below), in nature it is combined usually with oxygen as ferric oxide (commonly observed as rust) or as a carbonate or sulphide salt. The oxide is usually hydrolysed and is metabolically inaccessible.

Humans obtain iron from their diet, largely from red meat in the form of haem and from certain vegetables as organic iron. Only a small proportion of ingested iron (about 10%) is actually absorbed, unless the body's requirements are increased. Absorption is in the reduced, ferrous, form and is thus enabled by the action of stomach acid and may be increased by ingestion of acids such as vitamin C and amino acids with the iron in the food. Once the iron has entered the gut cells it is bound by apoferritin which is present in limited supply, the iron-protein complex being ferritin. It then requires the presence of free plasma transferrin to move it out into the circulation. Thus a limit is imposed on the amount of available iron that can be absorbed and used by the body. Any residual iron in the cells of the gut is removed when the cells themselves die and pass into the gut lumen.

Humans may also be exposed to iron in the workplace environment, iron being a recognised component of industrial dusts in many occupations. These range from the welding and cutting of metal, where a fume containing up to 30 or 40% iron oxide is generated, to iron ore mining, fettling and metal working, where the iron oxide is found in the form of larger particles. Inhalation of iron-containing dust is thus a recognised accompaniment of a number of trades. Owing to the nature of industry, however, in most of these the iron oxide is but one component of a mixed dust or fume. Again, the iron in these circumstances is in the oxidised, ferric form and is relatively insoluble. However, the environment of the lung acinus is a reducing one, containing vitamin C and reduced glutathione, so there is potential for the ferric iron to be converted to the more toxic ferrous form (Quinlan *et al* 2002). Recent

investigations have shown that the lung reacts to inhalation of iron by producing increased amounts of ferritin, which has bound and inactivated the iron, and lactoferrin and transferrin receptors, which increase access of the iron to the ferritin protein (Ghio *et al* 1998). Thus the lung has a sophisticated mechanism for preventing absorption of toxic iron and for ensuring that it can be dealt with by the normal macrophage defences. In evolutionary terms this mechanism is unlikely to have developed as a response to inhalation of iron in recent history, but is more likely to reflect the embryological development of the lung from the fetal foregut.

4.3 IRON AND THE LUNGS

Iron will access the lungs only in particulate form, as ferric oxide or as one of the industrial iron ores, haematite (also ferric oxide), siderite (ferric carbonate) or pyrite (ferric sulphide).

Much of the iron inhaled in particles will be engulfed directly by macrophages for transport out of the lung. Since there is an active mechanism in the lung for its combination with protein and inactivation as ferritin, any free iron is dealt with in this way and also removed in macrophages. In the insoluble form in which the iron is presented to the macrophage, it does not become involved in metabolic processes and thus does not contribute to the body's iron stores, rather being removed up the airways.

As indicated above, iron may be presented to the lungs in different combinations. It may also be presented in different physical form. This means that the consequences of inhaling iron differ in different trades.

- Iron miners are usually exposed to quartz as well as iron, and are therefore at risk of the form of lung fibrosis known as silicosis.
- Haematite miners in Cumbria were also exposed to radon gas and had an increased risk of lung cancer as a consequence (Boyd *et al* 1970).
- Fettlers are exposed to quartz and may develop silicosis.
- Welders, in contrast to all these other groups of workers, are exposed to iron oxide as a fume, that is, in the form of nanometer-sized particles. In addition, that fume commonly contains other metals, typically chromium, nickel and manganese, from the steel and the electrodes (see IARC 1990). Welders may also be exposed to toxic gases such as ozone or oxides of nitrogen. The health effects of welding are discussed further in Chapter 7.
- There is probably only one group of workers, silver polishers, who are exposed to essentially pure ferric oxide. They have been shown to accumulate iron in the lung without serious consequences (Barrie and Harding 1947).

4.4 DISEASES ASSOCIATED WITH IRON

The best-known medical condition associated with iron is haemochromatosis (see Cox 2003). This is inherited, and the genetic abnormality is relatively common in the British population, the homozygous state occurring in about one in 100 to one in 400 individuals. However, only a small proportion of such individuals actually develop the disease – in one large study this was one in 150; i.e. one in 150 of the subgroup that is in the homozygous state. Haemochromatosis is therefore a rare condition. It is caused by a metabolic alteration in the manner in which the gut absorbs iron, resulting in too much being taken in. Once the body stores are full, excess free iron accumulates in organs such as the liver, pancreas and heart, leading to fibrosis, manifesting as the serious diseases cirrhosis, diabetes and heart failure. A similar condition may occur as a consequence of repeated blood transfusions among patients with certain blood disorders.

There is however no evidence that it occurs as a result of absorption of iron through the lungs. The workers with the highest exposures to iron are welders, in whom historical exposures of up to 10,000µg/m^3 (or indeed much higher) have been recorded (IARC 1990). It is not

unreasonable to suppose that such workers might have inhaled up to 50mg of iron over a shift, far higher than the daily requirement of an adult male of 1mg. In spite of this, no excess risk of haemochromatosis has ever been reported in welders. The likely explanation is that the iron is retained in the lungs in the defensive cells, macrophages, and gradually removed.

With respect to lung disease, the issue of the effects of iron is complicated by the mixed dusts that most iron-exposed workers inhale, as mentioned above. The only condition specific to iron inhalation is called siderosis, and has been described in welders, silver polishers and haematite miners. It is characterised by accumulation of iron in macrophages in the lung, which because of the high radio-density of iron may become visible as spots on chest radiographs. Silver polishers exposed to high concentrations of iron oxide over decades have been found to have no lung fibrosis or excess accumulation of iron in internal tissues such as the liver, the iron being found in macrophages (Barrie and Harding 1947). In rare extreme cases heavy iron accumulation has been associated with minimal lung fibrosis in welders but progressive fibrosis and impairment of lung function has not been described. It is regarded medically as a "benign pneumoconiosis", that is it causes x-ray shadows without harming the patient (see Morgan, 1995). The x-ray shadows regress after exposure to iron ceases.

There is a known association of welding with risk of pneumonia. This first came to light from study of occupational mortality statistics, in which the trades of welding, moulding and core making, all of which involve exposure to metal fume, were observed to be over-represented among those who had died of pneumonia (Coggon *et al* 1994). Subsequent studies have shown that individuals of working age with pneumonia are almost twice as likely to have been exposed recently to metal fumes compared with control patients with non-chest illnesses (Palmer *et al* 2003). The question remains as to whether the important exposure is to the fume or to the metal or both, but as iron is known to be a growth factor for bacteria it seems likely that inhalation of the metal in this form predisposes to pneumonia.

5. INVESTIGATIONS OF THE PHYSICO-CHEMICAL CHARACTERISTICS OF TUNNEL DUST

5.1 OBJECTIVES

The main objective of this part of the work was to characterise the physical properties and composition of the airborne dust being used in the toxicity tests, and to make measurements that would allow estimation of the personal exposure levels of London Underground staff. In particular, we aimed to:

- measure long-term particle mass concentrations ($PM_{2.5}$) and particle number concentrations on station platforms and in the cabs of trains;
- make additional measurements of airborne mass concentration for comparative purposes (PM_{10} and respirable dust);
- measure airborne size distributions and estimate particle surface area distributions for airborne dust on station platforms;
- measure the concentration of the principal metal constituents and quartz content of the airborne dust on station platforms;
- estimate the likely exposure levels of drivers and station staff;
- obtain comparative data on the airborne dust concentrations at street level in London.

5.2 METHODS

5.2.1 Sites for measurements

Guided by Trade Union representatives and London Underground staff, we selected three sites for the study. These were:

- Holland Park station (at the west end of the eastbound platform on the Central Line);
- Hampstead station (at the south end of the northbound platform on the Northern Line) and
- Oxford Circus station (at the north end of the northbound platform on the Victoria Line).

These sites were selected after discussions with members of the London Underground Dust Action Group and an inspection visit to ensure that it was practicable to site the sampling equipment on the platform. The stations were believed to cover a range of depths and circumstances, and each was situated on a different line.

5.2.2 Platform measurements

The measurements were made in January 2003, using the same equipment as was used in the pilot study (described in the Appendix).

Two direct reading monitors were used in the study: a P-Trak and DustTrak. $PM_{2.5}$ was measured using a portable battery operated DustTrak light scattering monitor (manufactured by TSI Inc., St. Paul, Minnesota, USA). This device continuously draws air through a $PM_{2.5}$ size-selective inlet into the sensing chamber where a beam of laser light is shone through the air stream. The particles present in the air act like tiny mirrors scattering light in all directions. A lens at right angles to both the airflow and laser beam collects part of the scattered light and focuses it onto a sensor. The amount of light scattered is proportional to the mass of the particles in the air. The DustTrak monitor must be calibrated because the light scattering response is dependent on the type of dust being sampled.

The particle number concentration was measured using a P-Trak monitor (TSI Inc.). The operation principle is similar to the DustTrak. Particles are again drawn through the P-Trak

using a built-in pump. Before entering the sensing zone the particles pass through a saturator tube where they mix with an alcohol vapour and the mixture is then drawn into a condenser tube where the alcohol condenses on the particles causing them to grow into larger droplets that can be counted more easily. These droplets then pass through a laser beam producing scattered light pulses that are sensed by a photodetector and counted to determine particle number concentration. The P-Trak is designed to count particles between 0.02 and 1μm.

Three other sampling devices were used to collect airborne dust samples for subsequent laboratory analysis. These were:

- a $PM_{2.5}$ sampler;
- a PM_{10} sampler and
- a respirable dust sampler (which approximates to a $PM_{3.5}$ sampler).

All of these comprise a battery-operated sampling pump and a sampling head connected to the pump by flexible plastic tubing. The air is drawn through a pre-weighed filter located in the sampling head and any particles are trapped on the filter. The sampling heads are designed to select the particle sizes of interest. So for example the PM_{10} and $PM_{2.5}$ sampling heads contain a section of polyurethane foam which is designed to remove particles greater than the stated size according to the agreed standard criteria. The respirable dust sampler (Casella) comprises a cyclone pre-selector, which removes oversize particles by a centrifugal process. The flow rate for the cyclone sampler was adjusted to 2.2 litres/min, to ensure that the size selection corresponded to the International Standard Organisation criteria.

The airflow rate through the sampling head was measured at the beginning and end of the sampling period and at a number of intermediate times. At the end of the sampling all of the filters were returned to the IOM laboratory where they were re-weighed and, if required, subjected to further chemical analysis. The mass concentration of dust was calculated from the change in filter weight, the duration of sampling and the average flow rate.

All of the respirable dust samples were analysed by infra-red spectroscopy to determine the mass of quartz on the filter. This was done using the method published by the Health and Safety Executive for direct on filter assessment of quartz in respirable dust samples (HSE, 1987).

Sampling pumps and other bulky equipment were placed inside a cupboard on Holland Park (the same position as in the pilot study) and above a cupboard at Hampstead station. The sampling heads were then located on the outside of the cupboard approximately 2.5m above the platform. In both situations the sampling site was located at the far end of the platform, i.e. nearest to where trains entered the station. For the measurements made at Oxford Circus, the equipment was placed on a table at the far end of the platform, separated from the passengers by a barrier.

For practical reasons and to cover a whole shift, measurements typically began around 7am and continued until 5pm. This included one of the two peaks of traffic in line with the shift patterns of drivers. At the beginning of each day the flow rate through each pump-based sampler was measured using a calibrated bubble flow-meter. These measurements were repeated periodically during the day. If the measured flow rate deviated by more than 10% from the initial reading the system was investigated to remedy any potential problems and, if necessary, flow was readjusted. A final set of flow rate measurements was made at the end of the sampling period. At the end of each day's sampling the filters were changed and placed in a secure storage area.

Samples were collected for $PM_{2.5}$, PM_{10} and respirable dust using the same methods as in the pilot study. In addition, samples were collected using battery-operated pumps with open face

cowl holders fitted with Nuclepore polycarbonate filters. These samples were collected at a relatively low flow rate for the purpose of analysing the particle size distribution by transmission electron microscopy. The Nuclepore filters were coated with several layers of carbon. Portions of the carbon-coated filters were then excised and mounted on 200 square mesh, 3.05mm copper Transmission Electron Microscope (TEM) grids. The copper grids were placed in a Jaffe washer, filled with chloroform and left overnight. The chloroform digested the polycarbonate filters and left the particles suspended in the carbon. Each copper grid was placed in the TEM and examined at low magnification for uniformity of deposit. The particle analysis was carried out using a Link AN10000 semi-automatic system with the TEM set at 5000X magnification on a slow scan speed. For each sample, approximately 1000 particles were analysed (each mean diameter being the result of ten individual measurements per particle). It will be noted that this measures actual rather than aerodynamic diameter, and therefore underestimates the aerodynamic diameter of dense, iron-containing particles.

The filters from the $PM_{2.5}$, respirable dust and PM_{10} samples were weighed and the weight gain was adjusted for the average field blank weights. Concentrations in $\mu g/m^3$ were calculated as described in the pilot study (see Appendix 1). The respirable dust samples were again analysed for quartz content using infrared spectroscopy. The $PM_{2.5}$ samples were analysed by inductively coupled plasma/atomic emission spectrometry for five metals: iron, chromium, copper, zinc, and manganese (OSHA, 1991).

Measurements of particle mass concentrations ($PM_{2.5}$) were also made using a DustTrak and particle number concentrations using the P-Trak. The results from the DustTrak were adjusted using the gravimetric $PM_{2.5}$ measurements made on the platform. This was done by scaling the Average DustTrak data to correspond to the measurement made using the gravimetric sampler; in this way we are confident that these data are properly representative.

Long-term high-volume $PM_{2.5}$ samples were also collected from the roof of the London Underground Ltd Griffith House offices in central London. The methods used here were identical to those used to collect samples on the station platform. These samples were intended for use in the toxicity studies as a comparison dust.

5.2.3 Sampling to assess personal exposure of train drivers (cab measurements)

Personal sampling in the drivers' cabs was also carried out for three days on each line. This was done using a second set of DustTrak and P-Trak samplers. Both samplers were positioned inside the cab as close as possible to the driver's breathing zone and the driver was accompanied throughout this time by one of the researchers. The sampling time started with the booking time for the driver until the end of the shift. These measurements were continuous except for lunch breaks, which typically lasted between 30 and 60 minutes. The results were adjusted to represent the whole shift, except when stated otherwise.

The times when the driver was underground and in the cab on the surface were recorded to enable comparisons of the concentration in each situation to be made. The platform data used for comparison comprised the concentrations half an hour before and after the time when the measurements were made on the surface. Data from the DustTrak and P-Trak used inside the cabs were adjusted so that they corresponded to the platform instruments, which had been adjusted to correspond to the gravimetric $PM_{2.5}$ data. This adjustment was made on the basis of a number of side-by-side comparisons of the four instruments on the station platform.

5.3 RESULTS

5.3.1 Particle size data from each station

Examination of the airborne tunnel dust by transmission electron microscope showed that the aerosol collected on each station platform was qualitatively similar. Figure 5.1 shows a photomicrograph of the dust from Holland Park station.

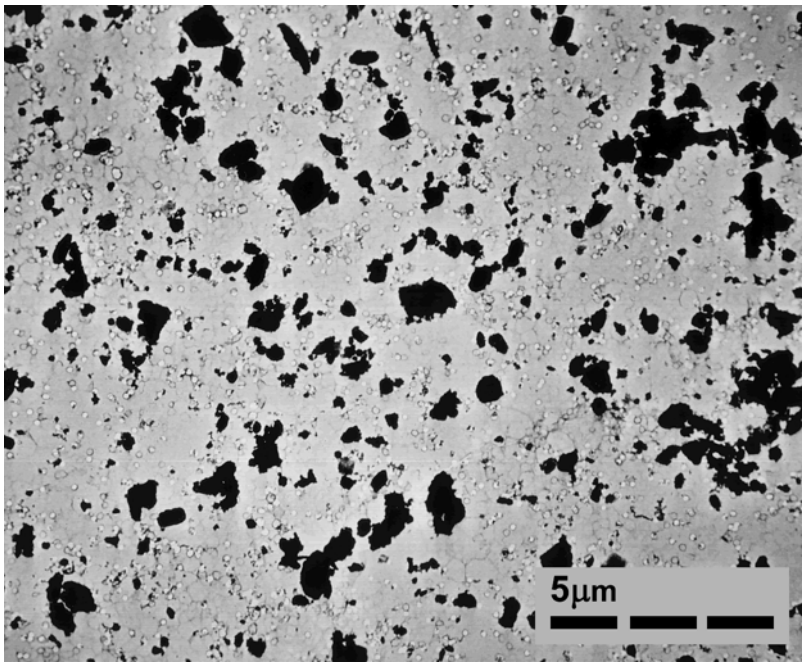

Figure 5.1 Photomicrograph of airborne tunnel dust from Holland Park station

The scale bar in the bottom right hand corner of image is 15µm long and it is therefore clear that many of the particles are smaller than 1µm. Many of the particles appear to comprise several smaller particles aggregated together. The image analysis software used to measure the particle size distributions attempts to separate individual particles, although it is likely that in a number of cases the larger measured “particles” are in fact aggregates.

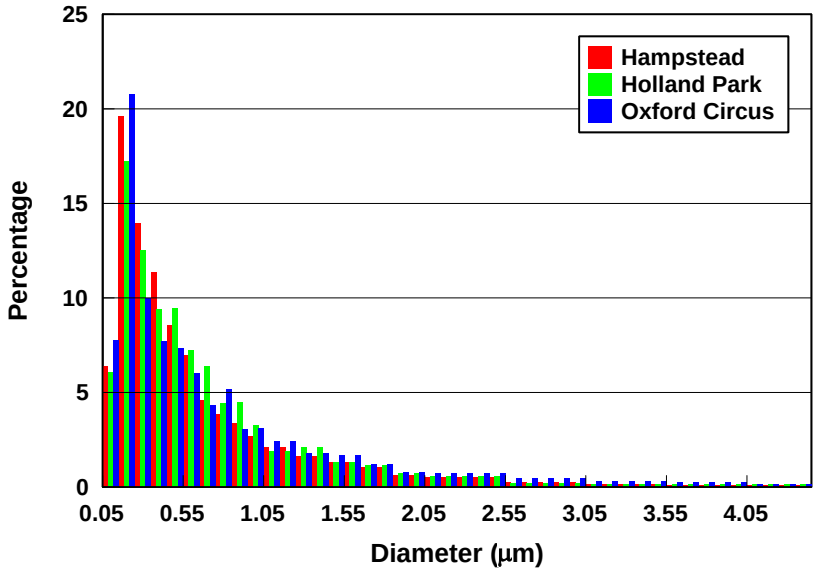

Figure 5.2 Particle size distributions for each station platform

Figure 5.2 shows the particle size measurements from the three station platforms in the form of a histogram.

The distributions from the three stations are remarkably similar, with the data from Hampstead being slightly finer. The median diameter for the dust from Oxford Circus and Holland Park is 0.4μm and for Hampstead 0.35μm. In each case about 80% of the particles have a diameter less than 1μm. Again, note that this underestimates the aerodynamic diameter of the denser particles.

The median size of airborne dust found in urban streets is generally finer than this. For example, a report from the Committee on the Medical Effects of Air Pollutants quotes the size of airborne dust from the air in London to range from 0.09 to 0.13μm (COMEAP, 1995).

