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Informing transport health impact assessment in London

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OCTOBER 2000

Commissioned by
NHS Executive
London

Transport is one of the **London Health Strategy's four main priorities**:

- Regeneration
- Inequalities
- Black and minority ethnic health
- Transport

And health impact assessment is also one of the additional **underpinning priorities**:

- Health impact assessment
- Community development and involvement
- Establishing the London Health Observatory
- London's Health communications

Further information on the developing London Health Strategy is available on the website: **www.londonshealth.gov.uk**

The website contains a range of publications and reports, including:

- ***'A Short Guide to Health Impact Assessment'***
- A fuller ***'Resource for Health Impact Assessment'***

This report was commissioned by the Research & Development directorate, NHS Executive London, to contribute to the on-going development of the London Health Strategy and the work of the new London Health Commission.

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Executive Summary

Assessing how transport impacts on health in London

Transport can affect health in a range of both positive and negative ways. It can help people access services, reduce isolation and increase opportunities for work and social activities, all of which have the potential to improve health. However there is also potential for negative impacts through, for example, accidents and air pollution.

In London, 'Transport' has been identified as one of the four key priorities in the London Health Strategy, which aims to support efforts across the capital to improve health and reduce inequalities. In this context the NHS Executive in London was keen to commission work that would help inform understanding of the impacts of transport on health, and provide support to people developing policies, programmes and projects across London.

Increasingly health impact assessment is proving to be a valuable tool for policy makers and practitioners at all levels to assess the impacts of proposals on health. The work in this document has therefore been designed to help contribute to and inform this process. Assessing comprehensively the impacts of transport on health is not easy and so the research has sought to describe what we are able to quantify and assess and at the same time highlight areas where the evidence is less clear and work is still required.

The research has looked for the first time at how transport in the widest sense – from walking and cycling, to lorry and car and bus traffic – affects the health of Londoners. As an initial study, it focuses on assessing and evaluating the more direct impacts of transport and health, where health is defined in its widest sense as a state of physical, mental and social well-being.

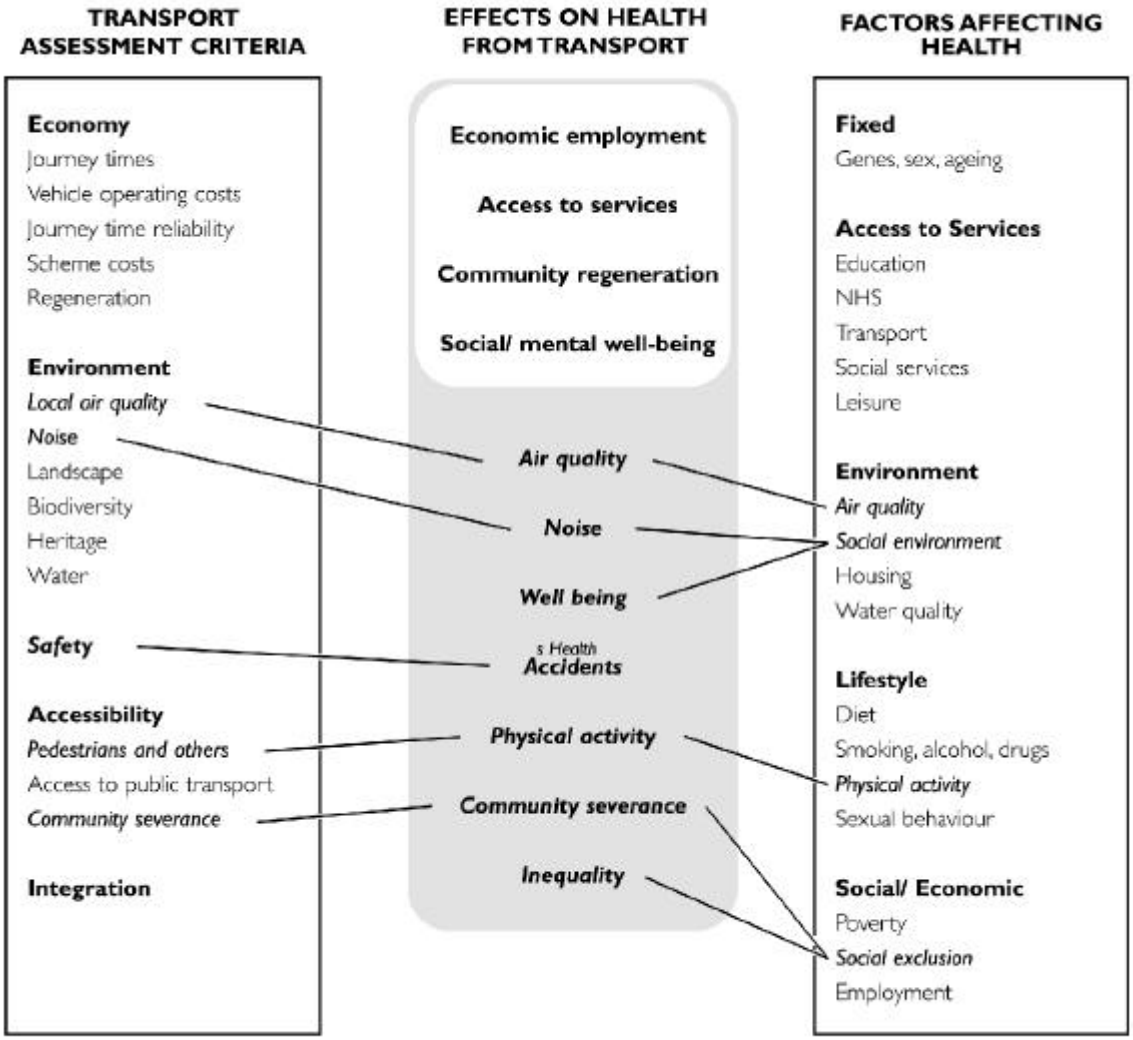
The direct health effects of transport included in the study are:
• Traffic accidents;
• Air-pollution related health effects from transport emissions;
• Noise-related health effects from transport activity;
• Health benefits from physical activity such as cycling and walking;
• Community severance, mental health and inequality effects.