5.3.2 Summarised data for the P-trak and DustTrak samplers

Table 5.1 Average dust concentrations on the platform for the three stations

Station	Location	$PM_{2.5}$ (μg/m^3) Mean ± SD	PNC (particles/cm^3) Mean ± SD
Holland Park Station	Central Line	300 ± 50	29,000 ± 6,700
Hampstead Station	Northern Line	480 ± 26	14,000 ± 2,500
Oxford Circus Station	Victoria Line	270 ± 21	24,000 ± 4,500

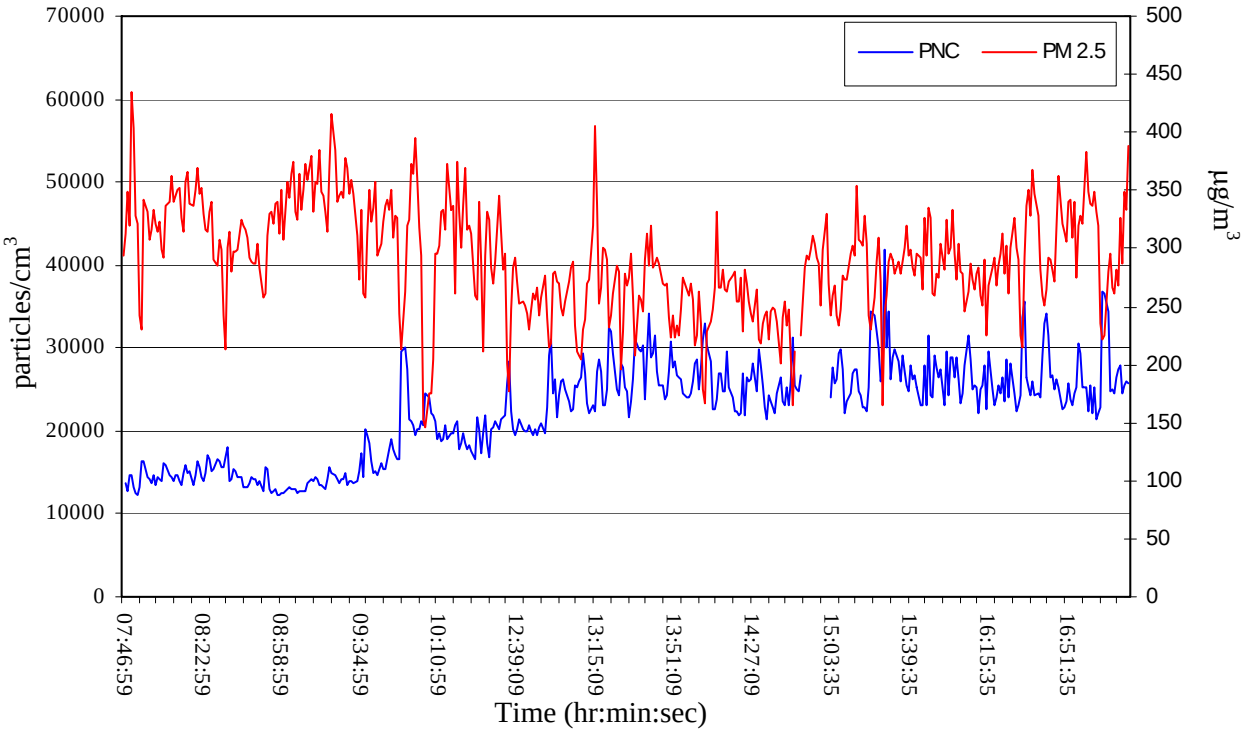

Figure 5.1 Dust concentrations at Oxford Circus Station *(Victoria Line) 15/01/03*

The results from the DustTrak and P-Trak monitoring at the three stations, each over three days, are shown in Table 5.1. Average $PM_{2.5}$ concentrations ranged from 270 g/m^3 at Oxford Circus station to 480 g/m^3 at Hampstead station. The corresponding data for PNC ranged from 14,000 to 29,000 particles/cm^3, although here the lowest average level was seen at Hampstead station.

Further measurements were made at a later date at sites on the surface close to the station entrances at Hampstead and Oxford Circus, but away from vehicular traffic. These measurements showed that although the gravimetric measurements at the two sites were comparable, the particle number concentrations at Hampstead were much lower than at Oxford Circus. We have therefore concluded that the lower particle number concentrations for Hampstead in the table above reflect the lower concentration in the surface air that is being drawn into the underground at this location.

Figure 5.3 shows data from both instruments for a single day's sampling. Here the decline in $PM_{2.5}$ concentration from morning to afternoon and then the rise later in the day is less pronounced than was seen in the pilot study (see Appendix – Figure A1.2), although it is still apparent. On this occasion the particle number concentration rose steadily from early morning until just after midday and then remained fairly constant, in keeping with induction of outside air into the system, and the concentrations of $PM_{2.5}$ seemed to reflect the pattern of rail traffic through the station.

5.3.3 Comparison of concentrations on the platform and the surface

Table 5.2 Ratio of concentrations on the underground platform to surface concentration measurements on various days of sampling

Station	Line	Ratio of the concentration underground to that on the surface	
		PNC	$PM_{2.5}$
Hampstead Station	Northern Line	0.38	16.0
Oxford Circus	Victoria Line	*	9.2
		0.60	8.0
		0.68	7.1

* data not available

Measurements on the platform and on the surface were made on one day at Hampstead (Northern Line) and all three days at Oxford Circus (Victoria Line). The results were similar to those seen in the pilot study; high number counts on the surface and low gravimetric concentrations, with the opposite pattern underground (Table 5.2). This was true at both stations, although the differences were somewhat greater at Hampstead than Oxford Circus (approximately a factor of two different for both the PNC and $PM_{2.5}$ data). Figure 5.4 shows the platform and corresponding surface data for the 17/01/03 at Oxford Circus (Victoria Line). The platform data used for this comparison were taken half an hour before and after recording the surface data.

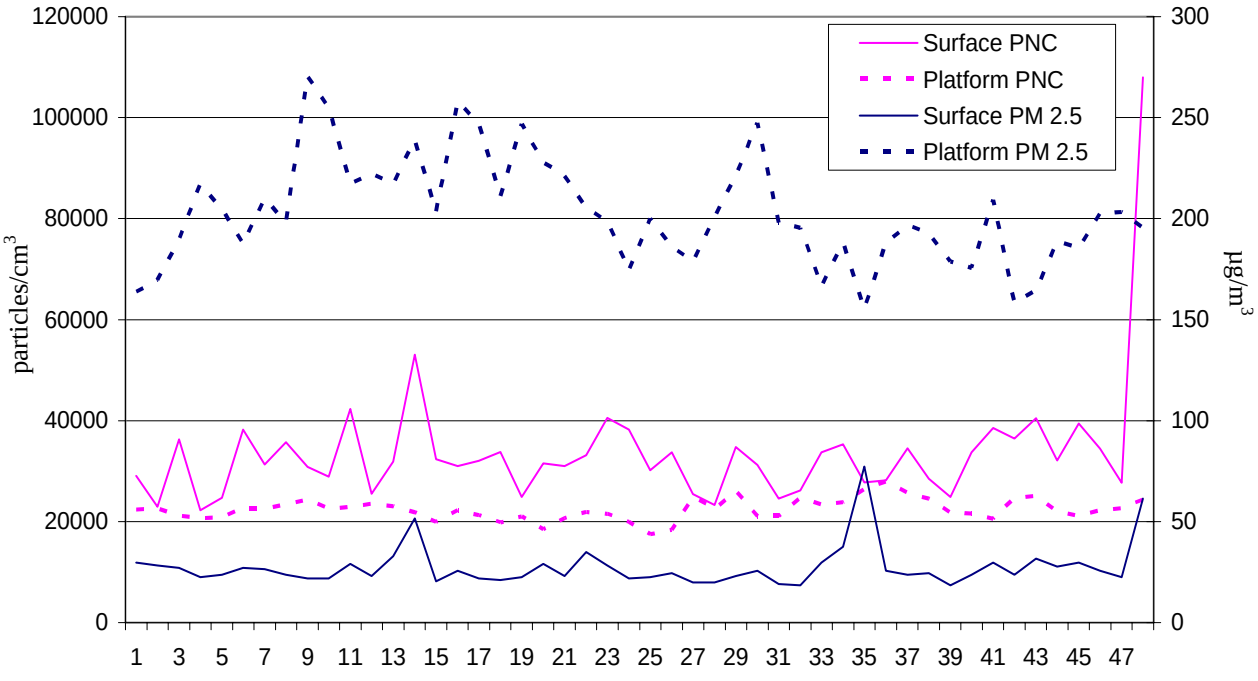

Figure 5.4 Surface dust concentrations and corresponding platform data

5.3.4 Concentrations in the cab for the three lines

Table 5.3 summarises the concentrations in the cabs of the trains on the three lines over three separate days. Both particle number concentration and gravimetric data are shown.

Table 5.3 Mean and standard deviation of PM2.5 and PNC in drivers' cabs

Location	$PM_{2.5}$ (μg/m^3) Mean ± SD	PNC (particles/cm^3) Mean ± SD
Central Line	130 ± 12	23,000 ± 3,500
Northern Line	200 ± 1	17,000 ± 1,700
Victoria Line	180 ± 13	22,000 ± 5,000

The measurements for all the three lines showed average levels of $PM_{2.5}$ ranging from 130 to 200μg/m^3, with the highest concentrations on the Northern line and the lowest on the Central line. PNC were lowest on the Northern line (17,000 particles/cm^3 compared with 22,000 and 23,000 particles/cm^3 on the other two lines). It should be noted that the cab data cover the entire shift, including measurements when the train was underground and on the surface. The number counts will reflect principally the exposure when the train is above ground while the mass reflects the underground exposure.

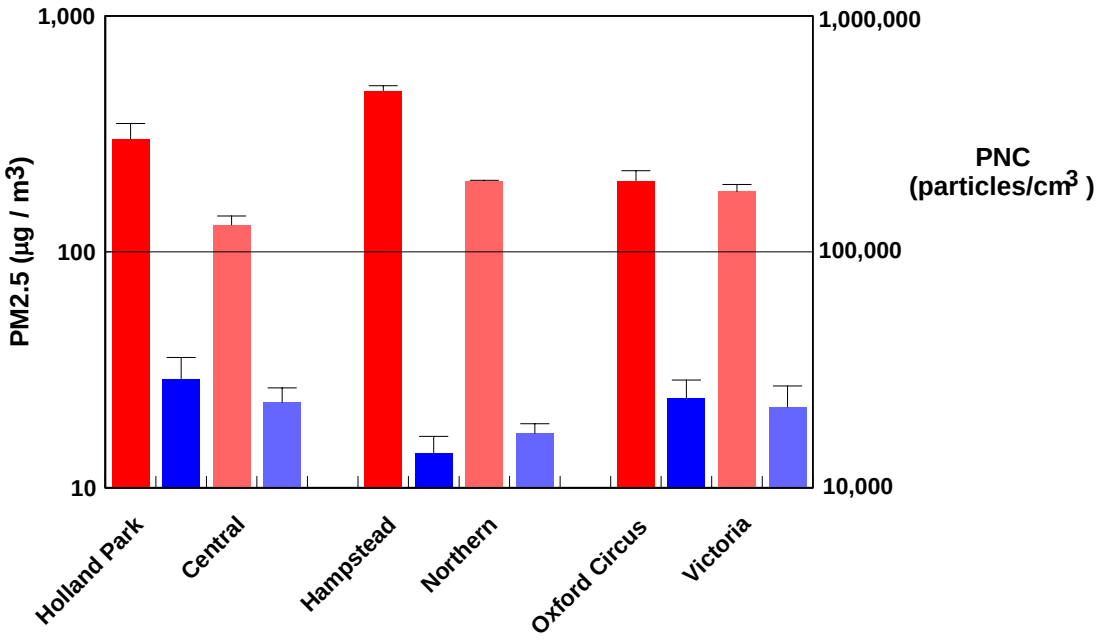

Figure 5.5 Comparison of data from the station and cab sampling. The columns represent the average of the measurements and the bars the variation.

Data from both the cab sampling and the station sampling are shown in Figure 5.5. The heavier shaded columns represent data from the stations and the lighter shaded columns the cab samples, indicated by the line (Central, Northern, Victoria). Blue (lower columns) represents PNC and red (higher columns) the $PM_{2.5}$ data.

The highest gravimetric concentrations and the lowest average PNCs were seen at Hampstead station and the Northern line cab samples, where the trains run through Hampstead.

Figure 5.6 shows continuous data for dust concentrations, both $PM_{2.5}$ and number concentration, over a whole shift in the cab of a train travelling on the Central line. Concentrations were elevated corresponding to periods when the train was in tunnels (Fig 5.6, next page). Interestingly, in this case the particle number concentrations also showed increases at similar times, the correlation between the two concentration measures being 0.43. The probable explanation is given in the next section.

5.3.5 Concentrations in cabs while trains were in and out of tunnels for the three lines

Data on the ratio of concentrations measured while the trains were in tunnels to the concentrations at times when they were out of tunnels, for $PM_{2.5}$ and PNC, are shown in Table 5.4. There are only data for one day for the Victoria line since for the remaining two days of measurements the trains were underground for the whole shift.

Table 5.4 Ratio of concentrations in cabs while underground to the concentrations while the train was on the surface

Line	Ratio of the concentration underground in train cabs to that on the surface	
	PNC	**PM 2.5**
CENTRAL LINE	1.85	9.26
	1.55	9.03
	1.65	10.9
NORTHERN LINE	1.63	6.94
	1.04	4.74
	1.35	8.35
VICTORIA LINE	1.21	6.58

The $PM_{2.5}$ concentrations were substantially higher when the trains were in tunnels, which is similar to the trend that was found on the station platforms and noted above (Table 5.2). However, the PNCs were also slightly higher while the trains were in tunnels, even though at those stations the number concentration was higher on the surface. The main source of the very fine particles that contribute to the PNC is road traffic on the surface, while the main sources of the particles making up the $PM_{2.5}$ concentrations are in the tunnels. The different pattern of data shown in Tables 5.2 and 5.4 is almost certainly because the air in the tunnels comes from the surrounding area at street level. The underground sections are mostly found towards the centre of London where the PNCs above ground are generally higher than the surrounding areas. The sections of line on the surface tended to be further from the centre of London where the surface PNCs are lower. Therefore, the data for PNC in Table 5.4 represent a comparison of PNCs in the central parts of London with those areas further out, as for example can be seen by the lower PNCs at Hampstead compared with the other two stations (Table 5.1).

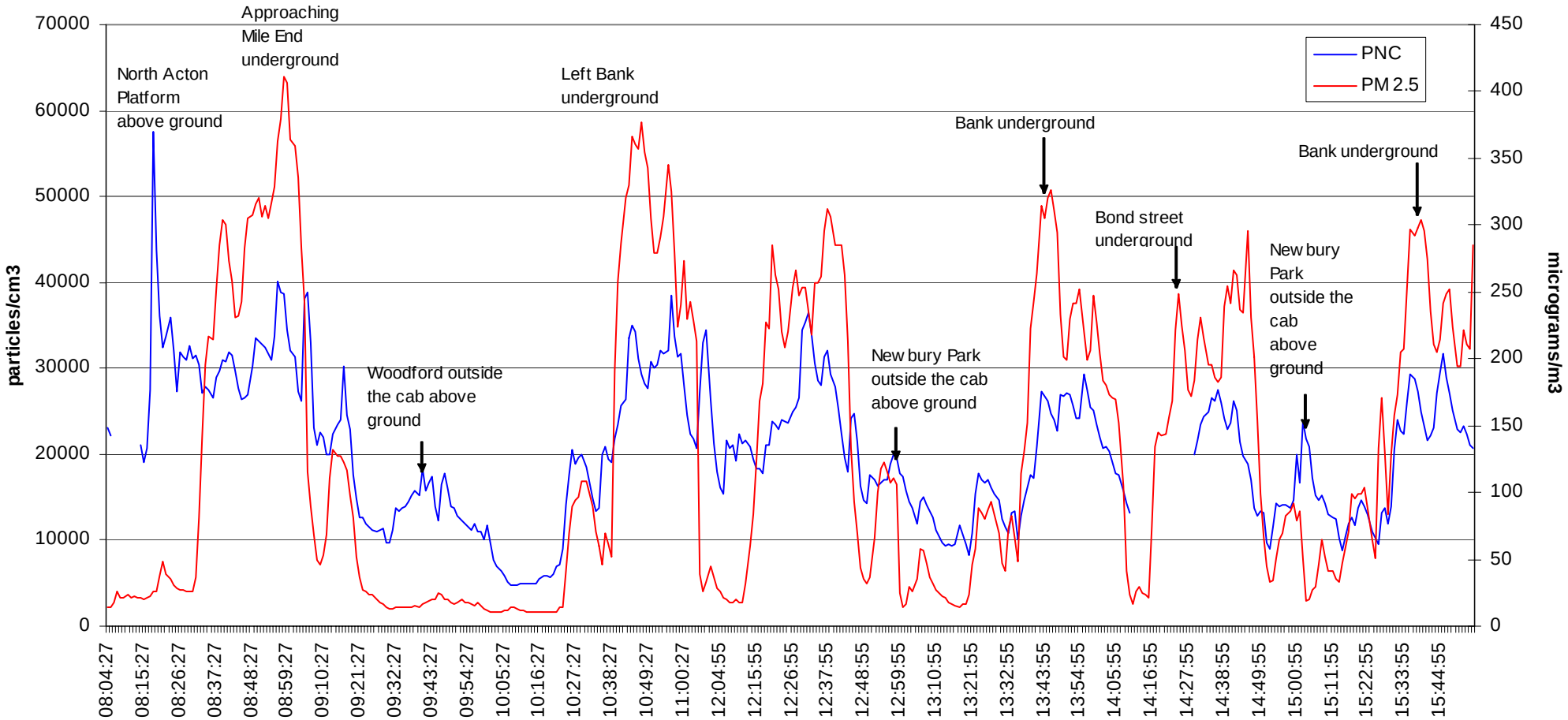

Figure 5.6 Cab data for the whole shift 07/01/03 Central Line

5.3.6 Results from pump-based monitoring

Data from the pump-based sampling on station platforms is shown in Table 5.5. Here the highest concentrations were again seen at Hampstead and this is true for $PM_{2.5}$, PM_{10} and respirable dust. In addition the lowest measured concentrations at all three stations were for $PM_{2.5}$, which was expected since this is the finest fraction of the dust. The highest concentrations were for PM_{10} for analogous reasons.

Table 5.5 Data from pump-based monitoring on station platforms

Station	Location	$PM_{2.5}$ (μg/m^3) Mean ± SD	Respirable (μg/m^3) Mean ± SD	PM_{10} (μg/m^3) Mean ± SD
Holland Park	Central Line	310 ± 17	790 ± 37	1000 ± 160
Hampstead	Northern Line	420 ± 14	1400 ± 47	1500 ± 120
Oxford Circus*	Victoria Line	300 ± 18	920 ± 63	1100 ± 33

* two PM_{10} measurements excluded because of errors in sampling/analysis

Table 5.6 Proportions of quartz and metals in dust samples

Station	Location	Quartz in respirable dust (%) Mean ± SD	Iron in $PM_{2.5}$ (%) Mean ± SD
Holland Park	Central Line	1.0 ± 0.5	90 ± 17
Hampstead	Northern Line	2.0 ± 0.3	93 ± 10
Oxford Circus*	Victoria Line	0.9 ± 0.1	85 ± 18

The respirable samples were all analysed for quartz and metal concentration and these data are summarised in Table 5.6. Quartz made up approximately 2% of the respirable dust in Hampstead and approximately 1% at the other two stations. Almost all of the dust in the $PM_{2.5}$ samples was analysed as iron – note the quartz and metal composition data are not strictly comparable because they are analysed on different size fractions. There were small amounts of chromium (0.1-0.2%), manganese (0.6-1%) and copper (0.1-1.5%). No zinc was detected on any of the samples (<0.1%).

The proportion of quartz in these samples was slightly lower than measured in the pilot study, but it should be remembered that the quartz content is a very small proportion of the respirable dust and the total amount of dust collected is much smaller than would typically be collected in workplaces where quartz was present. The measurements are close to the analytical detection limit and although we can be sure the proportion of quartz and the concentration of quartz in the air are very low the exact proportion cannot be accurately determined.