The study has not assessed the substantial indirect benefits of transport, which include delivery of food to our shops and goods and services to the population, as well as moving people around the capital. Such benefits are clearly important and will need to form part of the equation when health impact assessment is applied to transport policy or practice.

Looked at in the widest context this study provides an important contribution to work aiming to minimise the ill effects and maximise the benefits of transport in London.

The Study

The context of the current study with respect to HIA and transport appraisal is shown in the shaded area below.



It is important to stress that this research project is only a first step and further work is required to inform our collective understanding of factors impacting on health and their inter-relationships. This stage describes the evidence and discusses whether quantification is possible. To fit with the scoping goal of HIA, it has assessed the relative size of the health effects for different categories relating to transport in London, to provide some guidance on how important these effects are relative to each other. One of the key aims of this phase of the work was to identify impacts that can be quantified and those for which quantification is problematic. By doing so, we hoped to identify gaps in the knowledge base and highlight research priorities.

Within this first phase it is impossible to recommend one generic approach and a framework which is universally applicable for researchers and decision-makers in health assessment and in transport appraisal, and one that can be applied to detailed schemes and at the same time to London wide policies. Future work will need to build on the information here by exploring the more difficult to assess and marginal effects on health arising from transport decisions.

Finally, as part of this study to identify evidence and potential frameworks, a number of organisations were consulted (e.g. Department of Health, Greater London Authority, Transport for London and the Department for Environment, Transport and the Regions). It is clear from this work that wider consultation continues to be important. This will need to involve both potential users of the information from this study, as well as a wider group of stakeholders to assess how to relate the relative effects from different categories of impact and against other criteria (both for health and for transport). This summary report covers the following areas:

- Accidents
- Air pollution
- Noise
- Physical activity
- Community severance and other effects
- The relative effects of transport on health
- Research recommendations
- Quantification frameworks

The evaluation by effect category is presented below.

Accidents

The most obvious and one of the most significant effects of transport on health is traffic accidents. The impacts of transport on health from accidents are obvious; the cause and effect chain is simple and immediate. The assessment of accidents is therefore easier than with other categories considered in this study. There are already established quantification methodologies for predicting transport accidents and casualties. These methods use historical accident data as a means of predicting future accident rates from new schemes or policies, adjusting for road type and speed, as these influence the numbers and severity of accidents.

The current levels of health impacts from road transport accidents in London are recorded and reported. It is worth noting that there are differences in the trends in transport statistics in London compared to the UK generally: accident rates in London remain relatively constant year on year, whilst rates in the rest of the country decline.

Finally, the distribution of accidents does impact on certain road-users and age groups disproportionately. The relative risk of serious injury or death by distance and by journey for pedestrians, cyclists and motor-cyclists is much higher than for other road users (indeed of the accident fatalities in London 1998, more than half were pedestrians). There are also links between transport choice (and accident rates) and both social class and vulnerable groups, particularly children.

Air pollution

Studies of pollution episodes (such as the London smog episodes of the 1950s) have shown that very high levels of ambient air pollution are associated with strong increases in adverse health effects. Recent studies also reveal smaller increases in adverse health effects at the current levels of ambient air pollution typically present in urban areas. These health effects include a range of endpoints, such as premature mortality (deaths brought forward), respiratory and cardio-vascular hospital admissions, and possibly exacerbation of asthma,

respiratory symptoms and loss of lung function. The evidence for these effects is strongest for the pollutants PM_{10} , SO_2 and ozone and the relationships are widely accepted as causal. Recent studies also suggest that long-term exposure to these pollutants, especially particles, may also damage health and that these effects may be substantially greater than the acute effects described above.

Transport is a major source of these atmospheric pollutants in urban areas and therefore can be assumed to have adverse health effects. Frameworks for quantifying the health impacts of transport-related air pollution do exist. The frameworks require a series of steps and involve additional analysis to that typically found in transport model outputs. Such approaches first quantify emissions from transport vehicles (taking into account that emissions vary with vehicle type, fuel type, technology and speed). They then proceed to assess the effects of these emissions on local air pollution concentrations, usually with the use of dispersion models. The pollution data is then combined with data on population to estimate the population weighted air pollution increase. The final step is to quantify health impacts with the use of exposure-response functions from epidemiological studies, which link ambient air quality to health endpoints. There is however debate on the exact exposure-response functions and health endpoints that should be included in any assessment framework.

For this study we have compared the results from three sets of exposure-response functions: from the Department of Health's COMEAP (Committee on the Medical Effects of Air Pollution) sub-group, from the EC's ExternE Project and from functions from specific London epidemiological research studies. We have combined the first two of these to provide a framework for quantification of health damages from transport-related air pollution in London. This uses COMEAP recommendations to quantify those effects for which there is the greatest confidence (least uncertainty) and ExternE recommendations to estimate additional health impacts where the evidence is strong but where quantification is more uncertain. Impacts have been classified into uncertainty bands, to reflect the different confidence levels attached to different health endpoints and quantification approaches. The approach is applicable for looking at marginal changes resulting from transport decisions. We have used this approach to compare the potential importance of air pollution relative to other categories of impact. This analysis leads us to conclude that air pollution related health impacts from transport may be equivalent to, if not greater than transport accidents in London.

Noise

Noise is a major nuisance and is widely recognised as a dis-benefit affecting daily life. It may also lead to a number of health impacts through a variety of direct and indirect effects, although there is considerable debate on the reliability of the evidence. Transport is a major source of ambient noise levels and therefore may have important health impacts.

The evidence for noise impacts has been assessed, as discussed in recent major reviews. The conclusive impact of the health effects of noise is mostly limited to cases of hearing loss and tinnitus caused by long periods of (occupational) exposure. These effects are generally not important at the typical levels of noise arising from transport. A number of studies also point to potential physiological and psychological impacts from the noise levels associated with transport (from road, rail and aircraft), including speech interference, annoyance, sleep disturbance, educational performance, cardiovascular and physiological effects, and mental health effects.

There is general agreement that noise is a source of annoyance. There are, however, problems in interpreting the potential health impacts of both direct and indirect routes from noise stimulus to the annoyance effect. These problems arise because annoyance is related to the duration and the frequency components of sound and because annoyance relies on subjective measures and the sensitivity or susceptibility of individuals. The remaining evidence for effects of environmental noise on health are strongest for sleep disturbance, ischaemic heart disease and performance by school children. It is stressed that much of the evidence in support of actual health effects other than annoyance and some indicators of sleep disturbance is quite weak. The data on other possible health consequences, such as low birth-weight and psychiatric disorders, are inconclusive.

Existing exposure-response relationships are confounded by a number of variables that serve to scatter data points around the cause-effect curves. The scientific evidence suggests a threshold below which no effects are expected to occur, although this cannot be considered definitive. As well as this uncertainty in relation to exposure-response functions, there are other major problems in developing noise and health quantification frameworks. These relate to subjectivity and individual responses to noise, as well as how well average noise levels (commonly used to predict noise amenity effects) actually relate to a metric which is relevant for health endpoints. In summary, although it is possible to assess quantitatively the noise levels from transport, it is very difficult to evaluate quantitatively what the health consequences of these levels are. A qualitative approach is possible, though there remains considerable debate on the reliability of evidence relating to health effects.

Physical Activity

Physical activity has significant health benefits in reducing coronary heart disease and in reducing other health impacts such as diabetes, obesity, hypertension, cancer, osteoporosis and even depression. Transport (through the encouragement of cycling and walking) can therefore provide significant health benefits. Health benefits are realised through 30 minutes of moderate activity on most, and preferably all, days of the week. Physical activity is recommended (rather than participation in organised sports or exercising) because most people can incorporate moderate physical activity into their daily routine – and it is in this respect that there is a link between transport and health.

There is quantification evidence relating physical activity to health endpoints. In theory this should allow the estimation of possible benefits for transport-related travel (such as cycling and walking). Inverse causal relationships exist between physical activity and all-cause mortality. These relationships may be significant (equivalent to the difference in mortality of non-smokers and smokers). There are also similar relationships for the risk of developing coronary heart disease, type 2 diabetes mellitus, cancer mortality (and specifically cancer of the colon) and hip fractures in elderly men and women.

There are also a number of other benefits from physical activity, for which good evidence exists but for which quantified estimates of benefits are not available. These include reduced long-term risk of osteoporosis, greater well-being and self-esteem, as well as benefits in reducing mild depression and mild anxiety. There may also be benefits in later life, including improvements in balance, co-ordination, mobility, strength and endurance, in the control of chronic disease, and in reducing weight especially for the obese, as well as general benefits in keeping people mobile. It is likely these benefits would be realised through cycling to work. They may also be realised by walking to work, though this activity level may not reach the

necessary level of intensity of activity for some groups (e.g. young adults). However, the greatest health benefits are gained by increasing activity levels in elderly, sedentary or obese individuals, and walking will have major benefits for such groups. The risks from cycling and walking (musculoskeletal injuries and accidents) are less significant than the benefits obtained.

Problems in providing reliable quantitative estimates relate to the lack of data on the linearity of functions and the presence or absence of thresholds. Moreover, the effects on modal shift and travel patterns in response to specific projects or policies are not well characterised. Finally, there are also important questions about how to assess background health status and activity levels.

Community Severance and Other Effects

As stated earlier in this report, this study explored the direct effects of transport on health. It did not set out to assess the significant benefits transport has on health (indirectly) through access to goods and services, and through economic and social development. These benefits are extremely important and must be included when looking at transport more generally. There are, however, a number of other effects that arise directly from transport, which have not been covered in previous sections.

The first of these is community severance, which arises when roads bearing high levels of traffic cut through housing areas. The physical presence of the traffic, particularly heavy goods vehicles, as well as the risk of accidents, presents a barrier to the community, limiting or disrupting interpersonal networks and reducing social contact. There is evidence regarding likely health effects of community severance, though the effect is indirect. Studies indicate social contact may be inversely proportional to the volume of traffic. These social factors in turn may influence disease causation. Firstly, deleterious factors cause stress which increases susceptibility to disease. Secondly, social support may be restricted, which is believed to have a moderating effect in dealing with noxious stimuli.

There is evidence which indicates that social contact does result in lower all cause mortality. However, translating these studies into quantitative frameworks is extremely difficult because it is hard to isolate a link between transport activity and the degree of community severance that occurs. Some qualitative estimates are possible based on traffic volume, though it is stressed there is a high degree of site specificity with effects.