5.4 LONDON URBAN AIRBORNE DUST DATA

PM_{10} and $PM_{2.5}$ data from London Bloomsbury and either Brent or Marylebone Road were obtained for comparison. Both Bloomsbury and Marylebone Road are in central London, but

Brent is approximately 10km North and West of these sites. The monitoring data are shown in Table 5.7 for each of the days that measurements were made on the station platforms.

Table 5.7 Daily average concentrations ofPM_{10} and $PM_{2.5}$ for two locations in London corresponding to the sampling programme underground

Station/ date	PM_{10} ($\mu g/m^3$)		$PM_{2.5}$ ($\mu g/m^3$)	
	Bloomsbury	**Brent**	**Bloomsbury**	**Marylebone Road**
Holland Park				
07/01/2003	27.0	12.0	11.5	14.2
08/01/2003	27.1	15.2	13.4	11.1
09/01/2003	27.7	15.9	12.1	9.8
Average	**27.3**	**14.3**	**12.4**	**11.7**
Hampstead				
10/01/2003	26.2	14.4	11.5	8.0
13/01/2003	22.9	11.5	7.8	16.0
14/01/2003	24.7	14.4	7.0	18.0
Average	**24.6**	**13.4**	**8.8**	**14.0**
Oxford Circus				
15/01/2003	31.4	20.7	10.4	21.5
16/01/2003	31.2	23.1	9.7	23.0
17/01/2003	24.6	14.8	8.8	16.2
Average	**29.1**	**19.5**	**9.6**	**20.2**

For example, on the first day of sampling at Holland Park (07/01/03) the PM_{10} concentration at Bloomsbury was 27.0$\mu g/m^3$ and the corresponding $PM_{2.5}$ concentration was 11.5$\mu g/m^3$. At Marylebone Road the $PM_{2.5}$ concentration on this day was 14.2$\mu g/m^3$. Average recorded $PM_{2.5}$ level for London Bloomsbury was 10$\mu g/m^3$ and for London Marylebone 15$\mu g/m^3$.

There was a consistent difference in PM_{10} concentration between Bloomsbury (27.0$\mu g/m^3$) and Brent (15.8$\mu g/m^3$), reflecting the difference on airborne particle levels between central and outer London.

There are currently no data available other than those we have provided on particle number concentration in central London, and no measurements of particle size distribution have been made for ambient air comparable to those we have made for underground air. The very large majority of such particles by number are less than 100nm.

5.5 ESTIMATE OF LUL WORKER EXPOSURE TO AIRBORNE DUST

In order to interpret the information from the toxicity evaluation it is necessary to have some estimate of the personal exposure of the staff working in the underground. The information from the monitoring exercises provides the basis for these estimates, although in addition it is necessary to know the duration of time spent in the environment measured.

For the drivers, the measurement data are directly representative of their conditions since they were collected inside the cab for the majority of the work shift. For the station staff this is less clear, since the staff, do not spend all of their time on the platform, and perhaps not even the majority of their shift. The best available information is that the maximum amount of time of

staff on the platform at Holland Park is 2 hours, at Hampstead 1.5 hours and at Oxford Circus 5 hours. The $PM_{2.5}$ exposure levels for station staff are therefore based on a time-weighted average of the concentration measured on the platform on the assumption that when they are not on the platform they are unexposed to tunnel dust.

Table 5.8 shows the estimated average personal exposure levels for both station staff and drivers at each of the three stations/lines investigated. These data are based on the information presented in Tables 5.1 and 5.3.

Table 5.8 Estimates of average personal exposure of LUL staff to airborne particles

Station/Line	Station staff		Drivers	
	$PM_{2.5}$ ($\mu g/m^3$)	*PNC (particles/cm^3)*	*$PM_{2.5}$ ($\mu g/m^3$)*	*PNC (particles/cm^3)*
Holland Park/ Central	75	29,000	130	23,000
Hampstead/ Northern	90	14,000	200	17,000
Oxford Circus/ Victoria	170	24,000	180	22,000

The low personal particle number exposure levels for staff at Hampstead and for drivers on the Northern line probably reflect the relatively low particle number concentrations at ground level outside central London. This was supported by some additional measurements made at ground level in the vicinity of Hampstead and Oxford Circus stations. The particle number concentrations in the underground are lower than at the surface in the same location (see Table 5.2) and we expect that the exposure of station staff and drivers would be lower than if they carried out their duties on the surface. Because the PNCs are dominated by the particles from the surface rather than tunnel dust we do not believe that it is appropriate to use these data for evaluation of any possible risks from tunnel dust.

$PM_{2.5}$ exposures therefore represent the best basis to assess possible risks from tunnel dust. Although we have only monitored at three stations and in the cabs of trains on three lines we believe that these data are representative of the range of possible conditions in the system as a whole and so should provide a sensible basis for exposure estimation. However, it would be prudent to allow for the possibility of higher exposures and so we have used the highest mean exposure plus two standard deviations from the station and the cab measurements, which should provide a value towards the highest likely measurement. For drivers this would correspond to 210$\mu g/m^3$ (based on the data for drivers on Victoria line) and for station staff it is 190$\mu g/m^3$ (based on measurements on Oxford Circus station, assuming staff can spend up to 63% of their time on the platform). For practical purposes it may be appropriate to assume these are the same, i.e. approximately 200$\mu g/m^3$, based on an 8-hour average period. Averaged over 24 hours this would correspond to 67$\mu g/m^3$.

It is likely that the exposure of commuters would be less than those of the staff. We have assumed that members of the general public will be exposed to the same dust concentrations underground, although the duration of their exposure will be much less than staff. This seems a reasonable basis for assessing exposure of commuters: the dust concentration in the tunnels will be very thoroughly mixed because of the very vigorous mixing of air arising from train

movements. Spending approximately 2 hours in trains or on station platforms per day with average exposure level similar to drivers – say, at most, 200μg/m^3 – would increase 24-hour average concentration by 17μg/m^3.

6. INVESTIGATIONS OF THE BIOLOGICAL REACTIVITY OF TUNNEL DUST

6.1 BACKGROUND

6.1.1 Relevance of toxicology tests

The toxicology strategy is aimed at detecting the ability of particles to cause inflammation since inflammation generally underlies the known adverse effects of particles, for example fibrosis, emphysema and exacerbations of airways disease. It should however be noted that inflammation is primarily a protective reaction of the body, without which animals would be unable to survive attack by micro-organisms. In other words, a particle that initiates inflammation is one recognised by the body as something with the potential to cause harm if inhaled in sufficient quantity over sufficient time. In Figure 6.1 the endpoints measured are shown in Boxes in relation to the patho-biological sequence involved in the production of particle-related diseases. More details of the toxicological background to particle effects are given in Chapter 3.

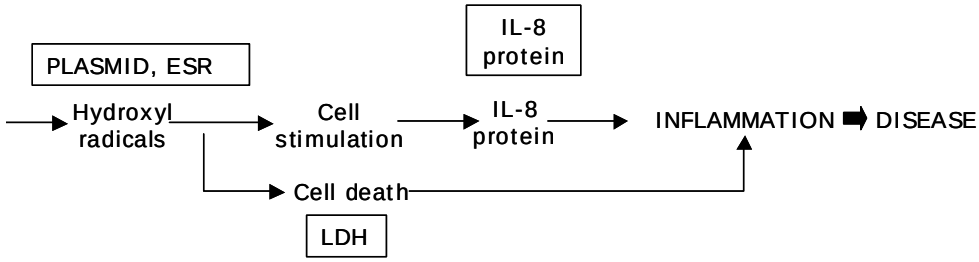

Figure 6.1 Postulated events in the initiation of inflammation by particles

Table 6.1 gives more information on the assays used and their rationale for inclusion in the toxicology study of the tunnel dusts.

Table 6.1: Details of the assays used to determine the toxicity of tunnel dust

Category	Assay	Description	Rationale for inclusion
Cell assays using the A549 lung epithelial cell line	LDH release	Lactate dehydrogenase is a cytoplasmic enzyme released only by cells that have their plasma membrane damaged.	If particles damage and kill cells then this leads to inflammation
	IL-8 release	Interleukin-8 is a chemokine released by epithelial cells that attracts inflammatory cells (PMN)	Exaggerated production of IL-8 by cells in contact with particles means that the particles would cause inflammation in the lungs
Cell-free assays to detect the reactivity of the particle surface	Electron Spin Resonance	This assay measures the production of the hydroxyl radical	Hydroxyl radicals produced by particles can initiate inflammatory responses at low levels and can damage and kill cells at higher levels
	Plasmid DNA scission	Measures the ability of free radicals from particles, principally hydroxyl radical, to cut DNA	

6.1.2 Dosimetry considerations

As noted earlier, toxicology tests in cells or in cell-free systems represent an attempt to detect toxicity using simpler and more ethical alternatives to using humans or animal exposures. These systems cannot hope to replicate the complexity of a human tissue or organ. One result of this is that, in general, far higher doses are required to get a pro-inflammatory effect in a cell test than is required in animals to get an inflammatory response – see, e.g., Appendix 2. There is no point in carrying out toxicity tests if there is no response and so doses tend to be increased until an effect is produced and this often requires high doses. This means that the tests are best at providing data on whether dusts *can*, at high enough doses, induce some response in cells. Also, they provide some useful data on the comparative toxicity of particles. They are however uninformative on the doses that cause effects in humans.

We used a computer model that calculates the amount of particles that would deposit in the lungs of someone spending 7 hours on the London Underground at the highest levels that were encountered. The dose was expressed as the total mass depositing on the centri-acinar surface and this was compared with the top 100μg/ml dose used in the cell test. In the cell test systems the dose was tens of thousands of times greater than that calculated for someone in the London Underground. It should therefore be borne in mind that the data show the tunnel dust to be more likely to cause inflammation than the control dust but these tests do not help us to know whether the levels of dust in the LU could reach the level where either the control dusts or the tunnel dust could ever actually cause inflammation in the populations exposed (i.e. workforce or travelling public).

6.2 MATERIALS AND METHODS

Note: The technical methods given here are necessary if other scientists are to validate or duplicate this work. They may be of little interest to the non-specialist, who may prefer to skip to the results and discussion in paragraph 6.3.

6.2.1 Dust Samples

$PM_{2.5}$ was sampled at Griffith House but there was only sufficient mass for the IL-8 test. We therefore used PM_{10} from Manchester that was available to us for another study and that was collected on a Tapered Element Oscillating Microbalance (TEOM) filter. The TEOM filters are manufactured from glass fibre, and in retrieving the particles from the filter some glass fibres were also harvested. Previous studies using fibres from blank TEOM filters have shown the TEOM glass fibres to be ineffective in stimulating IL-8. Other samples were collected from the station platforms as described in the previous chapter.

Particles were retrieved from the filters by sonication in either phosphate buffered saline or into dH_2O. Briefly, filters were cut into 4 pieces and each piece immersed into PBS or dH_2O in a 1.5 ml Eppendorf tube, and sonicated. The optical densities of these particles in solution, was determined spectrophotometrically at an absorbance of 340 nm and the concentration obtained from a carbon black standard curve. (Carbon black is often used as a surrogate for the carbonaceous component of air pollution particles.)

Samples sonicated in PBS were used for cell exposure, whereas those sonicated in dH_2O were used in cell-free assays: plasmid DNA assay and electron spin resonance.

Particle size data were obtained for a sample of titanium dioxide and a sample of welding fume that were used as control dusts in a number of toxicity assays. The measurements were made using the methods described in Section 5.3.2 of this report.

6.2.2 A549 Cell culture and treatments

The type II human alveolar-like epithelial (A549) cells, derived from an adenocarcinoma, were grown in Dulbecco's Modified Eagle Medium containing 10 % foetal calf serum, 2 mM glutamate, and penicillin (100 IU/ml)/streptomycin (100 μg/ml) in 5% CO_2 at 37 C. At confluency, cells were incubated for 24 hrs in 2 % fetal calf serum followed by exposure to London Underground particles (100 μg/ml), PM_{10} (100 μg/ml), TiO_2 (100 μg/ml), or DQ12 quartz for 8 hrs. Culture media from these stimulated cells were collected and analysed for IL-8 using a standard enzyme-linked immunosorbent assay. In addition, RNA was extracted from the particle-exposed cells using the TriZol reagent.

In selected experiments, the cytotoxicity of the London Underground particles in A549 cells was evaluated over a range of doses (1-100 μg/ml) for a 24 hr-period. The culture medium was then collected and the level of lactate dehydrogenase (LDH), an intracellular enzyme, was measured.

6.2.3 Detection of cytotoxicity by LDH Assay

Cell death is classically evaluated by the quantification of plasma membrane damage. LDH is a stable cytoplasmic enzyme present in all cells. It is rapidly released into the culture supernatant upon damage to the cell membrane. The LDH kit used was Roche Cytotoxicity detection kit (LDH) Cat No 1 644 793 (2000 tests); it enables the measurement of LDH activity in the culture supernatants in a 96 well plate format. The following controls were utilised

- Background control – LDH activity in assay medium;
- Low control – LDH released from untreated cells;
- High control – total lysis (1% Triton X-100).

Following incubation, the 96-well plate containing the treated cells was centrifuged at 250G for 10 mins, and 100μl of the supernatants was removed from each well, without disturbing the cell pellet, and transferred into a replicate, optically clear, flat bottomed ELISA plate. 100μl reaction mixture (per plate- 250μl solution 1 + 11.25ml solution 2, reagents provided by the manufacturer) was added to each well, mixed, incubated for 30min, and the absorbance measured at 490nm on a spectrophotometer plate reader. To determine percentage cytotoxicity, the blank (background control, media only, no cells) is subtracted from all wells and the mean absorbance for treatments determined:-

$$\text{Cytotoxicity (\%)} = \frac{\text{experimental value - low control}}{\text{high control - low control}} \times 100$$

6.2.4 Enzyme-linked immunosorbent assays (ELISA) for IL-8

Flat-bottomed 96-well microtitre plates (E.I.A/R.I.A. plate, Costar, Cambridge, MA) were coated overnight at room temperature with 100μl of IL-8 capture antibody (4μg/ml in PBS) provided by in the R&D IL-8 Duoset kit. Plates were rinsed three times with wash buffer (0.05 % Tween 20 in Tris-buffered saline, pH 7.4) and incubated for 1 hour with Blocking buffer to block non-specific binding sites. Wells were aspirated and rinsed with wash buffer (3X) and 100 μl of sample or standards was added per well. Plates were incubated for 1 hour on a plate shaker. Wells were again rinsed and 100μl of the biotinylated detection antibody (20ng/ml) was added per well and incubated on the plate shaker for 1 hour.

Following rinsing with wash buffer, 100μl of streptavidin-horse radish peroxidase was added to each well and incubated on the shaker for 20 minutes. A substrate stock of 3,3', 5,5,-tetramethylbenzidine (TMB) was prepared by dissolving 10 mg/ml in dimethylsulfoxide

(DMSO). To prepare the substrate buffer, 100µl of TMB was added per 10ml of a 100mM solution of sodium acetate/citrate, pH 4.9 with 5µl of H_2O_2 (30 %).

After washing, 100µl of substrate buffer was added to each well and allowed to incubate for 20 minutes. Plates were wrapped in aluminium foil, as TMB is light sensitive. The reaction was terminated by the addition of 100µl of 1 mM of sulphuric acid and the plate read at an optical density of 450nm in a spectrophotometric plate reader. The values were determined from a standard curve using recombinant IL-8 (R & D).

6.2.5 Electron Spin Resonance

The ability of the particles to release hydroxyl-radical was evaluated by ESR by Prof Paul Borm, University of Dussseldorf. Briefly, 250µl of particle suspension was mixed with 250µl of 0.5 M H_2O_2 in phosphate buffered saline (PBS) and 500µl of 0.05M of DMPO (Sigma-Aldrich, Taufkirchen, Germany) in distilled deionized water. The suspension was incubated for 15 min at 37 °C, and filtered through a 0.22µm pore filter (Sartorius, Göttingen, Germany). The clear filtrate was then transferred to a capillary and spin trap signal was measured with a Miniscope ESR spectrometer (Magnettech, Berlin, Germany). The ESR spectra were recorded at room temperature using the following instrumental conditions: magnetic field, 3360 G; sweep width, 100 G; scan time, 30 s; number of scans, 3; modulation amplitude, 1.975 G; receiver gain, 1000. Quantification was done by accumulation of three different spectra, each averaging three different scans. The spectra were quantified by double integration. As a negative control, a mixture of water, H_2O_2, and DMPO was used. The ESR analyses for the different particle preparations were performed in a single experiment and analyzed in duplicate

6.2.6 Plasmid assay

DNA scission assay for the detection of free radical activity: 290ng of supercoiled plasmid DNA (PsiX174) was used for each assay. Plasmid DNA was incubated with various concentrations of particles and carried out in a final volume of 20µl at 37ºC for 8 hours with shaking. Volumes, when necessary, were adjusted to 20µl using distilled H_2O. A linearised control was generated by digesting the plasmid DNA with the restriction enzyme PST1 (0.5µl) along with its corresponding buffer (1µl). Following incubation, samples were mixed with 5µl of 6X gel loading dye and electrophoresed on a 0.8 % agarose gel prepared in 1X Tris buffer EDTA (TBE) in the absence of ethidium bromide. Following electrophoresis, agarose gels were stained with ethidium bromide/TBE solution (100µl/100ml) for 30 minutes. Gels were scanned and analysed by densitometry using the UVP Grab and Gelplate program (UltraViolet Products, Limited, Cambridge, UK). Three lanes of bands are normally detected: relaxed, linear, and supercoiled DNA. The amount of free radical activity of a sample is reported as the percentage of DNA damage produced. The relaxed and linear bands of DNA represent the cleaved fractions of supercoiled DNA (damaged DNA). The PsiX174 solution contains approximately 80-90 % supercoiled DNA and 10-20% relaxed DNA. The percentage of DNA damage is calculated by adding the band intensities of the relaxed and linear forms of DNA (damaged DNA) divided by the total DNA (relaxed, linear, and supercoiled), multiplied by 100.

6.3 RESULTS

6.3.1 Particle size data

The welding fume and titanium dioxide both had smaller average diameters than the tunnel dust. Figure 6.2 shows the particle size distributions for both dusts as histograms. The median diameter of the TiO_2 was approximately 0.25µm and the corresponding value for the

welding fume was 0.15µm. There were no diameter measurements above approximately 1µm for the welding fume.

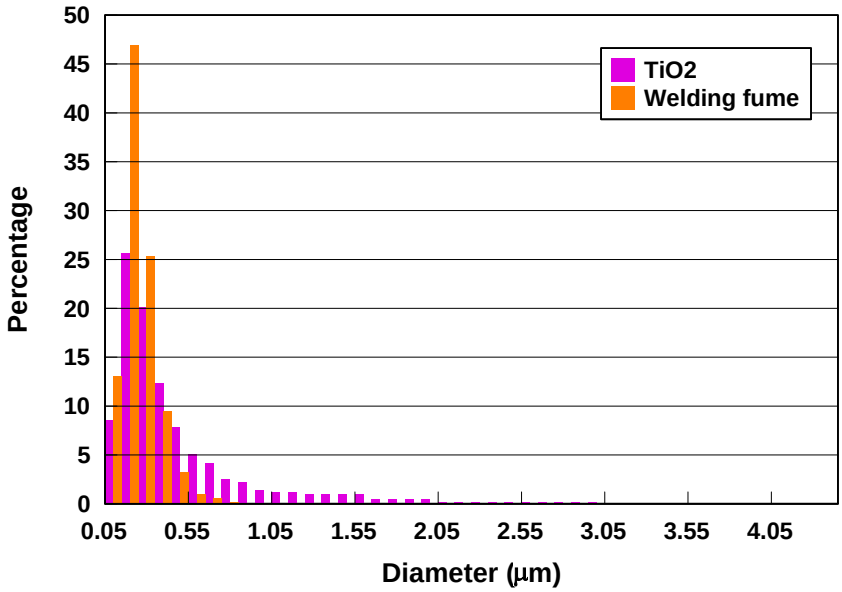

Figure 6.2 Particle size data for TiO_2 and welding fume

The size data from the tunnel dust and control dusts is compared in Figure 6.3. Here the information is presented as a cumulative distribution with the diameter plotted on a log-scale and the percentage of measurements greater than the stated size on a probability scale. In this type of plot a log-normal distribution would be represented by a straight line.

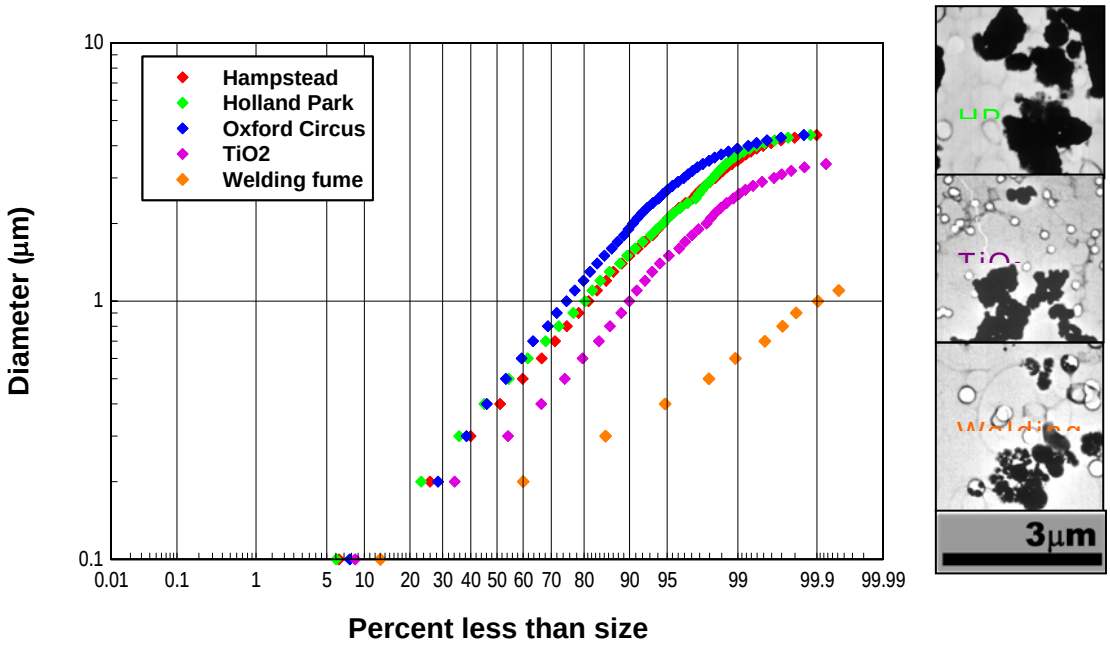

Figure 6.3 Log-probability plot comparing the tunnel dust size distributions with the data for the control dusts, plus photomicrographs

On the graph each point represents a bar on the histogram. For example, the lowest point for welding fume (orange) shows that 13% of the measured diameters were less than 0.1µm. The

next point for welding fume shows that 60% of the measurements were less than 0.2μm and so on.

Data that are lower in the graph have smaller average diameters. The data show the three tunnel dust samples with similar size distributions, the TiO_2 with slightly finer particles and the welding fume, which is much finer than all of the other dusts. The inset photomicrographs show tunnel dust from Holland Park and the two control dusts at a magnification of 10,000 times; the scale is shown at the bottom.

The data are approximately log-normal for particle sizes below about 2μm; above this the particle size distribution is progressively truncated because of the size selective nature of the airborne dust sampler used (i.e. the $PM_{2.5}$ sampler specially designed for this project).

Particles of median diameter less than 1μm are likely to be deposited in the centri-acinar region of the lung. Once deposited, they are confronted by alveolar macrophages that ingest and remove them from this region via the mucociliary escalator. The deposition and clearance of submicron particles (MD=0.7μm) in healthy and stressed individuals is demonstrated in Appendix 2.

6.3.2 Cytotoxicity

The samples used were as described in 6.2.1. Sample GH denotes a $PM_{2.5}$ sample collected in a background site near the Offices of the LU as a control. In Figure 6.4 and subsequently, the tunnel dusts are labelled as HP (Holland Park), H (Hampstead) and OC (Oxford Circus). The Figure shows that all three tunnel dusts had the ability to cause death to epithelial cells as measured by ability to induce release of LDH. There is however evidence that OC is less toxic than the other two tunnel dusts. At the highest dose, HP caused about 17% of the total releasable LDH to be freed. (This is an average figure across the whole set of experimental cells. It could mean that about 17% of cells were killed, or all cells were damaged to the extent of 17%, or some mixture of these.) The negative control TiO_2 caused about 2 % release of LDH at the highest dose and PM_{10} (from Manchester) caused about 7% LDH release at the highest dose.

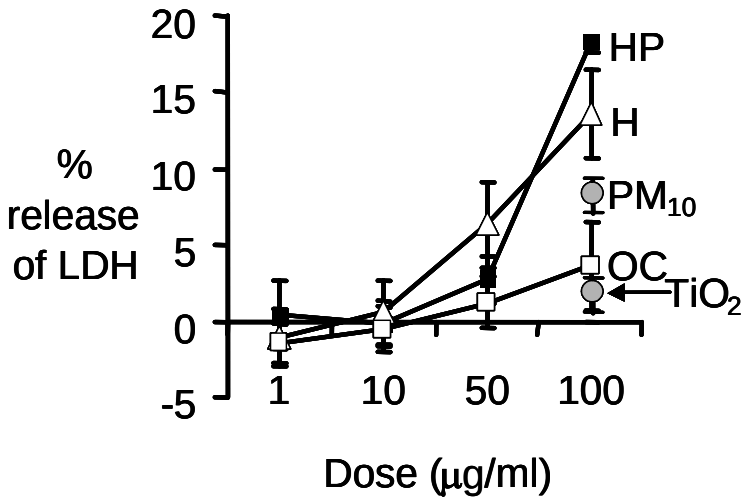

Figure 6.4 Percentage of total LDH released by cells treated with various doses of different particles. Data are mean ± SEM of triplicate wells in 3 separate experiments.

6.3.3 Interleukin-8

The release of IL-8 in response to treatment with various particles is shown in Figure 6.5. The error bars are very small and are lost within the symbols. There was a dose-dependent stimulation of IL-8 with all of the tunnel dusts. However, there is a suggestion that the OC dust is flattening out in its ability to stimulate IL-8, at the higher dose. The $PM_{2.5}$ from a control site (where GH designates a $PM_{2.5}$ sample collected at Griffith House) produced much less stimulation than the tunnel dusts at the highest dose and TiO_2 was the least active in causing IL-8 release at the highest dose.

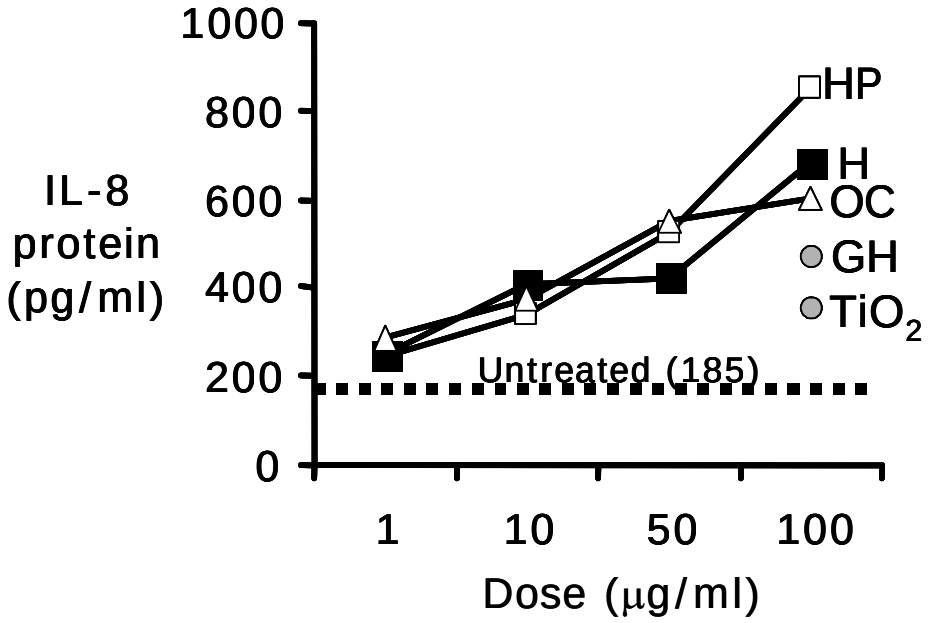

Figure 6.5 IL-8 release induced by various doses of the different particles. Data presented are mean of triplicate wells in 3 separate experiments; SEM omitted for clarity but were <10% of the mean

6.3.4 Role of soluble transition metals in the IL-8- releasing activity of the tunnel dust

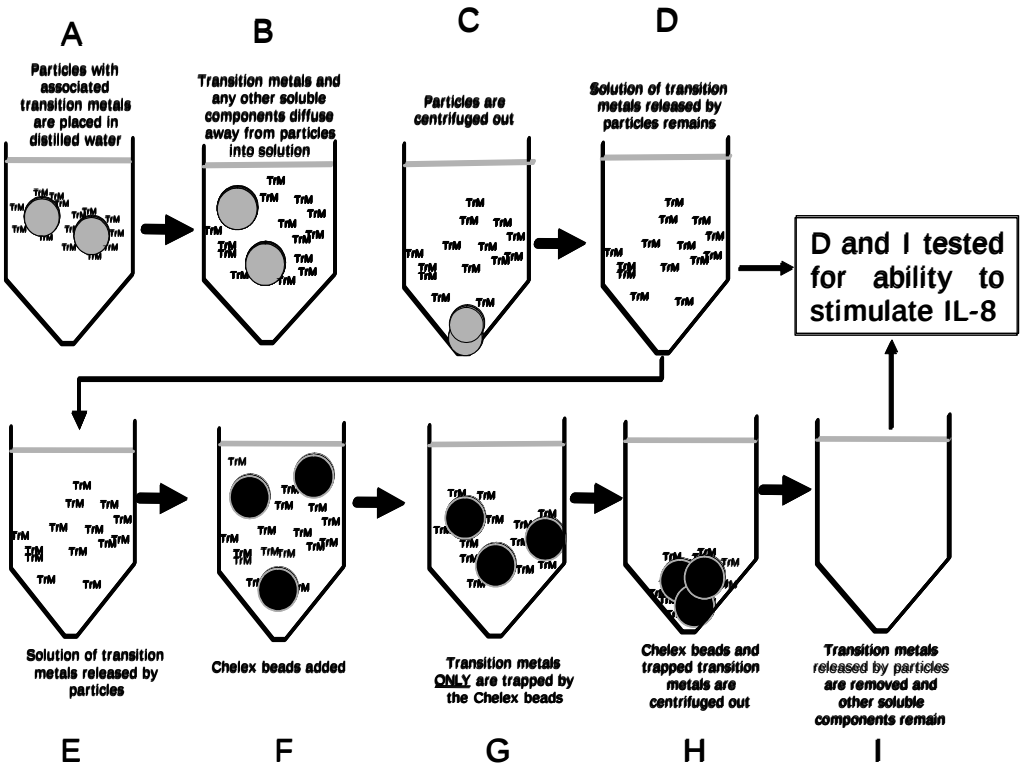

Figure 6.6 Strategy for testing the role of transition metals

The high iron content of the tunnel dusts places it in a similar category to welding fume, another particle we have worked with. Based on a paradigm that has evolved from our welding fume studies, we tested the ability of soluble components of the tunnel dust, especially transition metals, to stimulate IL-8 release. The method used to determine the role of transition metals is shown in Figure 6.6. The main aim was to collect the soluble material from the particles (see top line in Figure 6.6), based on the assumption that iron, a transition metal, would be the most abundant toxic component released by the particles. We then tested this soluble extract (D in Figure 6.6) for its ability to stimulate IL-8 release – comparison of D and I critically tests the role of soluble transition metals in the ability to stimulate IL-8 release.

However, other soluble components could have been released and to specifically test the role of transition metals, we used chelex beads to inactivate transition metals (see bottom line in Figure 6.6); these beads bind transition metals and can then be spun out. Thus the transition metals have been selectively removed, whilst any other soluble components remain. The chelex-treated soluble components from the particles, I in Figure 6.6, were then tested for ability to release IL-8 in comparison with D and thereby the role of transition metal was critically tested.

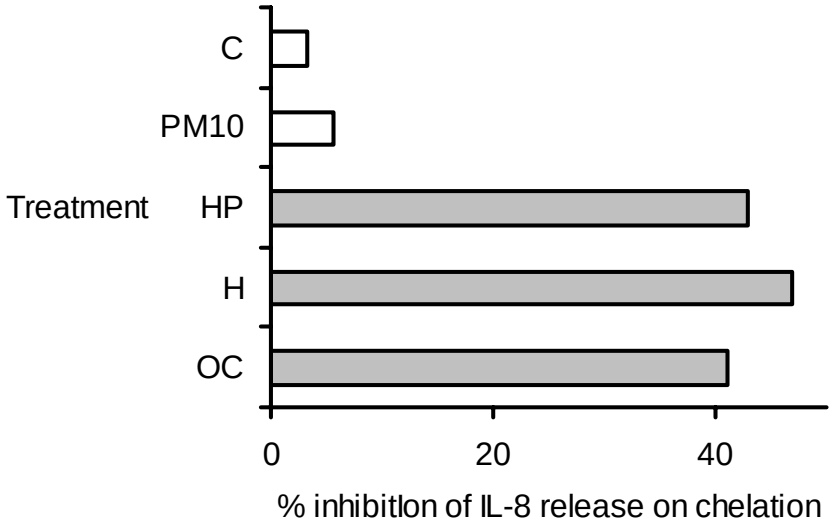

Figure 6.7 Percentage inhibition of IL-release by treatment of soluble components with chelex beads. Referring to Figure 4, the % inhibition is calculated as 100-(IL-8 release by I/IL-8 release by D) x 100. Data is the mean of triplicate wells in a single experiment.

The control levels were rather higher than in the experiments reported in Figure 6.5, and the amount of stimulation by the soluble components (D in Figure 6.6) was between 400 and 500pg/ml – about half of that produced by the whole particles (Particles + soluble components). The production of IL-8 by the soluble components of tunnel dust washing could be dramatically lowered, to almost half, by removal of the transition metals by chelex beads (Figure 6.7). This compares with similar dramatic reduction in the IL-8 releasing activity of the soluble components of welding fume when they are treated with chelex beads. Chelation had little effect on control medium or soluble components of PM_{10} (Figure 6.7). These results imply that much of the potential of the underground dust, as that of the welding fume, to initiate inflammation resides in ionic iron on the surface of the particles.

6.3.5 Plasmid assay

The plasmid assay revealed the tunnel dusts to have considerable free radical activity. The TiO_2 and PM_{10} London controls had activity that was no different from controls.

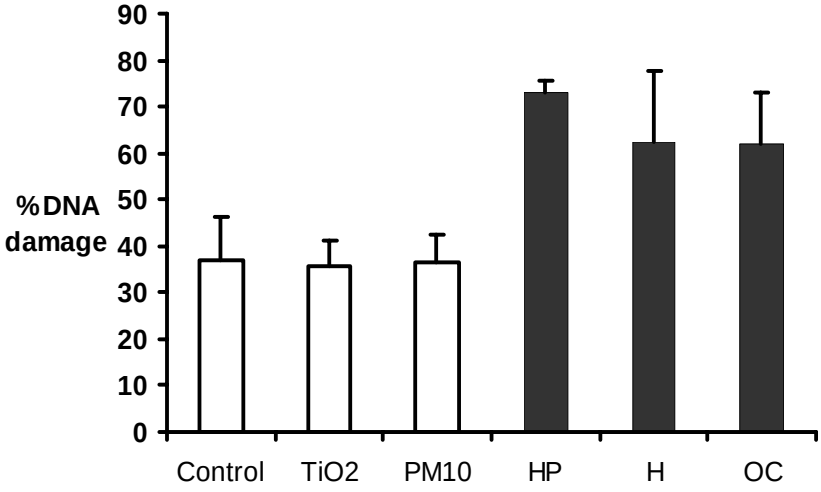

Figure 6.8 Amount of plasmid DNA damage caused by various particle types. Data are mean of duplicate lanes in 3 separate experiments.

6.3.6 Electron spin resonance (ESR)

Electron spin resonance data showed a surprising variability amongst the different tunnel dusts but all were substantially more able to produce hydroxyl radical than TiO_2.

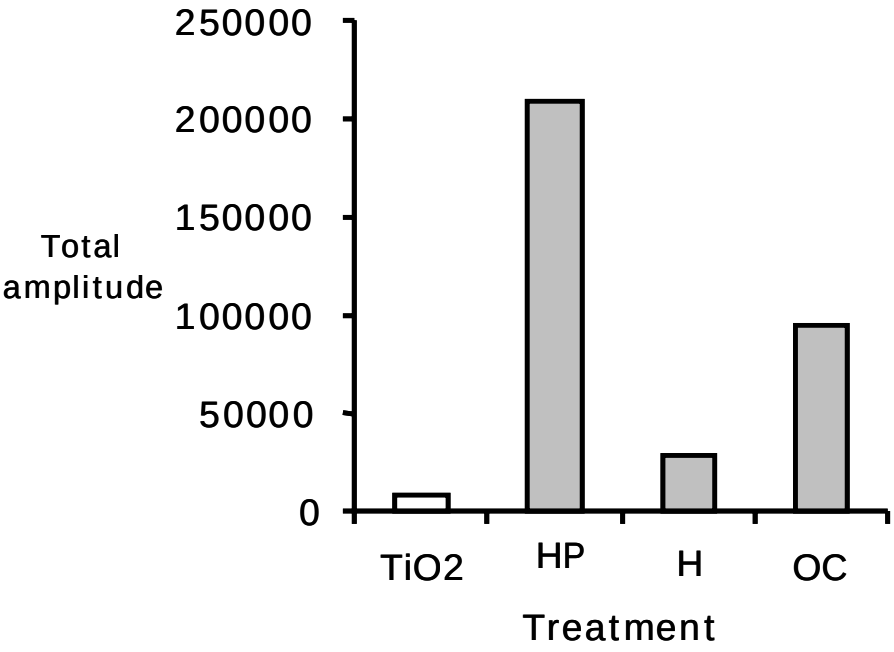

Figure 6.9 ESR signal for hydroxyl radical produced by tunnel dusts and TiO_2. Data are mean of duplicates in a single experiment

6.3.7 Benchmarking to other particles

In order to place the toxicity of the tunnel dusts in context we have chosen quartz and welding fume data available from recent and ongoing studies for comparison. Figures 6.10 and 6.11 show the data obtained in the present study for tunnel dust (the average of the 3 stations is shown) and TiO_2 plus data from other studies for quartz and welding fume. The quartz data was obtained by Miss Claire Monteiller (IL-8) and Miss Kirsty Sherriffs (LDH) whilst the LDH and IL-8 data for welding fume (average of 3 different welding fumes) were kindly supplied by Miss Jane McNeilly.