The possible inequality effects of other impacts (accidents, air pollution, noise and physical activity) have also been assessed. For all of these categories, disproportional effects have been identified for vulnerable groups. Of these the relationships between income/poverty and air pollution, and the fear/risk of vulnerable groups (cyclists, children) in terms of traffic accidents are highlighted as most important. For both of these quantification of effects may be possible. Finally, a number of other categories of effects have been reviewed. These include transport access and social or economic exclusion and the potential stress and psychological effects caused by transport. For both of these we conclude that effects on health are likely to be low relative to other impact categories, and that at present, quantification is not possible.

The Relative Effects of Transport on Health

One of aims of this initial phase was to assess the relative importance of different health effects of transport in London, and to illustrate where evidence existed and where quantification could be undertaken. By doing so we have aimed to provide some feel for how important transport is with respect to health.

For accidents, statistics are available on the current health effects from transport in Greater London. The latest figures report that there were 45,547 road accident casualties in London in 1998. Of these, 226 were fatalities, 6,632 were serious and 38,689 were minor.

Comparing these against the effects of air pollution is interesting. Using the framework outlined here, we have estimated the current levels of air pollution related effects from all transport in London. We estimate 380 fatalities (deaths brought forward) and 350 respiratory hospital admissions per year occur in London from transport-related pollution (excluding ozone). Interestingly the number of fatalities is of a similar order of magnitude to the numbers of deaths in London from traffic accidents, though it is stressed there are important differences in the age and health-state of people affected by the two. Many of the deaths associated with pollution are probably in the elderly and the sick and the period of life lost may be small. The attribution of causality is also far more certain for accidents than for air pollution. In addition, there are also estimated to be an additional 815 cardio-vascular/ cerebro-vascular hospital admissions and half a million minor respiratory symptoms from transport-related air pollution in London each year, though a slightly higher uncertainty rating is attached to these numbers. Air pollution in London is also thought to lead to changes in life expectancy (chronic mortality). We estimate that transport-related air pollution leads to the loss of around 34 thousand years of life per year, but note there is a higher uncertainty in this value than for the endpoints above. Finally, there are a number of other possible impacts that have been reported in US studies. The use of these estimates leads to additional health impacts from transport emissions in London including a small number of deaths (around 30 per year) from carcinogenic emissions, and a very large number (half a million) from respiratory symptoms. These effects are given the highest uncertainty rating. Overall, these values indicate transport-related air pollution is as important (perhaps more so) than accidents with respect to the health of Londoners.

Moreover, the combined health impacts from accidents and transport-related air pollution are estimated here to be responsible for at least 1% of annual deaths in London. They are also responsible for thousands of serious health effects / injuries and tens of thousand (possibly hundreds of thousands) of minor health effects / injuries each year. However, these impacts should be evaluated against the significant benefit transport produces.

The study has also looked at the potential benefits from transport-related physical activity in London. Further work is needed to equate risks in equivalent terms to air pollution-related health effects and accidents. Nonetheless, initial calculations indicate the benefits of physical activity from the current numbers of people cycling or walking to work in London could be of a similar order to the dis-benefits from accidents or air pollution (i.e. in terms of the life expectancy gained per year for London). We highlight the derivation of more accurate versions of these numbers as one of the immediate research priorities following this study. The relevant numbers of people affected by health impacts from noise and severance are harder to evaluate. Data on noise levels across London are not currently available. Nonetheless, it can be concluded that a very large number of people in London (> several hundred thousand) are exposed to noise levels above the WHO environmental guidelines, a threshold below which few people are seriously annoyed. For both noise and community

severance, potential health impacts occur as a secondary consequence of transport activity. Initial estimates might be possible, especially for noise, when applied for the evaluation of specific schemes or policies (rather than generically for London as for other categories).

Research Recommendations

There are a number of different categories of research recommendations that arise from a study such as this. The first centre on the primary research required to improve our understanding of the links between transport and health. Many of these are longer-term research priorities. The second concern the more immediate or short-term priorities. Even though an area can be investigated in detail with a research programme, in many cases some additional input is needed to the debate now, to help inform decision makers in the short term. Areas identified, by impact category are summarised below:

Accidents
The research recommendations from accidents are fewer than other parts of the study, as the methods of appraisal are widely accepted, and much research effort has been input into this area. Nonetheless there do remain questions about the relationships of certain key assumptions, for example the link between marginal changes in traffic volume and accident rate and severity. There are also health specific areas that warrant more research. It would be interesting to look at the effects of accidents on the NHS, both in terms of numbers (with a break down by impact type) and health costs. It would also be interesting to investigate how potentially important mental health effects (trauma) from injuries might be, as well as further studies into the fear of accidents affecting people's decision to walk or cycle more generally.

Air pollution
There is a great need for further research around air pollution. Any research recommendations here should be compared to other ongoing programmes though we highlight the issue of uncertainty analysis as a priority, as well as further work to improve the estimates of chronic effects. In addition we recommend the use of an approach to look at specific scheme and policy measures, particularly in the context of current legislation with respect to health-based local air quality standards (e.g. as part of the National Air Quality Strategy).

Noise
A number of research areas have been identified. In the area of primary health impact data research should include longitudinal field studies and natural experiments (e.g. changes in the siting of an airport) in preference to laboratory and cross-sectional studies. Appropriate study design should also consider impacts on vulnerable groups, confounding effects and effect modifiers. Studies should also take account of relevant socio-economic and political factors across different communities exposed to noise sources. Further research is required for studies that suggest that endocrine status, motivation and annoyance are affected by noise exposure in children. There is a need to establish whether these effects persist over time, or change in size, and a need to distinguish between the immediate and delayed effects of noise.