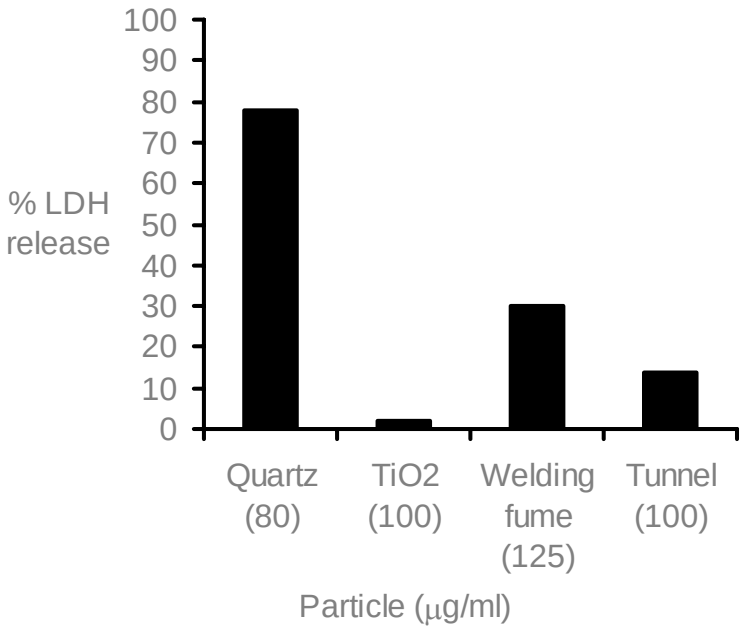

Figure 6.10 Percentage release of LDH following treatment with the indicated doses of different particles. See text for explanation of these data.

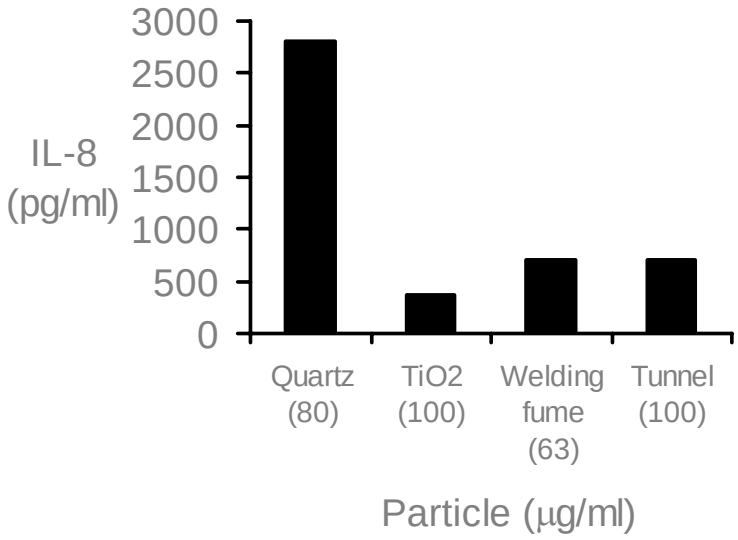

Figure 6.11 Release of IL-8 following treatment with the indicated doses of different particles. See text for explanation of these data.

None of these historic data used 100μg/ml and so the closest dose used (80 or 125μg/ml) is shown; all data represent the mean from three separate experiments. Figures 6.10 and 6.11 show that quartz and TiO_2 do indeed show effects at the extreme ends of the toxicity spectrum, with quartz being highly lethal whilst also able to cause stimulate on of IL-8 before death ensued, and TiO_2 being low in activity. The data also show that tunnel dust and welding fume in the two assays are (a) similar in activity (b) much closer to TiO_2 than to quartz in activity. Although these data do not come from experiments carried out for this study, they have been obtained in experiments carried out within our own laboratory within the last 6

months. Therefore although there could be some variation between experiments, we are confident that similar data would be obtained if all four particle types were tested side-by-side in experiments.

7. OVERVIEW DISCUSSION AND CONCLUSIONS

7.1 WHAT HAVE WE DONE?

Our aim has been to give an informed assessment of the risks to the health of workers of long-term exposure underground to tunnel dust and, as far as practicable, to supplement this with some comments on any risks to the health of the travelling public also. To do this, we have carried out and reported a detailed and careful study of tunnel dust in the London Underground rail system, from the viewpoint of its possible effects on health. The study has included several linked components.

a. A review of what was already known about tunnel dust, insofar as is relevant to its potential to damage health;
b. A new assessment of those characteristics of the dust which are understood to affect its likely toxicity – size distribution and composition, with special reference to the dust's iron content. This assessment was informed by a new programme of measurements, involving particle size data and compositional analysis, as well as measurements of PM_{10} and $PM_{2.5}$ on stations and in the cabs of trains;
c. New measurements of personal exposures of train drivers and estimates of the likely exposures of those working in stations. We have also extrapolated from these results to estimates of the exposures of the travelling public who spend lesser amounts of time in the system than do the workers.
d. New measurements from *in vitro* studies that allow us to comment on the toxicity of the dust, i.e. its ability to cause inflammation, and so enable us to make some tentative extrapolations regarding its ability to cause inflammation in people;
e. A limited review of who is exposed to tunnel dust underground, from the viewpoint of their likely susceptibility to dust-related diseases.

For ease of understanding by the many interested people who are not specialists on dust and health – members of the management and workforce of London Underground and also the wider travelling public – we have framed our discussion in terms of answers to a series of questions.

7.2 WHAT HAVE WE FOUND?

In summary, we have found the following.

7.2.1 Nature of tunnel dust

Tunnel dust is very different from outdoor ambient PM in terms of its composition, size distribution and the concentrations to which people are exposed. This has important implications for its risks to health.

i. ***Composition of tunnel dust – the role of iron and silica:*** The dust to which people are exposed in the Underground comprises around 90% iron (which will be largely in the form of ferric oxide), with only trivial amounts (c1%) of quartz and even less of other metals. This means that the possible toxicity of the dust is likely to be that associated with the inhalation of iron oxide. ***Implication:*** In particular, workers (or the public) are not at risk of silicosis.

ii. ***Size distribution of tunnel dust:*** The size distribution of the dust differs substantially from that of ambient PM_{10}, in that it contains relatively smaller numbers of ultrafine particles (derived from above ground) but larger numbers of particles ranging from 0.2μm upwards. Many of the smaller underground dusts will have a higher

aerodynamic diameter than that measured directly by electron microscopy. ***Implication:*** All the particles that we have measured have the potential to penetrate into the lung acinus. However, a higher proportion of those above ground than of the underground dust reaching that level will be deposited there and pass through into the interstitial space. In our view, this is likely to have important implications with respect to potential toxicity.

iii. ***Concentrations of tunnel dust underground:*** Our study has confirmed that, measured in mass terms (i.e. in units of $\mu g/m^3$), dust concentrations underground are substantially higher than above ground. On the other hand, in terms of particle number, dust concentrations underground are lower than above ground at the same locations in London. ***Implication:*** People while underground are therefore exposed to fewer ultrafine particles, and fewer particles overall, but more large particles and a substantially greater total mass of particles than someone in the general environment above ground. This greater total mass is a consequence both of larger and more dense particles underground.

7.2.2 Toxicity: the potential to cause inflammation

i. *There was evidence of some toxicity from cell studies:* Tunnel dust showed evidence of some toxicity in laboratory cell studies, with very large doses being able to provoke cells to release substances that could cause inflammation in the lung.

ii. *There were supporting indications, from the non-cellular studies, that the dust could be toxic:* There was coherence between the results obtained with the different assays, with the free radical chemical assays (non-cellular tests) supporting the findings in the cell assays.

iii. *Comparisons with other workplace dusts:* This toxicity is much less than that of quartz but greater than that of the non-toxic dust titanium dioxide when given at comparable doses. It is roughly comparable to that of welding fume.

iv. *Comparisons with ambient PM:* Tunnel dust was slightly greater in activity than the PM_{10} samples that were available. However, PM_{10} in different locations and on different days varies markedly in its toxicity; the comparison should be treated cautiously.

v. *Chelation of tunnel dust* (i.e. removing metal ions from 'coating' the surface of the dust), a process that occurs naturally in the lungs of living people, reduced its toxicity to about one-half that of unchelated dust.

vi. *Welding fume as a benchmark dust:* The tunnel dusts were similar to welding fume in terms of their high iron content and the role played by the transition metals in the stimulation of IL-8 release. Overall, tunnel dust was closely similar to welding fume in term of toxic potency.

7.2.3 Exposures of workers and of the travelling public

Exposures of workers are low compared with relevant occupational standards. Concentrations, in $\mu g/m^3$, are substantially higher than those for PM_{10} or $PM_{2.5}$ above ground, and would be a cause for concern if the risks were similar to those of the general public from PM_{10} or $PM_{2.5}$. However (see later – Sections 7.4 and 7.9) we strongly believe that this is not the case.

i. In our opinion, any toxicity of the dust should be related principally to the fine particle component, and should take account of particle number as well as mass. In the present study, the fine particle component is measured in mass terms as $PM_{2.5}$.

ii. Exposures of drivers are of the order of 100-200μg/m^3 $PM_{2.5}$ or 17-22,000 particles/cm^3 over a shift. The exposures of station staff depend on the length of time spent on the platforms, but would range from about 50 to 400μg/m^3 or 10-20,000 particles/cm^3.

iii. The exposures of workers and of the travelling public underground to ultrafine particles are lower than above ground.

iv. Likely exposures of both drivers and station staff to $PM_{2.5}$ will be of the order of 200μg/m^3 over a typical shift. This is much lower than workplace standards for relatively low toxicity occupational dusts, and much higher than (more than 10 times as high as) the mass concentrations of ambient $PM_{2.5}$ above ground.

v. We estimate that, in mass terms, the exposure of a member of the public from a typical journey is in the region of 15-20 μg/m^3 $PM_{2.5}$ averaged over 24hrs, i.e. comparable to the ambient exposure to the very different above ground $PM_{2.5}$ for a full day on the surface in central London.

7.2.4 Susceptibility of the exposed populations

Our investigations were very limited. However, workers are in general much less susceptible than the general population (which includes the very young, the very old and the very ill) and, in terms of its age distribution, the travelling public was much more similar to a workforce than to the general wider population. This does not mean that such potentially more susceptible people do not travel in the Underground but that any weakly adverse health effects would be relatively rare and more difficult to detect than above ground in the general population.

7.3 HOW RELIABLE ARE THE BIOLOGICAL REACTIVITY TESTS AND WHAT DO WE MAKE OF THE RESULTS?

Before proceeding to draw conclusions about the risks to health from exposure to tunnel dust, we will discuss some aspects more fully, notably

- the biological reactivity tests;
- the role of iron; and
- what may be the most relevant of the available well-studied (or 'exemplar') dusts with which to compare tunnel dust in terms of its risks to health.

7.3.1 What the single cell line tests can (and cannot) tell us

As noted earlier, we carried out two kinds of tests. Some of these tests investigated how lung epithelial cells react to various dusts, including tunnel dust. Others were studies of those chemical characteristics of the dusts which are understood to influence biological reactivity.

Inflammation in context: Both kinds of tests examine the potential of dusts to cause inflammation. Again as noted earlier, however, this is but one of several factors – all of them important – which influence the risks to health of exposure to tunnel dust. The other factors, noted above, are (i) the size and composition of the dust – this influences not only *whether* inflammation may occur, but also *where*; (ii) the dose that the cells experience which is related to exposure levels and (iii) the susceptibility of the exposed population.

Reliability of the tests in identifying potential to cause inflammation: In terms of ability to cause inflammation, note that effects in the whole lung or animal can be predicted only in very general terms from these cellular and non-cellular tests. These tests were developed originally to investigate and identify mechanisms of disease rather than to make quantitative comparisons of toxicity across dusts of different kinds. Their usefulness for quantitative purposes is still not properly established or agreed. This is principally because these non-cellular or single cell line experiments study in isolation a part of a process which, in the living person, is far more complex. Even a series of *in vitro* tests cannot reproduce or simulate adequately, in quantitative terms, how the lung as a whole (or the human body as a whole) will respond to tunnel or other dusts. For example, part of the lung's response is to attempt to bind and remove (chelate) inhaled metals, including iron. This is something that cannot be done 'spontaneously' in single cell line tests. However, it was possible, in the investigations carried out, to study chelated dusts in the single cell tests.

Also, extraordinarily high doses are needed before any effects can be identified reliably in the cell tests. We have calculated the doses used in the toxicological tests in relation to those that would reach the lung acinus in people exposed underground – see Appendix 2. Depending on the level of activity of the individual, the concentration given to cells in these studies was between 15,000 and 20,000 times that which the relevant parts of the lungs of an underground worker would receive over a shift. It is unclear (because it is impossible to study) whether or not the same relative potencies exist between the different dusts at 'ordinary' concentrations. We think that the most reasonable assumption is that they do, but we note that this is an assumption and so, in our overall evaluation, we put emphasis, where possible, on results of studies in humans.

On the other hand, there is a consistency in the results of the experiments we have carried out. *Within* individual experiments, there is, for the various dusts, a clear relationship between dose and the magnitude of response, once the threshold for initiating response has been exceeded. And, as noted earlier, there is a broad consistency of findings, *across* the various tests used, regarding the relative potency of the several dusts studied. So it is important to respect the thrust of the findings while ensuring that they are interpreted in context.

7.3.2 Cell assay results

The data suggest that the tunnel dusts in these high concentrations have the ability to generate free radicals and cause cell damage and release of IL-8, properties that might cause them to produce inflammation in the lungs. The potency of the tunnel dusts in this regard, on a mass basis, was greater than that of TiO_2. TiO_2 serves as a useful benchmark dust because it has very weak if any ability to cause lung injury. The most active of the tunnel dusts had 4 to 10 times more activity than TiO_2 in the cell assays.

The tunnel dusts were also more active than PM_{10} from an urban environment. There was only sufficient of the Griffith House (GH) sample for use in the IL-8 assay, and therefore PM_{10} from other sources had to be used as controls in the other assays. In terms of IL-8 release and cytotoxicity the tunnel dusts were 2-3 times more potent than the GH or the PM_{10} samples. PM_{10} is very variable in composition because of variation in season, weather, and in the sources from day to day. This gives rise to considerable variability in the composition and size of the PM_{10} in any sample, as demonstrated in a study, funded by the Department of the Environment, Food and Rural Affairs (DEFRA) that is currently nearing completion. That study collected PM_{10} from various sites across the UK and tested their ability to cause inflammation. The ability of individual samples was very variable, with some samples causing no inflammation and other samples causing substantial inflammation. This variability in PM_{10} is described in a paper to be published in the Proceedings of a Conference in Germany in the summer of 2003 (Stone *et al*, in press).

Only two PM_{10} samples were used in the present study, and we know nothing of their composition, but they showed limited evidence of pro-inflammatory effects. This is consistent with our findings that some PM_{10} samples are less inflammogenic than others. The Griffith House sample was probably a 'low toxicity' PM sample collected on a day when the secondary component of PM_{10} – the salts that are known to be of low toxicity – dominated. The comparison with TiO_2 in our opinion therefore represents a more stable benchmark against which to judge the toxic potency of the tunnel dusts. TiO_2 has been used extensively in particle toxicology studies over the last 30 years and has consistently been shown to be of low toxicity – often having no effect on cells at low doses. The sample used here (Tioxide TiO_2) has been used extensively for this purpose and has always proved to be of negligible toxicity. The only two cases where TiO_2 has been shown to produce toxic effects are (i) when it is in the ultrafine form and (ii) when it is delivered to rats at such high doses that it causes rat lung overload. Neither of these exceptions is relevant here and we believe that the TiO_2 control is a good negative control.

When a soluble fraction of the tunnel dust was prepared, it was found to be stimulatory of IL-8 release, and chelation of this solution to remove transition metals produced an inhibition to below control levels. The cell assays therefore show

- a consistent trend for increased activity of tunnel dust over TiO_2 and a similar or slightly greater activity of tunnel dust than the PM_{10} samples used here
- evidence that the IL-8–releasing activity of tunnel dust is due in large part to the soluble transition metal component, an effect we have documented for welding fume, another workplace dust with high iron content.

7.3.3 Assays of free radical activity

These assays are chemical and do not involve cells. Both assays showed greater chemical activity in production of free radicals, principally hydroxyl radical, in the tunnel dusts than in TiO_2 or PM_{10}. These assays measure only purely chemical effects of the dusts in the absence of cells and so could show the kinds of reactions that might occur in the lung lining fluid. The greater ability of the tunnel dusts to generate free radicals means that they are likely to cause oxidative stress in the lungs. However, it should be noted that the intact lung has effective antioxidant properties as well as chelators to bind the iron that gives rise to the oxidative stress.

7.4 IS IT FAIR AND REALISTIC TO USE THE GENERAL PUBLIC HEALTH RISKS OF AMBIENT PM_{10} TO 'BENCHMARK' THE RISKS FROM TUNNEL DUST?

We are dealing here with two related questions: Are the dust exposures of people underground 'high'? And, are the associated risks to health in some sense also 'high'?

Our strategy in answering these questions has been to 'benchmark' tunnel dust by reference to other, well-researched ('exemplar') dusts; and thereby to evaluate indirectly (but, we hope, reliably) the risks to health from tunnel dust. In doing this, it is important as far as practicable to compare like with like. So, just as a dose of something taken by mouth will have very different effects, depending on what it is – trivially, a food or a poison – so an inhaled dose should be considered as 'high' only in relation to what is inhaled, who is inhaling it, and knowledge of the effects of that substance on the body.

As noted in Chapter 1, these questions are sometimes answered in terms of concentrations and standards for ambient particles. At the start of our study we were sceptical about this comparison; it is timely to re-visit it, in the light of our results.

PM_{10}, or similar measures of ambient pollution, are widely believed to be responsible for thousands of premature deaths in cities of developed countries each year, and it has been suggested that some of this may be due to ionic iron on the surface of the particles. This has led to the suggestion that other particles containing iron must be equally dangerous. However, there is a difference in the way in which iron reacts with the lung cells depending on its form. The particles we have studied comprise largely unavailable iron oxide. A tiny proportion of the iron, on the surface of particles, may be in ionised form. It is only this latter, ionic, iron that takes part in the chemical reactions that may in some cases lead to inflammation. The dangers of PM_{10} need also to be put into perspective. It is actually very difficult to show that PM_{10} can cause serious adverse effects in people, requiring studies involving hundreds of thousands of individuals to show excess death rates. This is because it is actually of very low toxicity and is inhaled in low concentrations, and so the risks are largely confined to people who already suffer possibly serious heart and/or lung disease, that is, a generally vulnerable and elderly group.

Ambient dust concentrations are most often measured in mass terms (e.g. as $\mu g/m^3$ of PM_{10} or $PM_{2.5}$); and ambient dust standards are set in mass terms also. Our study has confirmed that the concentrations of dust inhaled by people in the Underground are markedly higher in mass terms than those inhaled by the same people above ground, and in the limited *in vitro* biological tests we carried out, tunnel dust was roughly similar to, or slightly greater in activity than, the ambient PM_{10} samples that were available.

Superficially, then, it might seem that PM_{10} or $PM_{2.5}$ are indeed appropriate surrogates for tunnel dust; that the many studies of ambient PM could be used to estimate the risks of tunnel dust; and that concentrations of tunnel dust underground should be assessed as 'high' relative to ambient concentrations of, or standards for, PM_{10} or $PM_{2.5}$.

We do not agree; and there are many reasons why we think that such a comparison is inappropriate and likely to mislead.

a. The dusts inhaled are very different in the two situations. Above ground, the dust comprises a complex mixture of predominantly very small (less than 0.1μm) particles made of carbon and salts of ammonium with trace amounts of metals and carcinogens – a consequence, principally, of combustion. Underground, the dust comprises almost entirely iron oxide in larger particles, mostly greater than 0.2μm – a consequence, principally, of abrasion. Our results have confirmed these differences. On general grounds, we would not expect their effects to be similar.
b. Evidence of similarity comes principally from the results of the *in vitro* tests we have carried out. However, as noted above, these tests are somewhat blunt instruments for assessing the relative potency of dusts to cause inflammation. We think that they are reliable in differentiating between a test dust and, respectively, a highly toxic dust such as quartz, or an apparently inert dust such as TiO_2. Their power to differentiate between dusts of low to moderate toxicity is less clear. In the present study, there is the added complication that emerging evidence shows large variations in the potency of ambient PM to cause inflammation and we do not know where, in that spectrum, the samples we analysed lie.
c. Some of the results from the sampling show clearly the differences in particle number and in mass concentration between the two kinds of dusts. In particular, the dust concentrations underground are much lower in terms of particle numbers than ambient PM – it is a relatively much coarser dust. This implies that tunnel dust is much less likely to be deposited in the acinar region of the lung and, perhaps more importantly, pass beyond there to the interstitial part of the lung where inflammation is believed to be more dangerous than it is in the airways themselves.
d. Using risks from ambient PM to 'benchmark' the risks of tunnel dust implies not only a comparison of exposures, but also a similarity in the population-at-risk and we have

noted (Section 1.5) that both the workforce, and the travelling public, differ markedly from the general population in terms of their likely susceptibility to inhaled dusts.

We remain, therefore, of the opinion that the risks to health of the general population from exposure to ambient PM, and the associated outdoor dust standards, are not a good guide to the risks of tunnel dust or to the standards that need to be maintained underground. Similarly, while it is likely that those who are old, very young, or have cardio-respiratory disease are also more susceptible to tunnel dust, the extent of that susceptibility cannot be established reliably by virtue of their susceptibility to ambient PM.

7.5 IS THERE A BETTER WAY OF 'BENCHMARKING' THE RISKS FROM LONG-TERM EXPOSURE TO TUNNEL DUST?

In our view, the best comparison to use in estimating and comparing the risks to health of tunnel dust is with industrial workers exposed to iron-rich dust (iron oxide) of roughly the same size range. This is because:

a. The dusts are similar in terms of size *and* metal (iron) content *and* (we think) in the bioavailability of that iron to cause damage in the lung, implying that the lung will respond in similar ways to the two kinds of dust;
b. This is supported by the limited biological tests carried out, which suggest that the dusts are similarly active on epithelial lung cells – though see above regarding limitations of those tests;
c. Our primary interest in the present study is the health of workers exposed long-term to tunnel dusts and, although study selection effects can vary between workers in different industries (e.g. the London Underground and other workers exposed to iron oxide), a comparison with risks in another working population is much more appropriate than a comparison with the general public;
d. The selected nature of the travelling public underground, compared with the general public, implies that a workforce comparison may also be better in estimating risks to the travelling public.

We therefore think that the best way to estimate the importance in health terms of exposures to tunnel dust, certainly to workers and probably also to the travelling public, is by reference to any known effects in workers exposed to iron oxide dust of roughly the same size range as tunnel dust.

7.6 WHAT HARM MAY COME FROM INHALING IRON?

7.6.1 Evidence from studies of groups of exposed workers

Iron exists in nature largely in the form of trivalent ferric iron, and this is the form found in steel and in dust in the Underground, where it is combined with oxygen as ferric oxide, Fe_2O_3. This red substance is familiar as rust and is used (as "jewellers' rouge") in the rather uncommon trade of silver polishing. It is also the form of iron mined in the UK as haematite and inhaled by welders working on mild steel and by iron foundry workers.

The rather rare medical condition known to be associated with inhalation of iron oxide in these trades is called siderosis. It is characterised by the accumulation of iron oxide in cells in the lung with no or slight fibrotic reaction, and is not associated with adverse effects on lung function. Since iron is radio-dense, the iron deposits may show up on chest radiographs. After exposure ceases, the radiological abnormalities regress.

Studies of these workers thus indicate that accumulation of iron in the lungs does not cause progressive disease such as may occur in workers exposed, for example, to coal or quartz. A progressive condition of lung fibrosis has been described in haematite miners and in foundry workers, but this is known to be due to the concurrent inhalation of quartz, and pathologically is distinguishable from silicosis only by the red staining of the tissue in the haematite miner's lung.

There is no useful information on the concentrations of iron that led to the development of siderosis in haematite miners or silver polishers. However, there is much more information on the exposures of welders. A typical mild steel welding operation will generate a fume containing several milligrams of iron oxide. Over a shift a welder may be exposed to between

1000 and 5000μg/m^3 for 4-6 hours, depending on the materials used, leading to inhalation of between 10 and 50mg of iron oxide. Someone involved in iron foundry work may similarly be exposed to between 1000 and 10,000μg/m^3. The higher levels of exposure might be expected to lead to radiological siderosis if prolonged over decades.

7.6.2 The harmfulness of inhaled iron depends on its bioavailability

There is an apparent contradiction between the relative harmlessness of iron when inhaled in these trades and the literature on the pathogenesis of pneumoconiosis. In the latter, there is evidence that ionic iron adsorbed onto the surface of particles of, say, asbestos or coal, may be responsible for the initiation of the inflammatory reaction that leads ultimately to fibrosis of the lung and possibly even to cancer. This is based on experimental studies in which removal of the iron from the particles reduces or eliminates their toxicity. It has also been suggested, coincidentally by one of the authors of this report (KD), that ionic iron may be responsible for some or all of the observed toxicity of ambient urban particles. This suggestion is based on similar experimental observations to those we report here, but using PM_{10}, in which removal (chelation) of the iron from particles reduces or removes their toxicity to cells.

The ability of iron to cause such tissue damaging reactions depends on its ability to exist in two forms, ferrous and ferric. As stated above, the iron taken into the lungs is in the ferric form and this is essentially non-toxic. It may become toxic when converted to the ferrous form by the action of antioxidants, some of which are present naturally in the lung. The experiments that we report here have shown that, in relatively very high doses indeed compared to those received by the lungs of Underground workers, the dust does have a potential to cause inflammation and thus, by inference, fibrosis and other endpoints like exacerbations of asthma, COPD and cardiovascular effects. It is likely that this is due to the release of free radicals, as shown in our studies of other dusts (e.g. Donaldson and Tran, 2002). This would be in keeping with the pathological findings, in some welders, of small amounts of fibrous tissue in the lungs in association with deposits of iron. But it is also in keeping with the absence of evidence of serious pneumoconiosis or of impairment of the function of the lungs occurring in such people, since the doses required to show effects in the laboratory studies are very high (>15,000 times those received by human lungs at work – Appendix 2).

An important difference between iron in PM_{10} and iron in the Underground is that in PM_{10} the iron is in ionised form and in association with ultrafine particles which can also generate free radicals at their surface by a non-transition metal mechanism that is not understood (Brown *et al*., 2000). In ambient PM_{10} these ultrafine particles, possibly along with iron, can penetrate through the epithelial lining layers of the lung into the interstitial space. That is, they become internalised into the body where they are available for biochemical reactions that may theoretically lead to heart or lung disease. The larger particles characteristic of Underground exposures, in contrast, would normally be taken up by the alveolar defensive cells, the

macrophages, and removed from the lung. Only with a very heavy overload of particles, such as may occur in some welders or heavily dust-exposed workers, would iron accumulate in the alveoli of the lung and cause radiological changes. This reasoning is consistent with the absence of any reported observation of radiological changes having occurred in Underground workers over many years of operation of such systems in many countries.

Finally, it is necessary to discuss the possibility of increased risk of pneumonia. This has been shown in welders and other workers exposed to iron fume. There are theoretical reasons to believe that this effect could be contributed to by the iron itself as well as by the fact that it is introduced into the lung in ultrafine particulate form. If this is so, it is not possible to exclude with confidence from the study reported here the possibility that those underground workers with the highest exposures to iron may run an increased risk of the disease. This is likely to be a smaller risk than in welders, both on account of the lower dose received by underground workers – discussed in the following Section – and also because welders are generally exposed to smaller particles.

7.7 COMPARED WITH WORKERS' EXPOSURES TO IRON OXIDE DUST, ARE THE TUNNEL DUST EXPOSURES HIGH?

Relatively few groups, but large numbers, of workers are exposed to iron oxide. These may be separated into two:

i. those in whom the iron inhaled is in relatively large particles such as iron mining, grinding and polishing, and
ii. those in whom it is a fume (by definition consisting largely of ultrafine particles), such as welding and burning.

London Underground exposures are most closely comparable to those occurring in the former category. However, it is necessary to include a caveat. Most iron-exposed industrial workers are exposed to a more complex mixture of particles than are Underground workers. For example, iron miners may be exposed also to high concentrations of quartz and radioactive gases, and this concomitant exposure to other pollutants explains the increased risk of lung cancer and silicosis described in some such groups of workers. Welders, apart from being exposed to very high concentrations of ultrafine particles, may also be exposed to significant concentrations of asbestos, nickel, chromium and polycyclic aromatic hydrocarbons, all of which may cause lung cancer.

Taking account of some of these factors, the UK Health and Safety Executive has set an Occupational Exposure Standard (OES) for iron, as a fume of ferric oxide, of 5000μg/m^3 (5mg/m^3). This means that, in the view of the HSE, workers could be exposed to such a level over an 8-hour shift on a regular basis without developing disease. In the words of the HSE, "An OES is set at a level at which (based on current scientific knowledge) there is no indication of risk to the health of workers exposed by inhalation day after day". The HSE has not considered that iron is sufficiently toxic to set the more stringent standard, a Maximum Exposure Limit, which applies to substances with the most serious health hazards and for which "safe levels of exposure cannot be determined".

Thus, inhaled iron oxide is regarded in industry as relatively less toxic than many other inhaled substances and, on the basis of the OES for iron oxide that the HSE maintains, the exposures of workers to tunnel dust underground are not high.

It appears that in practice also, the exposures of London Underground workers to tunnel dust are lower, in mass terms, than the exposure to fume of welders. Data on the exposures of welders have been summarised by IARC (1990); our examples are from stainless steel welding. In the Dutch study by van der Wal (1985), the exposure to total fume in various

processes (manual metal arc; metal inert gas, etc.) ranged from 600 to 40,000μg/m^3; background levels ranged from 500 to 1,200μg/m^3. Froats and Mason (1986) reported concentrations in the range 300 to 21,600μg/m^3 in the breathing zone of groups of welders in Canada. Results from other studies listed in IARC (1990), while not directly comparable, also suggest that the exposure to fume of the welders studied was clearly higher than that of the workers underground. Although corresponding data are not presented for numbers of particles, we can infer that the contrast is far greater for particle number, because welding fume particles are on average far smaller than tunnel dust.

7.8 WHAT EFFECTS MIGHT BE EXPECTED IN TRAIN DRIVERS AND STATION WORKERS?

Our results suggest that London Underground workers may be exposed to dust concentrations up about 200μg/m^3 over a shift, the dust being about 90% iron. In most cases exposures will be less than this. This concentration is less than one twentieth of the allowable limit suggested by the Health and Safety Executive for iron fume, a form of industrial pollution that we believe is likely to be more dangerous than the larger particulate form we have found in the Underground.

Even if the HSE were too high by a factor of 5 (and in some countries the occupational exposure standard for welders is 1000μg/m^3), these concentrations would still be well within industrial safety limits. Since welders are regularly exposed to concentrations close to the 5000μg/m^3 standard yet rarely show evidence of radiological change, it is most unlikely that London Underground workers would ever be shown to have such changes on their chest radiographs.

It has been shown that workers exposed to iron fume, welders and burners, have a higher than expected risk of pneumonia. It is not known why this is so, but it has been speculated that it may be because iron encourages the growth of certain bacteria or because of the inflammatory stimulus caused by heavy doses of ultrafine iron particles. However, there is no such evidence in relation to iron accumulation in workers exposed to the metal in the form of larger particles.

This finding is supported by limited direct evidence from a study of London Underground workers. In 1968/70 and 1971/72 London Transport compared bronchitis sickness absence statistics in train drivers with bus drivers and conductors, in a study whose results were reported to the trade unions. Had there been an increased risk in relation to iron exposure, it would have been expected to show excess absence from chest illnesses among the train drivers. In fact the absence rates were higher among the bus drivers and conductors in all age groups in both periods. We do not want to overplay the importance of these results because the comparisons are inexact – bus drivers and conductors are more exposed to infection from the travelling public; there are many chest illnesses other than pneumonia; and sickness absence is an imperfect indicator of sickness – and the data were gathered some time ago. However we do note that the limited data available are consistent with the general inferences we have made.

In summary, our views of the risks to workers are as follows.

a. The physical and chemical characteristics of London Underground dust lead us to the conclusion that some iron may accumulate in the lungs of workers, but in a concentration and form that would not be expected to lead to fibrosis.
b. Similarly, there is no reason to suppose that it could cause emphysema, cancer, asthma or bronchitis.

c. It would not be absorbed into the body in sufficient quantities to accumulate in tissues other than the lung, and would not therefore cause haemochromatosis – see Section 1.4, earlier.
d. It is possible that there is some increase in risk of pneumonia among workers exposed to tunnel dust, by analogy with the increased risk observed among welders. However, we think that the risks are very low, because

- the exposure of workers underground is clearly lower than that of welders;
- the dust to which they are exposed is coarser; and
- the limited direct evidence available does not give any evidence of a problem.

7.9 IS THE GENERAL PUBLIC AT RISK FROM DUST EXPOSURE BY TRAVELLING ON THE UNDERGROUND?

Since we have concluded that the Underground worker who is exposed long-term to tunnel dust is not at risk of significant illness from dust inhalation – other than possibly a very small risk of pneumonia – it may seem odd to consider this question at all. However, the *in vitro* tests show that tunnel dust is not inert, i.e. that in sufficiently high doses it has a potential to cause inflammation. And there are in the general public individuals not usually represented in industrial workforces, such as the elderly and people with serious heart or lung disease, who are considered to be among the most susceptible to the effects of outdoor air pollution. Some of these may travel on the Underground and be exposed to the same dusts, albeit for shorter durations.

We do not think that the travelling public is at any serious or substantial risk from travelling Underground. We have four main reasons for this view.

a. *Daily exposures are not high*: It is reasonable to assume that the level of exposure for the public is similar although the duration will be much less. Although, in mass terms, dust concentrations underground are markedly higher than above ground, the relatively short duration of time exposed implies that in general the overall exposures associated with commuting underground are not high – in mass terms, they are similar to a day exposed above ground. (There will, of course, be variability around this average.)
b. *Tunnel dust comprises larger particles than ambient air pollution*: While the effects of coarser dust within the PM_{10} size range cannot be ignored, there is a growing body of evidence that a major reason for the observed epidemiological effects of ambient pollution on heart and lung disease is the ultrafine size range of the great majority of urban particles (EPAQS, 2001). A possible reason is that these very small particles have greater potential to be deposited in the acinus and move into the lung interstitial space where inflammation is more likely to influence adversely the cardiovascular system. These small particles are in lower concentration in the Underground than at the surface in the same area of London.
c. *Iron dust is not especially harmful*: Tunnel dust consists principally of iron, and studies of workers exposed to iron – even as a fume – suggest at most a very limited risk to health.
d. *Population susceptibility:* The data provided by London Underground suggest that the customer population is similar in age distribution to a working population. Moreover, it would be expected that the most vulnerable individuals to air pollution, those with heart and/or lung disease, would be under-represented among customers because of the obvious difficulties involved in such travel for the disabled. It is in our view reasonable to regard LU customers as generally comparable in susceptibility to a healthy workforce – certainly, much more comparable in susceptibility to a working population than to the general population.

We would summarise this as follows: given the need to travel within London, is there any reason related to risks to health to choose one form of transport over another? Our view is that there is not, even for susceptible people. Choices regarding travel in London should be based on cost and convenience, not on health.

Indeed, there is reason on theoretical grounds to adopt a stronger position, and to believe that the Underground, in terms of exposure to pollution, is as safe as or even safer than travel by car or bus. It is sufficient however for policy purposes that travel Underground is not clearly more dangerous than above ground, and of that we are confident.

7.10 DOES THIS MEAN THAT THE DUST IS COMPLETELY HARMLESS?

7.10.1 Tunnel dust is not harmless but the risks of disease are very small

No, this does not imply that the dust is completely harmless. It does have some potential to cause inflammation and, when large numbers of people are exposed daily to any dust that has some potential to cause harm, then that dust may on some occasions contribute to the development or exacerbation of disease.

However, in our view the dust has a very low potential to cause harm, and with one exception any risks of disease are likely to be so small as to be unidentifiable and unattributable. It is possible that there is some increase in risk of pneumonia among workers exposed to tunnel dust, by analogy with the low but increased risk observed among welders, though there is no evidence of increased risk among workers exposed to iron oxide in larger particles. Again, there is no direct evidence to date, but comparison of health data with above ground workers might shed light on this. The brief periods of exposure of the travelling public make any risk of pneumonia in them very remote indeed.

Long-term exposure may cause some accumulation of iron in the lungs of some workers. The amount of accumulation would depend on the concentration of dust inhaled and the duration to which workers had been exposed. Again, we think that the risks are very low. Significant iron accumulation is easily seen on a chest radiograph, and many Underground workers over many years will certainly have had such films taken for suspected chest problems. It would be surprising therefore if such x-ray abnormalities had occurred but not been noticed. Perhaps more importantly, siderosis is a ‘benign’ pneumoconiosis, and not associated with impairment of lung function; and so we believe that the likelihood of harm coming to Underground workers as a result of their exposure to tunnel dust is very small indeed.

7.10.2 Implications of that viewpoint

It is always wise and prudent to keep the levels of any workplace and ambient dust as low as practicable. There have been successes in London Underground in this regard – for example, the reductions in quartz content of the dust, and the Dust Action Group as a forum within the organisation – and we encourage management and unions in the Underground to continue to work together to find practicable ways of keeping dust levels low.

However we do not think that the risks, such as they are, warrant any special or extraordinary measures to limit exposures either of the workforce (for example, by use of face masks) or the travelling public (for example, by issuing warnings to the public generally or to specific sub-sections within it).

7.11 HOW RELIABLY CAN WE COME TO THESE CONCLUSIONS WITHOUT STUDYING WORKERS? WHAT FURTHER STUDIES WOULD REDUCE THE UNCERTAINTIES TO A WORTHWHILE EXTENT?

7.11.1 We think that these conclusions are reliable….

The strategy adopted in the present study has been to estimate risks indirectly, by reference to some suitable comparison dust and exposed population. We think that the work we have done to identify such a comparison, and the conclusions we have drawn, are reliable and, though not expressed quantitatively, that they are a suitable basis for development of policy with regard to tunnel dust and risks to health of workers and of the travelling public.

We are aware that we have not carried out any new direct investigations of workers, or the travelling public. Such studies, when the risks are low, often prove disappointing. It is indeed the case that one can never *prove* the absence of harm to people except by showing that no harm has occurred over a prolonged period. However, even after such a study some doubt remains, since it might be argued that insufficient time had passed for harm to become apparent; or too few people had been studied to include sufficient of whoever might be most vulnerable; or any of several other reasons why the absence of an association does not prove the absence of a risk. (Indeed, we do not suggest the absence of a risk. Rather, we think that the risks of serious harm, either to workers or the travelling public, are very small indeed, and probably unidentifiably small in any feasible study.)

Proving absolute safety is usually impossible. It is therefore usual to refer to the likelihood of harm occurring, and to base one's action on the seriousness of that harm, and the probability of it occurring. And we think the approach we have adopted is, under the circumstances, a good way of evaluating that likelihood.

7.11.2 …but nevertheless, they should be kept under review

The relationship between particles and health in general – what kinds and concentrations of particles cause adverse health effects; what are those effects and what are the risks; how do these effects occur; what is the role of co-exposure to other pollutants – this remains an intensely active area of research in the field of outdoor air pollution, and is still an active research area for workplace dusts also. New evidence, and new understandings, emerge each year.

Our conclusions are based on argument by analogy, using our best current understanding of particles, their effects, and associated mechanisms of disease. We think that this understanding is quite robust, in the sense that it may and surely will be modified over time, but is unlikely to change markedly, in a way that would markedly change our conclusions.

It is, however, wise to look on any evaluation such as this one as a draft evaluation, open to change and improvement in the light of new evidence and new thinking. For that reason we recommend, therefore, that the conclusions we have reached, and our reasons for reaching them, be reviewed from time to time, in case the wider understanding on which they are based changes in any way that would modify those conclusions.

7.12 POSSIBLE RELEVANT FURTHER STUDIES

7.12.1 Are further studies necessary at this time?

We have considered what further studies might give new information, specific to workers or the travelling public, that would help appreciably in reducing the remaining uncertainties and tunnel dust and its effects on health. Some ideas are outlined, briefly, below.