More work is needed on the appraisal of noise sources and the evaluation of mechanisms to relate to health impacts. There is also a need to improve the measurement of noise sensitivity and annoyance. A metric is required against which health effects can be assessed and measures standardised. As with air pollution, any research recommendations here should be

compared to other programmes. There are however, some immediate areas that warrant research. These include a more detailed look at data sources in London to investigate noise levels. They also include the use of frameworks to look at specific policies or schemes. This would allow the evaluation of potential exposure response functions and would allow a first order calculation of the potential importance of noise (whilst accepting that confidence in estimates may be low) and so enable an evaluation of how important an issue noise and health might be for transport.

Physical activity

The assessment of physical activity benefits comprises one of the most interesting aspects of the current study. This area is highlighted as warranting a focus for follow-on studies in general. We have highlighted the following specific areas:

• The need for a series of systematic reviews of primary studies to answer the question "to what extent can physically-active transport influence health and disease outcomes?" For cancers, this would entail updating existing meta-analyses.

• Specific work to achieve sensible activity-effect functions and relate these to frameworks. This includes derivation of baseline factors for future analytical frameworks.

• The derivation of specific numbers for London for the endpoints listed.

• The use of the numbers to look at specific policies, i.e. what benefits might you see with modal shift to cycling and walking. This could be extended to investigating policies to say how benefits can be maximised, whilst reducing the risk of detrimental effects.

• Considering the relative risks of exercise benefits against accident risk and air pollution exposure specifically for London, as well as comparing against other policies (e.g. stopping smoking). This could also be extended to look at the health benefits from displacing private car journeys – for example calculating the direct health benefits and the avoidance of impacts from air pollution, accidents, etc.

• The interaction of the promotion of exercise in the context of other dimensions of policy such as safety and public transport capacity, e.g. how much public transport capacity is taken up by trips that could equally be walking trips especially in London? Is there a risk that lower fares and improved services would actually encourage less physical activity? What additional benefits are there in targeting the elderly for whom benefits may be greater to keep them mobile?

Community severance, other effects and inequality

A number of research gaps have been identified:

• An agreed measuring tool to assess community severance and use of this tool to evaluate background levels of community severance and to be able to model the effects of transport proposals on community severance;

• Research to quantify the effects of community severance on a range of health outcomes, including both morbidity and well-being, especially with respect to transport severance.

• Further research into the health effects of access and comparison with wider benefits offered by transport.

• Further studies into the mental health effects from transport, with respect to annoyance, frustration and anxiety from delays and congestion, and from fear of accidents.

• Quantification of the size of inequality effects on other impact categories, especially associations between poverty and levels of air pollution / accident risk to vulnerable groups.

From our discussions with numerous people during the course of the project, the one dominant aspect raised has been the need to focus the project by looking at the quantification

frameworks in relation to actual schemes or policies, i.e. looking at marginal changes in transport.

It is essential that the techniques be developed with the help of a few, well chosen, specific policy proposals. Until that is done the study here will only be a review of the existing knowledge of generic relationships – a valuable thing to have, but a long way from an operational evaluation framework or a useful tool for decision making.

This is particularly important for some effects, such as physical activity, which do not lend themselves to generic quantification. These effects tend to be highly site-specific, and so can only be put properly into context by looking at specific examples. We believe there are three areas of marginal changes that are a priority for investigation:

- Assessment of health effects at the transport scheme level,
- Assessment of transport effects on health from London wide policies,
- Assessment of health effects from NHS decisions that have an impact on transport. This is important given the NHS is one of the largest employers in London and has a major impact on transport provision and activity levels.

During the course of our discussions, a number of possible areas of investigation have been identified. These include

- Congestion charging;
- Speed reduction policies;
- Home zones;
- Pricing and fare structures, including road pricing;
- New public transport links;
- New access roads for re-generation (stronger links to network, east-Thames river crossing);
- NHS related effects, from say hospital closure or for green transport plans.

Finally, we have not considered the very large health benefits that transport has within this study, i.e. the indirect effects of transport. We highlight this as a major omission, but one that lay outside of the original remit.

Quantification Frameworks

The evidence in the study shows that transport may have important health effects through all of the categories assessed. These are summarised in the table below.

	+ = Beneficial effect		– = Detrimental effect.		
Endpoint	Accidents	Air pollution	Noise	Physical activity	Community severance
Physical injury	---			-	
Respiratory & cardio-vascular disease		---		+++	
Cancer		-		++	
Mental health/well being	-	-	--	+	--
Diabetes, obesity, osteoporosis				++	

Key, -/+ low, --/++ medium, ---/+++ high effect.

At present we conclude that it is not possible to quantify all the health impacts of transport with similar confidence. There is considerable uncertainty associated with many of the impacts we have addressed. This uncertainty is relevant to the effect itself (i.e. is it real?), as well as with respect to the reliability of quantification. It is worth noting however that impacts from different transport modes do vary. These are summarised below. This has important consequences for choices over modes and policies.

	+ = Beneficial effect		– = Detrimental effect.		
	Accidents	Air pollution	Noise	Physical activity	Severance
Air transport	-	--	---		
Road – motorised	---	---	---		---
Road – non-motorised	-			+++	
Rail	-	--	--		-
Underground	-	-	-		
River	-	-	-		

Key, -/+ low, --/++ medium, ---/+++ high effect.