However, against the background of what is known already, and our view that the risks are small, we are not recommending as necessary any further studies at this time.

7.12.2 Possible relevant studies of the London Underground workforce

The principal reason for performing a survey would be to measure risks as accurately as possible, and hopefully more accurately than by the indirect approach we have used. This would normally be done when there is some evidence that harm is occurring to workers and evidence upon which to base a standard is required. We have been involved in a number of such exercises in the past, and two examples are worth mentioning. In the first, a chemical manufacturer had found x-ray changes in men exposed to PVC granules, and required to know their effects on the individuals concerned as well as the levels of dust that were responsible. In the second, the Trade Unions involved in the wool industry were concerned that wool dust caused wheezy chests in workers. In both cases there was evidence of an effect, and in both cases it was necessary to know how much dust had caused how much disease. The results of both studies led to formulation of legal dust standards to protect workers in the industries concerned. This outcome was the justification, agreed by all concerned, for exposing workers to x-rays and asking them to answer detailed questionnaires about their health.

Studies of people may themselves carry some risks to health, and the risk of harm occurring may outweigh the possible benefits in knowledge gained about the underlying pollutant and its effects. In particular, an x-ray survey of a workforce involves additional radiation to all those involved, and this will increase very slightly the risk of leukaemia – not a good reason to avoid x-rays where they are necessary, but a reason to consider carefully whether they really will be of benefit. Or again, any large-scale x-ray survey will almost certainly lead to some findings of an abnormality, say a chest x-ray shadow, completely unassociated with any exposure to dust. Such findings are common, are rarely serious, but always cause great anxiety and often lead to unpleasant and sometimes dangerous investigations. An x-ray survey would therefore be recommended if it were thought that the possibility of finding something that could be managed in a way beneficial to the individual outweighed these possible disadvantages.

In the case of London Underground, our advice is that the likely advantages of a survey would not justify risking the possible disadvantages, since we have concluded that the likelihood of discovering significant ill health related to dust exposure is remote.

However, if London Underground management and workforce remain unconvinced by our arguments, two possible studies might be considered. First, we have not excluded the possibility of an increased risk of pneumonia, although we think any such risk would be small. A mortality study of LU workers or a case-control study of pneumonia in London hospitals would address this question. Both of these would be relatively expensive, for possibly little benefit; neither would involve x-ray surveys. Secondly, if there is residual dubiety about long-term lung effects, a survey of selected workers with prolonged underground exposure might be considered. While not recommending these studies, these are valid approaches if it was considered that they were potentially informative enough to merit further work, and we would be prepared to carry them out if London Underground wished.

7.12.3 Possible relevant studies of the travelling public

Again, we do not see further work as essential at this time. However, some ideas that might be useful include:

- More detailed study of susceptibility, either from fuller examination of existing LU data on passengers, or from new surveys, e.g. to confirm the relative healthiness of passengers compared with the general population – this is an aspect which should be

kept under review insofar as London Underground is more successful at making travel more accessible to a greater range of vulnerable people;
- Systematic study of existing LUL records of sudden (acute) illness to workers and passengers – very preliminary and cursory examination of these records suggests that occurrences are rare, but we have not examined this issue in any detail; and
- A panel study (e.g. symptoms, peak flow), to look at 'mild' and/or commonly occurring effects in relation to when and for how long someone travelled on the Underground as distinct from other activities that involved exposure above ground.

7.12.4 Possible further investigations of the nature of the dust and of its relative toxicity

Here also, we are not making any recommendations. However, there is one specific issue that is fairly easily amenable to further investigation – whether in the living organism the lung lining fluid would sequester the iron and reduce its toxicity. We could examine this *in vitro* by studying the effect of tunnel dust on the same cells as we used in the present study, but with the inclusion of lung lining fluid and ferritin. Such a 'bridging' study would refute or support the contention of sequestration and allow us to better interpret the existing data.

7.13 FINAL REMARKS

It was clear from the outset that the present study would be important, because a wide number, and variety, of stakeholders have a legitimate interest in having a comprehensive assessment of the risks to health from exposure to tunnel dust. Along the way, however, we have also found the study very interesting. We had expected this, because such a study is intrinsically multi-disciplinary, and it is always rewarding to try to build an accurate picture when results and understanding from several disciplines need to be integrated. This was especially the case in the present study, because the results initially did not form a simple pattern, and so our search for a coherent interpretation has involved pushing the edges of what we know about particles and health.

We are pleased with and confident in the conclusions we have reached, but recognise that the issues are not simple, and look forward to engaging in constructive discussion of our findings.

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10. GLOSSARY OF TERMS USED

Acinus. The part of the lung most distant from the windpipe, comprising the smallest airways and the gas-exchanging tissues.

Aerodynamic diameter. The diameter of an airborne particle in relation to a sphere of unit density. This physical characteristic determines the falling speed of a particle.

Alveolar macrophage. A scavenging cell found in the lung acinus able to remove particles and initiate inflammation

Ambient pollution. Pollution of the general outside air, in this report in contrast to that in the Underground.

Apoferritin. A protein that combines with iron in the body, important in its transport.

COMEAP. The UK Department of Health's Committee on the Medical Effects of Air Pollutants

COSHH. The Health and Safety Executive's Control of Sunstances Hazardous to Health Regulations.

Clearance. The removal of particles from the lung, either up the bronchial tubes or by macrophages.

Dust concentration. The amount of particles in a given volume of air – usually in this report in micrograms per cubic meter (mass concentrations) or in numbers per cubic centimeter (number concentrations).

Dust sampler. An instrument that measures the concentration of particles in the air.

DustTrak. An instrument that makes continuous measurements of particle mass concentrations, in this report $PM_{2.5}$.

Epidemiology. The science of the study of patterns of health and disease, and their determinants, in groups of people ('populations').

Ferric, ferrous. Terms used to define chemically two different forms of iron with differing capabilities of combining with other elements.

Ferritin. An iron-protein complex that acts to store iron in the body

HSE. The UK's Health and Safety Executive, responsible for workplace regulation.

Haemoglobin. The iron-containing protein in the blood responsible for carrying oxygen.

IOM. The Institute of Occupational Medicine in Edinburgh.

***In vivo*.** A term denoting toxicological studies in whole live animals.

In vitro. A term denoting laboratory studies usually on isolated cultured cells.

Inflammation. The process of accumulation of defensive cells in the body in order to protect it against invading organisms or to repair damaged tissue.

Inhalable dust. Dust of an aerodynamic diameter such that most will be able to pass into the bronchial tubes.

Macrophage. See alveolar macrophage.

MEL. Maximum exposure limit – a UK workplace standard that may not be exceeded, used for the most toxic substances.

Microgram. A unit of weight, one millionth of a gram, denoted μg.

Micrometer (micron). A unit of length, one millionth of a meter, denoted μm.

Mucociliary escalator. The method by which the airways of the lung remove particles – transported in a mucous layer by the action of beating microscopic hair-like projections from epithelial cells.

Nanometer. A unit of length, one billionth (thousand millionth) of a meter, denoted nm.

Neutrophil. A body defensive cell that can swallow particles and bacteria and produce defensive chemicals – a hallmark of acute inflammation, often referred to as a PMN.

OES. A UK occupational exposure standard for many substances less toxic than those covered by MEL.

Particulate pollution. A general term for man-made pollution of the air by particles.

Phagocytosis. The process whereby cells such as macrophages ingest particles and bacteria.

PM. Particulate matter – a term defined by a subscript referring to the aeodynamic diameter of the particles concerned. For example, $PM_{2.5}$ refers to particles with an aerodynamic diameter of 2.5micrometers (μm).

PMN. Polymorphonuclear leukocyte – see neutrophil

PNC. Particle number count.

P-Trak. An instrument for measuring the concentration of particles smaller than 1μm in air.

Quartz. Crystalline silicon dioxide, a mineral component of many rocks, that may cause silicosis if inhaled in sufficient quantity.

Respirable dust. Dust of an aerodynamic diameter (below about 7μm) such that most will be able to pass into the lung acinus.

Silicosis. A serious scarring lung disease caused by inhalation of Quartz.

Titanium dioxide. A mineral commonly used as a non-toxic control dust in inhalation and *in vitro* toxicology, also used as a component of sunscreens.

Toxicology. The study of the mechanisms of poisoning and of damage to body systems or cells by harmful substances.

Transferrin. A body protein concerned with transport of iron into cells.

Ultrafine particles. A term used to describe very small particles. Increasingly it is being used to define particles below 100 nanometers in diameter, sometimes called nanoparticles.

11. ABOUT THE AUTHORS OF THE REPORT

Organisations

The ***Institute of Occupational Medicine***, Edinburgh (IOM) is an independent charitable scientific institute, established in 1969 to provide research, consulting and training in occupational and environmental health and exposure. Its purpose is to do work of social benefit, for people at work and in the community and so has a policy of publishing its research work. Over the years the IOM has become one of the world's leading organisations for research into the effects on health of industrial pollution, particularly inhaled dusts; increasingly, it carries out research and consulting on environment and health also. Its work includes multi-disciplinary expert reviews for the EC, UK Government Departments (e.g. HSE; Health; DfID; DEFRA), the World Health Organisation (WHO) and specific industries. Currently IOM has about 100 staff. It is a WHO Collaborating Centre.

The ***Universities of Aberdeen and of Edinburgh,*** are two of the UK's oldest Universities. They both enjoy very high reputations for independence and scientific excellence.

People

Dr. John Cherrie PhD is a human exposure scientist working at the University of Aberdeen and the IOM. He has led exposure estimation in several international epidemiological studies of workers and studies of people exposed to particulate air pollution, including for the International Agency for Research in Cancer (IARC). He is a member of the UK Government's Expert Panel on Air Quality Standards (EPAQS) that recommends UK standards for ambient air.

Professor Ken Donaldson DSc is one of the world's leading researchers into the mechanisms of toxicity of inhaled particles. He has made major contributions to understanding the effects of asbestos, quartz and more recently ambient air pollution including, with Professor Seaton and others, the original hypothesis explaining the effects of air pollution on the heart. He is a member of the UK Government's advisory Committee on the Effects of Air Pollutants (COMEAP).

Fintan Hurley MA is an epidemiologist and statistician with more than 25 years research experience on the health effects of long-term exposure to workplace pollutants (dusts, fibres, silica, pesticides). He has an international reputation in quantifying the health impacts of particles and other ambient air pollutants. He is a member of COMEAP and was a member of EPAQS 1998-2002. He is currently Research Director at IOM.

Professor Anthony Seaton CBE, FMedSci is a chest and occupational physician and emeritus professor of Environmental and Occupational Medicine at Aberdeen University. Formerly Director of IOM, he has spent much of his career investigating the effects of the environment on the lung, and is an acknowledged world expert in lung and occupational diseases, having written over 200 scientific papers and several books on the subject. He was for 10 years and until recently chairman of EPAQS and a member of COMEAP.

Dr. Lang Tran PhD is a mathematical modeller with a substantial international reputation as a quantitative toxicologist. Since the early 1990s has carried out research on inhaled particles and the lung, including the lung's defensive responses.

APPENDIX 1: WORK IN PREPARATION FOR DUST MEASUREMENTS IN THE LONDON UNDERGROUND

A1.1 DEVELOPMENT OF A $PM_{2.5}$ SAMPLER TO COLLECT SAMPLES FOR THE TOXICITY STUDY

A1.1.1 Introduction

In order to collect sufficient airborne dust within a reasonable time period for the toxicity testing it was necessary to specially a high volume sampler. The particles of interest were those with diameter 2.5μm and below, and to ensure a sufficient mass of dust was collected it was judged that the sampler had to run at 16 l/min over several days. The IOM have already developed a number of samplers of this type and it was considered possible to adapt an existing design for the present study. It was decided that the sampler performance should be comparable with the published data for the US EPA $PM_{2.5}$ standard.

A1.1.2 Methods

The evaluation method used was consistent with that described in prEN 13205, *Workplace atmospheres - Assessment of performance of instruments for measurement of airborne particle concentrations* (CEN, 2001). The design of the test system was based on that described by Kenny and Liden (1991) used for the measurement of aerosol penetration through cyclone samplers. The approach requires comparison between the aerodynamic size distribution of an aerosol penetration through the selection stage under test and that challenging it.

A polydisperse aerosol of glass ballotini (Spheriglass 5000 CPOO) was generated in a calm air chamber using a TSI 3400 Fluidised Bed aerosol generator. The aerosol was modified by partial removal of the smallest (mainly <1.0 μm) particles using a virtual impactor, and charge level was controlled by injection of ionised air from a ^{210}Po α source.

Foam plugs were mounted in a modified IOM 37mm personal sampler cassette and exposed to the aerosol, with a sampling rate of 16l/min. The particle size composition of aerosol drawn through the samplers was analysed using a TSI aerosol particle sizer (APS 3320) and compared with aerosol drawn through an identical set of tubing, but with no foam inserted (the reference). Samples of one minute's duration were drawn through each system in turn, allowing a 1 minute gap between samples to ensure complete replacement of aerosol in the tubing. In each case three reference and two foam-penetration samples were taken.

A1.1.3 Analysis

Using an Excel spreadsheet, reference and penetration samples were averaged at each particle size, and foam penetration measured as a fraction of the reference aerosol. These data were transferred to the TableCurve computer package, penetration normalised to 100% at 1 micron to eliminate effects caused by non-linearity of the APS inlet below this size, and curves were fitted, from which the diameter where 50% of the aerosol penetrated (D50) was determined. Simplified (9-point) penetration curves were generated from the fitted curves for presentation in this report.

The performance data for one of the selectors was assessed against the target convention for $PM_{2.5}$ issued by the US EPA, (Federal Register, 1997) using the bias map approach described in prEN 13205 (CEN, 2001). The bias between the fitted performance curve and the target convention for an array of challenge size distributions was calculated. The $PM_{2.5}$ Federal

Reference Method (FRM) defines $PM_{2.5}$ largely in terms of conformity to the particle size-selection characteristics of the WINS impactor, (Federal Register, *ibid*) following a 10-micron inlet stage selector, for several specified airflow conditions, challenge aerosol compositions, and a range of temperatures.

The FRM also defines a procedure whereby a sampling device may attain 'equivalent' $PM_{2.5}$ status. The aerodynamic size selection curve of the WINS impactor is published (and presented for comparison in Figure A1, below) and the regulations require that any 'equivalent' $PM_{2.5}$ sampling device must have a D50 of 2.5 $\pm$ 0.2µm. The standard requires that a sampler should have a bias for $PM_{2.5}$ concentrations less than ± 5% (when compared with the WINS characteristic), although at the present time it is not clear exactly how far the range of size distributions extends. The sampling bias is calculated numerically for 3 generalised aerosol size distributions, and further tests are also required.

While it is not practical in this project to comply with all the FRM conditions, we can easily compare the selection curve of a candidate sampler to the WINS device, and determine its D50 to the required accuracy.

A1.1.4 $PM_{2.5}$ Selector results and discussion

The results for four candidate sampler selection foams (expressed in terms of D50) are given in Table A1.1.

Table A1.1 Measured D50s for four foam selectors

Selector	***1***	***2***	***3***	***4***
Thickness	27	31	26	21
D50	2.61	2.18	2.22	2.38
	2.64	2.07	2.65	2.30
	2.72		2.33	2.63
	2.72		2.30	
	2.67		2.28	
	2.85			
Mean D50	2.67	2.13	2.35	2.44
St Dev	0.13	0.08	0.16	0.17

Only selector 4, which was assembled from 4 foam discs, gave a mean D50 rather closer to 2.5µm than foam 1. However, it can be seen from the data that the pre-cut plug gave a selection that is within the EPA conditions for a $PM_{2.5}$ sampler and the variability was slightly lower than for the best assembled plug (i.e. selector 4). It also may be argued that since the slope of the characteristic for the foam sampler is more gradual than the cyclone cut on which the EPA standard is based, use of a foam sampler with a D50 slightly over 2.5 would remove fewer of the 2 – 2.5µm particles, which might be of interest in this study.

The selection curve for foam 1 (Single pre-cut foam 30mm diameter by 27mm thick, nominal 90ppi) is shown in Figure A1.1a, together with the curve conforming to the US EPA $PM_{2.5}$ convention. The bias map based on these data is shown in Figure A1.1b.

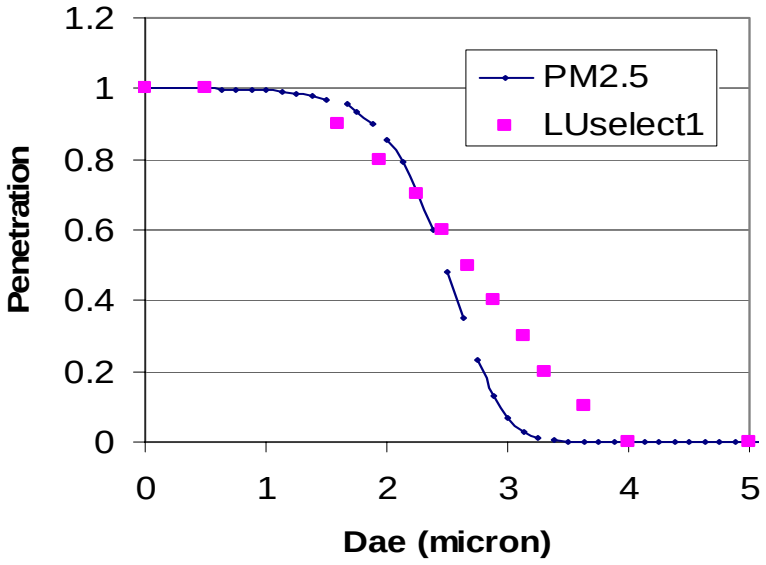

Figure A1.1a The size selection curve for foam selector 1 and the US $PM_{2.5}$ standard. Note:- US PM2.5 curve as published in US EPA, 40 CFR Part 53, Table F-4.

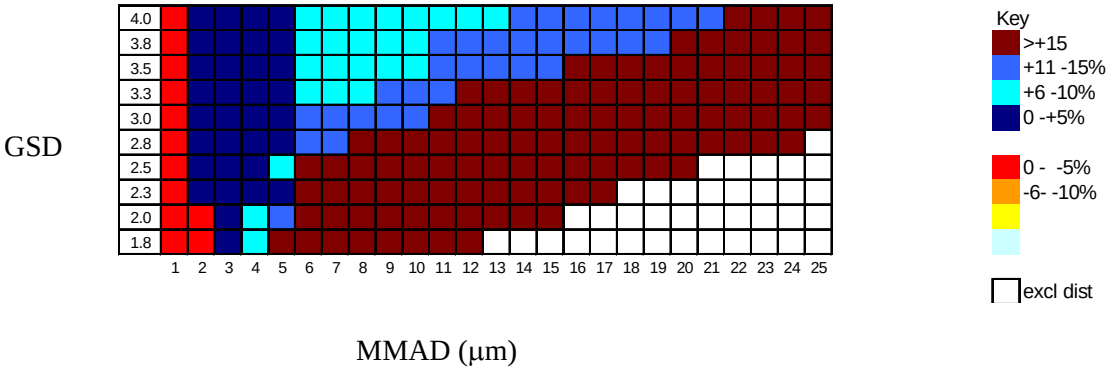

Figure A1.1b Bias map for foam 1

The measured D50 for this sampler insert is 0.17µm over the target D50 of 2.5µm. Examination of the full penetration curve (Figure A1.1a) shows initial slight under-sampling followed by a tendency to over-sample from 2.5 to 4µm. This was confirmed by considering the bias map in Figure A1.1b, which shows the likely bias in sampling with the foam selector when compared with the standard curve for a matrix of different aerosol size distributions. In this bias map each box corresponds to a distribution with given mass median aerodynamic diameter (MMAD) and geometric standard deviation (GSD). For all size distributions with a MMAD less than 1µm, the bias is slightly negative, while for most distributions between 1 and 5µm, the bias is within +10%. The largest biases are for distributions with larger MMADs, i.e. greater than about 5µm.

A1.1.5 Conclusions

The recommended sampler configuration for this application is the IOM 37mm porous foam personal sampler used in earlier studies, modified to have a simple 30mm diameter sharp-edged tubular inlet, and fitted with a single pre-cut foam of 30mm diameter and 27mm thickness, of nominal 90 ppi. pore size. The inlet flow rate should be 16 l/min.

A1.2 THE PILOT STUDY

A1.2.1 Objectives

A short pilot study was undertaken to check the practicality of the proposed measurement programme. This was completed over two days at one of the stations that would ultimately be selected for the main study and in a train cab. The key issues to be resolved related to the stability of the direct reading monitors in the underground railway environment and the safety and security of the sampling equipment.

A1.2.2 Methods

Measurements were made on two days at Holland Park station. The measurements were carried out with the sampling pumps and other equipment placed inside a cupboard with the sampling heads located outside the cupboard about 2.5m above the platform. Two direct reading instruments were used to obtain measurements of $PM_{2.5}$ and the particle number concentration for a proportion of each day. These same instruments were also used on one day to monitor the concentration in the cab of a train running on the Central line.

The concentration of $PM_{2.5}$ was measured using a portable battery operated DustTrak light scattering monitor (manufactured by TSI Inc., St. Paul, Minnesota, USA). This device continuously draws air through a $PM_{2.5}$ size-selective inlet into the sensing chamber where a beam of laser light is shone through the air stream. The particles present in the air act like tiny mirrors scattering light in all directions. A lens at right angles to both the airflow and laser beam collects part of the scattered light and focuses it onto a sensor. The amount of light scattered is proportional to the mass of the particles in the air. The DustTrak monitor must be calibrated because the light scattering response is dependant on the type of dust being sampled.

The particle number concentration was measured using a P-Trak monitor (TSI Inc.). The operation principle is similar to the DustTrak. Particles are again drawn through the P-Trak using a built-in pump. Before entering the sensing zone the particles pass through a saturator tube where they mix with an alcohol vapour and the mixture is then drawn into a condenser tube where the alcohol condenses on the particles causing them to grow into larger droplets that can be counted more easily. These droplets then pass through a laser beam producing scattered light pulses that are sensed by a photodetector and counted to determine particle number concentration. The P-Trak is designed to count particles between 0.02 and 1μm.

Three other sampling devices were used to collect airborne dust samples for subsequent laboratory analysis. These were:

- a $PM_{2.5}$ sampler;
- a PM_{10} sampler and
- a respirable dust sampler (which approximates to a $PM_{3.5}$ sampler).

All of these comprise a battery-operated sampling pump and a sampling head connected to the pump by flexible plastic tubing. The air is drawn through a pre-weighed filter located in the sampling head and any particles are trapped on the filter. The sampling heads are designed

to select the particle sizes of interest. So for example the PM_{10} and $PM_{2.5}$ sampling heads contain a section of polyurethane foam which is designed to remove particles greater than the stated size according to the agreed standard criteria. The respirable dust sampler (Casella) comprises a cyclone pre-selector, which removes oversize particles by a centrifugal process. The flow rate for the cyclone sampler was adjusted to 2.2 litres/min to ensure that the size selection corresponded to the International Standard Organisation criteria.

The airflow rate through the sampling head was measured at the beginning and end of the sampling period and at a number of intermediate times. At the end of the sampling all of the filters were returned to the IOM laboratory where they were re-weighed and, if required, subjected to further chemical analysis. The mass concentration of dust was calculated from the change in filter weight, the duration of sampling and the average flow rate.

All of the respirable dust samples were analysed by infra-red spectroscopy to determine the mass of quartz on the filter. This was done using the method published by the Health and Safety Executive for direct on filter assessment of quartz in respirable dust samples (HSE, 1987).

A1.2.3 Results from the pilot study

Table A1.2 summarises the gravimetric data from the pilot study measurements at Holland Park station. The $PM_{2.5}$ concentrations were less than the respirable dust concentrations, which were lower than the PM_{10} measurements, as expected. The $PM_{2.5}$ data were approximately one third of the respirable dust and PM_{10} concentrations. These data are similar to previous measurements made in the London Underground system.

Table A1.2 Summary of average airborne dust measurements made during the pilot study

	Day 1	Day 2
$PM_{2.5}$ (μg/m^3)	280	310
Respirable Dust, approximately $PM_{3.5}$ (μg/m^3)	970	990
PM_{10} (μg/m^3)	1070	1120

Two respirable dust samples were collected on each day and these samples were all analysed to assess quartz concentration. Table A1.3 shows these data and the proportion of quartz in the respirable dust.

Table A1.3 Respirable quartz concentrations and the proportion of quartz in respirable dust

Date	Quartz (μg/m^3)	Quartz (Percentage of respirable dust)
Day 1	20	2.3%
Day 1	20	2.2%
Day 2	30	2.8%
Day 2	40	4.2%

The quartz concentrations were all low, ranging from 20 to 40 μg/m^3. For comparison the maximum exposure limit for respirable quartz in the UK is 300μg/m^3, approximately ten times greater than the measured values. The proportion of quartz in the respirable dust was also low, between approximately 2 and 4%

The data from the first day of sampling using the DustTrak and P-Trak monitors are shown in Figure A1.2.

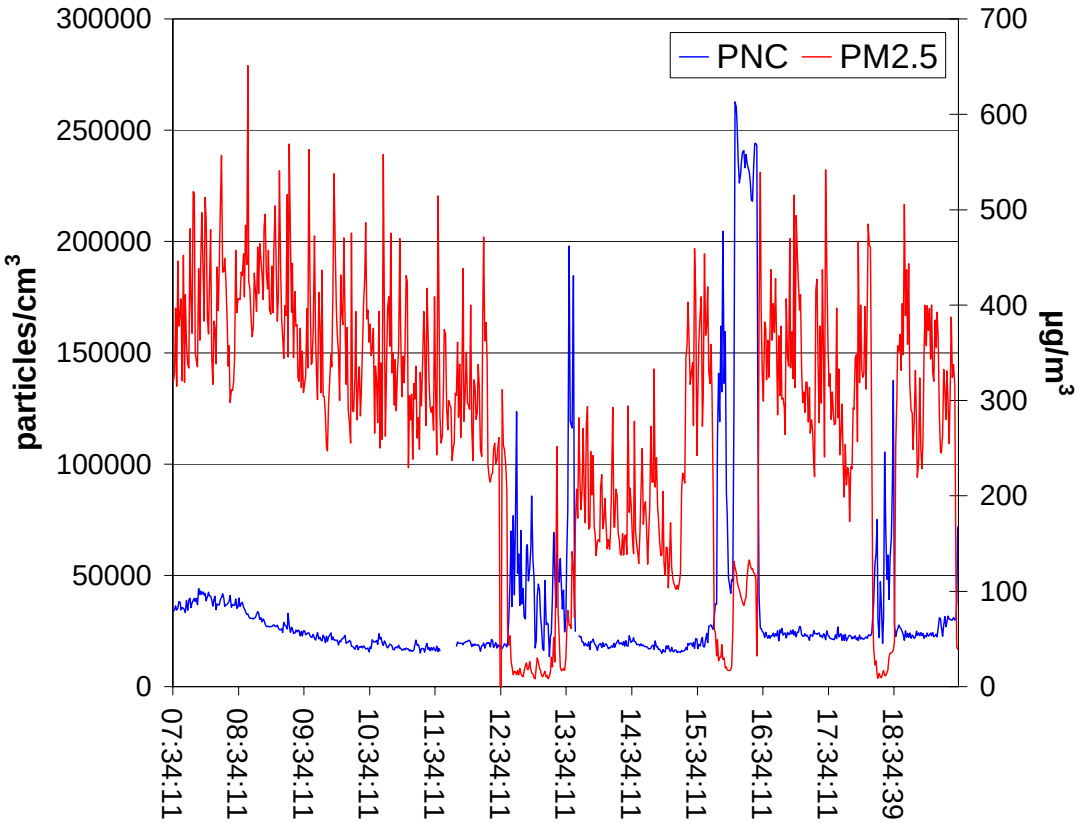

Figure A1.2 Time record of airborne dust concentrations made during day 1

The measurements commenced about 07:30 and continued until almost 19:00. The scale for the particle number concentration data is located on the left side of the figure and the gravimetric concentration data on the right side. For most of the day the monitors were located on the station platform, however on three occasions they were taken to the surface: between 12:43 -13:42, 15:51 - 16:30 and 18:17-18:33. These three time periods are clearly seen on the graph because the relationship between the two measurements changes; on the surface the number concentrations are relatively high and the mass concentrations relatively low, while underground on the platform the opposite pattern was observed.

The maximum number concentration recorded on the platform was approximately 41,000 particles/cm^3 and this occurred around 08:02. The maximum concentration of $PM_{2.5}$ occurred slightly later at 08:42 and it was approximately 650µg/m^3. Both sets of measurements tended to decline until mid-afternoon and then increased, although the pattern of change was more marked for the mass concentrations.

On the second day the monitors were carried by one of the researchers who accompanied a driver. Unfortunately only data from the DustTrak are available because of problems experienced with the equipment. These data are shown in Figure A1.3.

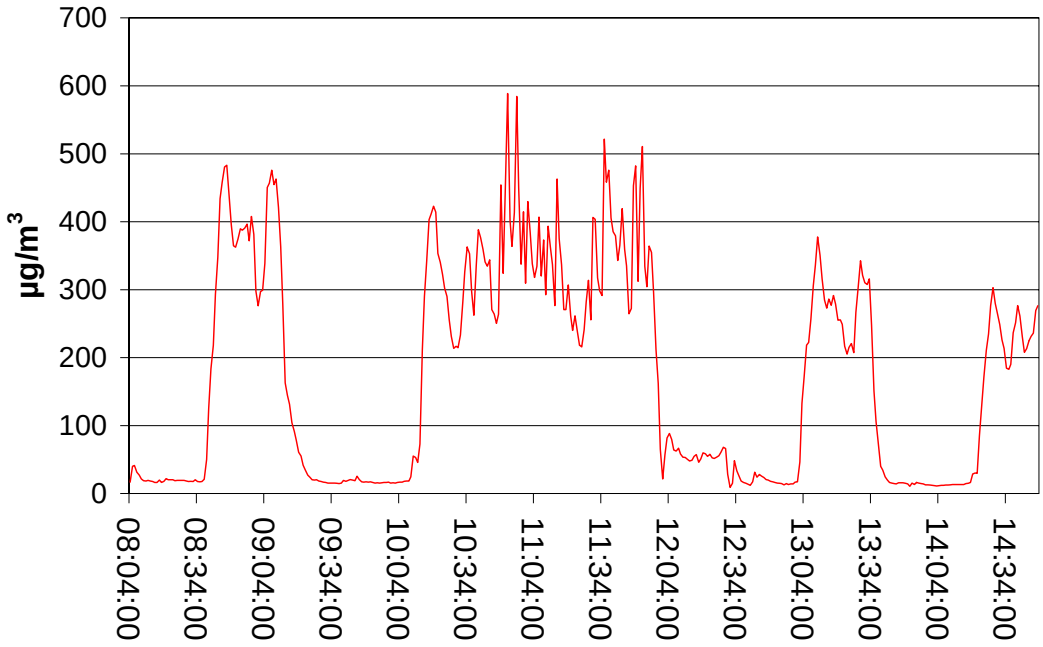

Figure A1.3 Time record of airborne dust concentrations made during day 2

All of these data were measured inside the cabin, except for very short time periods when changing from one side of the tube to the other and from 10:11 until 10:43 when the monitors were placed on Holland Park tube station platform. Initially the train is on the surface and then from 08:34 to 09:10, the first period of increased concentration, the train is underground. From 9:12 to 9:37 it is again on the surface and the concentration declines. All of the increased levels correspond to periods underground, with the exception of 12:01-12:30 when the driver was in the canteen at White City (there was no smoking although there was an open kitchen area). The highest concentration was 0.590µg/m^3 measured at 11:02.

A1.2.4 Implications for the main monitoring programme

The pilot study showed that it was practicable to collect a wide range of measurements from the station platform and to obtain real-time measurements of particle number concentration and $PM_{2.5}$ from the cabs of trains. However, although it was possible to gather these data it was apparent that the work was more time-consuming than we had originally anticipated. It was judged difficult to obtain samples over the whole period that stations were open and we decided to collect samples only from early morning until late afternoon.

APPENDIX 2: ESTIMATION OF DOSES TO CELLS AND TO HUMANS

This Appendix gives some details of methods and assumptions used in comparing the doses of tunnel dust administered to cells in the *in vitro* studies reported in Chapter 6, compared with the dose to the human lung of people exposed underground.

A2.1 ASSUMPTIONS AND RESULTS

A2.1.1 Healthy individuals

Calculations were carried out using the MMPD computer program V 1.0, developed at CIIT for calculation of deposition of particles in the human lung. Use of the program requires some assumptions about the dust of interest. The assumptions used about tunnel dust were based on what is known about the dust; these assumptions are listed in Table A2.1.

Table A2.1 Assumptions about LU particle characteristics

Density	6g/cm^3
CMD	0.7μm
GSD	1.0
Concentration	Average 300μg/m^3
Breathing Pattern	Nasal (resting)
Breathing frequency	12 Per minute
Tidal volume	625ml
Inspiration fraction	0.5
Hours per day	4 hours/day
Days per week	1 day

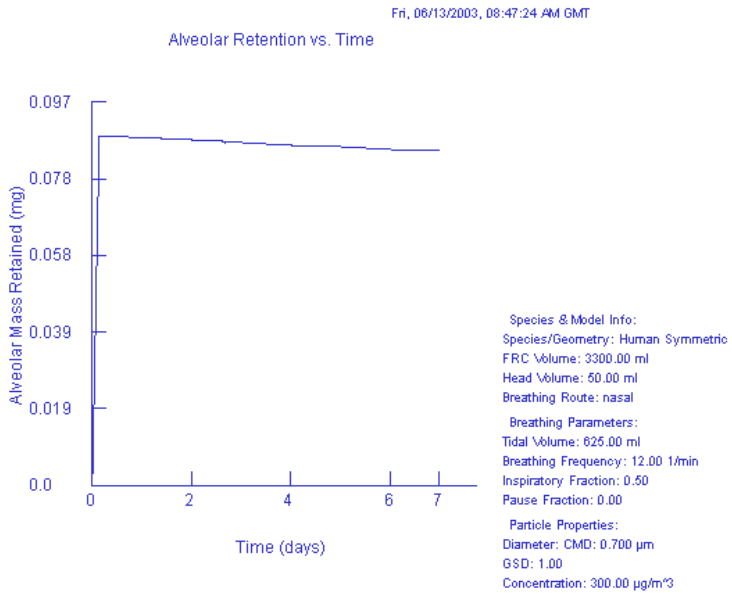

Figure A2.1. Plot of alveolar mass burden for an average human exposed as per Table A2.1

A2.1.2 Dose estimation for a stressed individual

We performed an extra run, this time for a 'stressed' individual. The parameters changed are breathing pattern, breathing frequency and tidal volume.

Table A2.1. Parameter values for a 'stressed' individual. The rest of the parameters are kept fixed, i.e. they are as per Table A2.1.

Breathing Pattern	Nasal-Oral
Breathing frequency	24 Per minute
Tidal volume	1200ml

The result of the 2nd simulation is given in Figure A2.2.

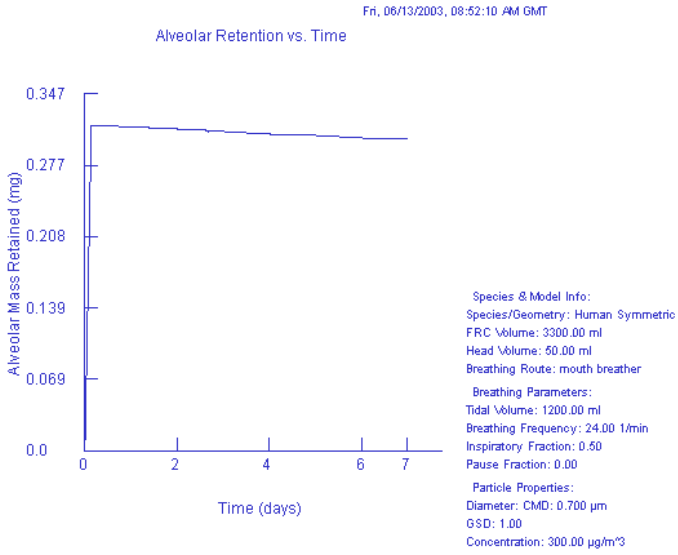

Figure A2.2. Plot of alveolar mass burden for a 'stressed' human exposed as per Table A2.2.

A2.2 DISCUSSION: COMPARISON OF DOSES TO CELLS AND TO HUMANS

In our *in vitro* systems the maximum dose used was 100µg/ml. This translates into a dose per unit surface area of (100/1.8)µg/cm^2; i.e. 55.5µg/cm^2

From the model we assumed a person exposed to 300µg/m^3 LU dust and obtained an alveolar burden of 90 µg after 4 hours exposure (see Figure A2.1). In order to compare this estimate with the *in vitro* dose we make the following assumptions:-

- the total surface area of the alveolar region of the human lung is 140m^2
- the proximal alveolar region is approximately 5% of the surface.

Under these circumstances the dose per unit surface area in the proximal alveolar region of a healthy person is 90µg /(5/100 x 140m^2). This equates to 90µg/7m^2 or 0.0013µg/cm^2.

Comparing the *in vitro* dose of 55.5µg/cm^2 with this modelled dose of 0.0013µg/cm^2 shows a 40,000-fold greater *in vitro* dose (55.5/0.0013 =42308).

For the 2nd case, of a stressed individual, the alveolar burden is approximately 300μg. In this case the dose per unit surface area is 300μg /7m^2 or 0.0043μg/cm^2; i.e. a little more than three times that of a healthy person and still much less than that used *in vitro*. Example of Heading 1 (Heading 1 style used)

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The IOM is a major independent centre of scientific excellence in the fields of occupational and environmental health, hygiene and safety. We aim to provide quality research, consultancy and training to help to ensure that people's health is not damaged by conditions at work or in the environment. Our principal research disciplines are exposure assessment, epidemiology, toxicology, ergonomics and behavioural and social sciences, with a strong focus on multi-disciplinary approaches to problem solving.

Our beginnings

Our first major research programme began in the 1950s, on respiratory health problems in the coal mining industry. Major themes were quantification of airborne dust concentrations in different jobs, characterisation of types and constituents of the dusts, measurement of health effects, relationships between exposure and disease, and proposals for prevention. This research became an international benchmark for epidemiological studies of occupational health, and was the primary influence on dust standards in mines in the UK, US and other countries.

Current themes

Our current work spans many other industries including asbestos, MMMF, pesticides, chemicals, energy, telecoms, metals, textiles, construction, agriculture as well as the environment. While diseases of the respiratory tract remain a major interest, our scope now extends to many other health outcomes such as mortality, cardiovascular effects, cancer, back pain, upper-limb disorders, hearing loss, skin diseases, thermal stress and psychological stress. Related work includes the development and application of measurement and control systems, mathematical models and survey methods.

Who we work for

Our work in these areas is conducted for a wide range of organisations in the UK, the EU, and the US, including Government departments, international agencies, industry associations, local authorities, charitable organisations, and industrial and commercial companies. The IOM is a World Heath Organisation (WHO) collaborating centre and is an approved institute of the Universities of Edinburgh and Aberdeen, enjoying collaborative research links with NIOSH, IARC, and many other institutes throughout the world.

Publication

We believe that our research findings should be publicly available and subject to the scrutiny of the international scientific community. We publish our findings in the peer reviewed scientific literature and through our own series of Research Reports.

Contact

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