A summary of the quantification framework for each impact category is presented below. For those areas which have been more intensively studied, and for which more summary information exists, we have been able to summarise information from key review studies and provide quantification methods. For the more uncertain aspects underlying literature has been explored. The table shows that there is still uncertainty over many areas. Moreover, people hold very different views about the causality for different effects and the area of transport and health remains a controversial one.

Overall, we conclude that it is possible to evaluate the health effects of accidents and air pollution, though stress that the uncertainty associated with the latter is higher and the consensus on effects lower. Frameworks exist for both categories to assess the marginal

effects of transport, though the analysis for air pollution is complex. It is also likely that the health benefits of cycling and walking can be quantified, though further work is needed to provide quantification methods that fit conventional frameworks.

It is possible to assess quantitatively the noise levels from transport, though it is very difficult to evaluate quantitatively what the health consequences of these levels are. A qualitative approach could be undertaken, though there remains considerable debate around the reliability of evidence relating to health effects. Finally, the evidence and assessment methods for other direct effects from transport are less well characterised, though it may be possible to qualitatively assess the potential health effects of community severance.

Category	Effect	Certainty of impact	Certainty and approach for quantification
Accidents	Injury	High	High Direct cause and effect based on historic rates.
Air Pollution	Respiratory and cardio vascular mortality and morbidity	Medium	Medium. Quantification possible through 1 Assessment of effects of traffic on air quality 2 Assessment of health impacts with exposure-response functions Debate on which health endpoints and which functions should be used.
Noise	Indirect through annoyance and sleep disturbance to well-being, mental health and mortality	Low	Low. Quantification **potentially** possible through 1 Assessment of effects of traffic on noise levels 2 Assessment of health impacts with exposure-response functions Many endpoints are secondary and are difficult to quantify. Questions over functions and how they relate to noise specifically. Issues of perception, sensitivity of individuals, thresholds, non-linearity.
Physical activity	Cardio-vascular, diabetes, cancer, (**beneficial**)	Medium – High	Low. Quantification **potentially** possible through 1 Assessment of benefits of physical activity 2 Assessment of background levels/confounders 3 Estimate changes in likely physical activity from transport policy or scheme Questions over linearity and threshold with functions. Some issues relating traffic activity to levels of effects.
Community Severance		Low	Low. Many endpoints are secondary and as such difficult to quantify, especially in relation to transport activity. Overall difficulty in linking traffic activity and levels of impact.

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1 Introduction

This study was commissioned by the NHS executive to investigate frameworks for Health Impact Assessment (HIA) for transport. The objectives of the study were to evaluate the **direct** relationships between transport and health in London and consider whether it is possible to use these for quantification. In the context of this study, health is not restricted to biomedical effects, but also includes a broad view of health as a state of physical, mental and social well-being.

The direct health effects of transport included in the study are:

- Traffic accidents;
- Air pollution related health effects from transport emissions;
- Noise related health effects from transport activity;
- Health benefits from physical activity from cycling and walking;
- Community severance, mental health and inequality effects.

The study aimed to cover only direct transport-specific effects. It therefore does not assess the very large indirect benefits of transport, including the quality of life and health benefits transport provides through access to goods and services and from economic and social development. We stress however that consideration of these indirect effects is essential when applying HIA to transport, or indeed in transport policy more generally.

The study has assessed the literature to examine positive and negative relationships between transport and health for each of the five categories listed above. Within this study, the aim has not been to assess the underlying studies, but instead to undertake rapid reviews, and from this (where possible) to propose relationships. For each category, the chapters have evaluated possible ways for incorporating and applying these relationships. Finally, in undertaking these studies we have tried to take a London specific perspective, in recognition of the important differences in transport in London relative to other areas of the UK.

1.1 Health Impact Assessment and Transport Appraisal

Health Impact Assessment is used to understand the potential health risks and benefits of proposals. '*HIA offers a systematic and democratic way of combining quantitative and qualitative evidence to review the effects on health of any policy, programme or project, and to submit recommendations to the relevant decisions maker. The approach provides an inclusive framework for examining patterns of potential health gains and losses*.' (Ison, 2000).

The core steps in HIA are:

- Screening;
- Scoping;
- Appraisal of the potential health effects/impacts;
- Decision-making;
- Monitoring and evaluation.

The current study aims to provide information that may be of use for a number of these steps. These are shown in the Figure below. It aims to provide information on the evidence for the direct health effects of transport, which is important during *screening*. The information in this study should show how important direct health effects from transport are (in total and by category), and so can help in judgements when using HIA as to whether potential health effects from transport are negligible or need more investigation.

It also provides important information for *appraisal*, which assesses a proposal's effect on the health of the population. The study aims to provide information that can be used in the identification and characterisation of health effects and potential changes in health risk. The information here should allow the evaluation of the nature of effects, their size and severity, and the time and likelihood of occurrence. Where possible, details are presented to allow quantification. We have also set out to assess when effects occur dis-proportionately in vulnerable, disadvantaged or marginalised groups, consistent with HIA.

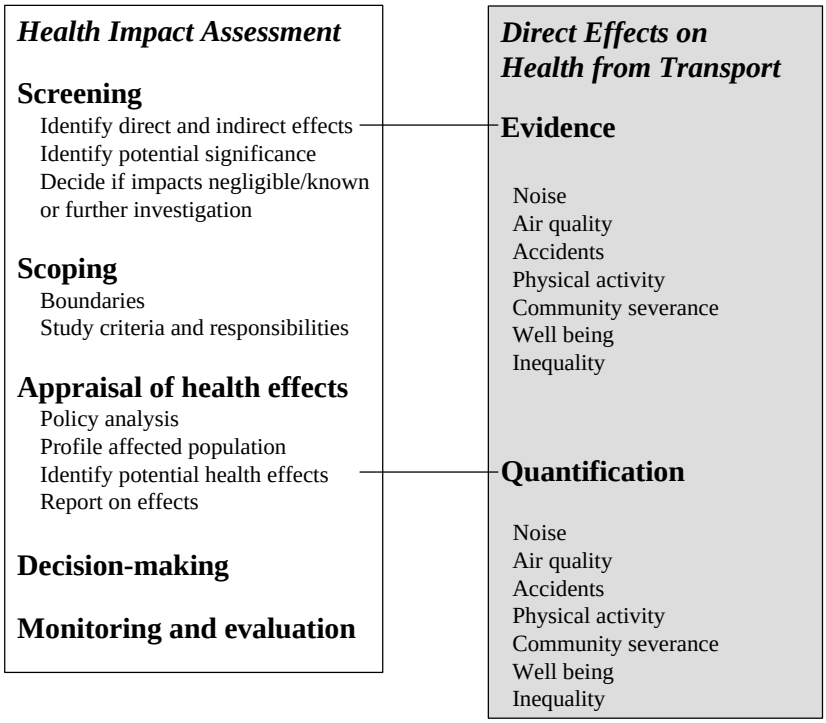

Figure 1.1 The input from the current study into HIA

The application of health impact assessment to transport is considerable and is increasing (see Isson, 2000). In undertaking this study however, we have however attempted to consider transport in a wider context than for Health Impact Assessment alone. There are many inter-connections between transport and health, as shown in the figure below (for example the role of transport in improving access, or employment and economic benefits, and for both the wider health benefits these lead to). These effects also extend beyond a purely health based assessment.

The full range of effects from transport policies, programmes and projects have traditionally been assessed using specific appraisal techniques. These techniques aim to cover the direct and indirect effects of transport across all criteria, and as such assess health alongside other

impact categories such economic costs, congestion, etc. The criteria included in transport appraisal are also shown in the Figure below (with criteria taken from the DETR's New Guide to Appraisal).

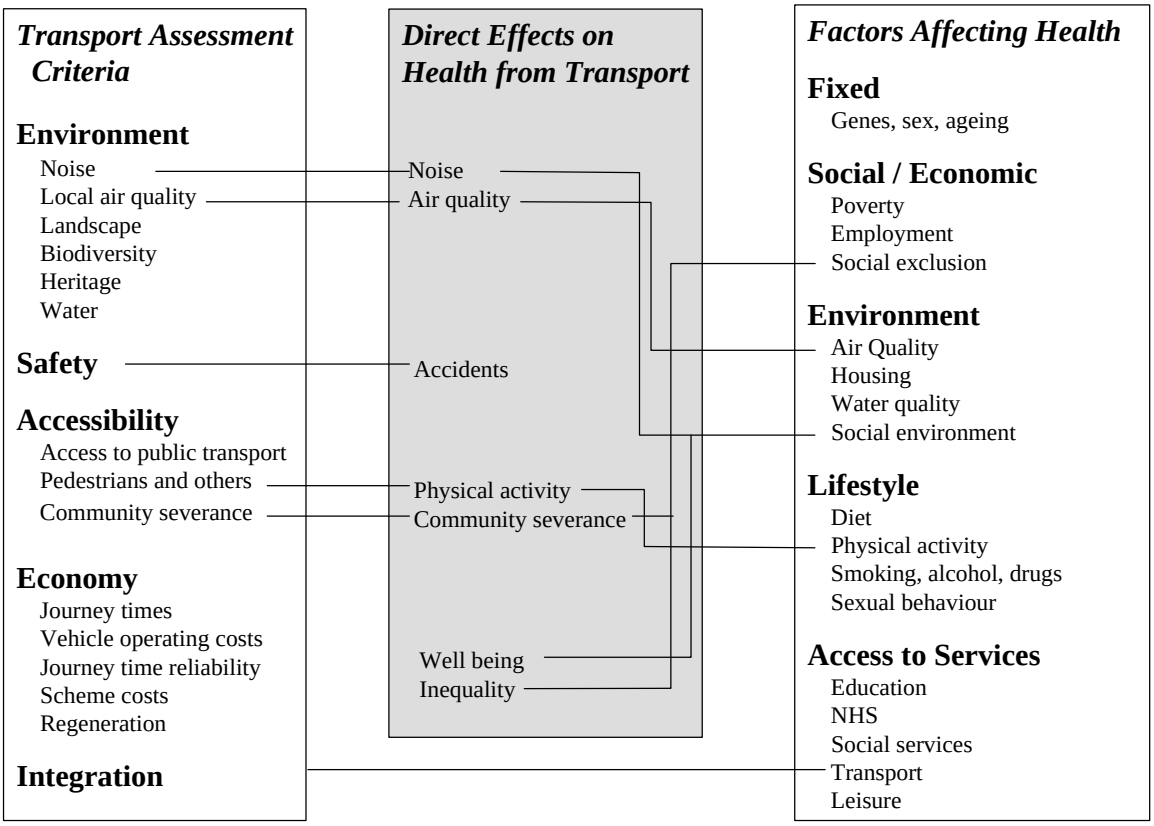

Figure 1.2 The connections between the current study, transport appraisal, and factors affecting health generally.

We stress that Health Impact Assessment **should not** be used as an alternative to existing appraisal for setting transport policy, as it only considers a sub-sector of the wider criteria involved. It can, however, be used alongside traditional transport appraisal to provide a better understanding of the health consequences of transport. By doing so it may be able to help improve the health aspects of transport policies, programmes or plans, as long as these are balanced against effects on other categories (environment, economy, etc.).

Because of this, it is important within this study for the existing transport appraisal techniques to be taken into account, in terms of the criteria used, the outputs, etc. This is important if the use of HIA is to be relevant to transport decision-makers and to ensure that the information from a HIA can be evaluated alongside existing criteria.

Finally, it is stressed that this research project is the first of a two part study. This stage provides details of the evidence and evaluates whether quantification is possible. To fit with the scoping goal of HIA, it has assessed the relative size of the health effects for different categories from transport in London, to provide some guidance on how important these effects are relative to each other and in the context of other factors affecting health. One of the key aims of this phase of the work has been to make explicit where impacts can be

quantified and where this is problematic: by doing so we hope to identify gaps in the knowledge and highlight research priorities. This is particularly important given the uncertainty for some of the impacts.

Within this first phase it is impossible to recommend one generic approach and framework that is universally applicable for researchers and decision-makers in health assessment and in transport appraisal, and one that can be applied to detailed schemes and at the same time to London wide policies. What this study hopes to achieve is to provide evidence and relationships which can be applied to these specific applications, once combined with the relevant level of detail and incorporated in the frameworks representative of the specific type of assessment.

The second phase of the work, to follow on from this study, is to use this information for looking at the **marginal effects** of changes in such frameworks to assess the health effects of new transport policies, schemes, or measures.

1.2 References

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2 Accidents

2.1 Introduction

The most obvious and one of the largest impacts of transport on health are traffic accidents. Quantification of accidents from transport are already included in transport policy and scheme assessments and are usually based on historical relationships between transport activity and accident rates. These approaches are well established and have agreed methodologies (unlike other categories of impact). Therefore this chapter only presents a brief summary of these issues, along with details of the levels of current accidents specifically for London.

2.2 The Evidence for Health Effects

The impacts of transport on health from accidents are obvious; the cause and effect chain is simple and immediate. The assessment of accidents is therefore easier than with other categories considered in this study.

2.3 Quantification

Accidents can be quantified using statistics relating to levels and types of transport activity. It is important when looking at statistics to differentiate between numbers of accidents and numbers of casualties – this is important here as we are concerned with health impacts (casualties) rather than accidents (and for example, an accident can have more than one casualty).

Accidents and casualties are classified by severity (fatal, serious or slight). With respect to accidents the severity is defined by the severity of the most severely injured casualty (fatal, seriously injured or slightly injured).

There are well-established quantification methodologies for predicting transport accidents and casualties. These methods use historical accident data as a means of predicting future accident rates from new schemes or policies. The most appropriate accident or casualty value figures to use will depend on the amount of information available for the scheme under review. This means that different approaches may be used for general London wide policies and specific scheme assessments. Methodologies for quantifying accidents in road appraisal (e.g. for new schemes) are given in DETR's COBA Manual (Design Manual for Roads and Bridges, Volume 13, Section 1, Part 2, Chapters 3, 4 and 5).

The approach predicts traffic flows with and without the road scheme under evaluation (see the *Traffic Appraisal Manual – TAM* [1]). Accidents rates (and costs) are predicted by individual link and junction type, using the approach in the manual, and for brevity, these are not repeated here. Note the overall approach to Road Appraisal has recently been updated (DETR's New Approach to Appraisal), however, the guidance (DETR, 2000a) still recommends the use of the standard methodology for calculating the number of accidents and casualties as set out in Volume 13 of the DMRB. This uses average accident severity split (i.e. the number of fatal, serious and slight casualties per accident) for a particular

[1] Traffic Appraisal Manual, Design Manual for Roads and Bridges, Volume 12, Highways Agency

road/junction classification which in turn is used to determine overall accident costs.[2] The number of accidents on a given length of road is expressed as an accident rate, defined as so many 'Personal Injury Accidents per million vehicle kilometres'.

Apart from length and flow level, in *COBA* there are two determinants of the number of accidents: the number and type of junctions and the type of links. *COBA* incorporates a method of separating out the effects of links and junctions on accidents. For links, local data can be obtained from the appropriate police or local authority and should normally cover the five years previous to the assessment and must cover at least three years. The method then calculates the local accident rate (accidents per million vehicle kilometres) for each link. For existing links where local accident data are not available and for new links, *COBA* uses default accident rates with average casualties per accident. Note there are 15 accident types relating to the types of roads. The accident types are further subdivided by speed limit. For existing junctions, the use of local accident data is recommended, with default values being used in the absence of such data. The classification of junctions for accident assessment distinguishes three broad categories, i.e. major/minor, signals and roundabouts (subdivided into standard, small and mini). In total there are 96 junction types in COBA, for which default model data are given in DMRB (1997), including accident costs and casualty rates by severity.

2.4 Levels of Accidents in London

Accident statistics are collected and reported separately for London (i.e. for Greater London – the 32 London boroughs and the city of London). Details of the most recent data are reported by London Research Centre's (now Greater London Authority) London Accident Analysis Unit (LRC, 1999). This data is presented below.

2.4.1 Numbers of Accidents in London

In 1998 there were 38,156 personal injury accidents reported to the Police in the Greater London area. This was a reduction of 1.4% from 1997, though total distances travelled also decreased by 1.2% between 1997 and 1998. The accidents and casualties in Greater London area accounted for 16% and 14% of all those in Great Britain in 1998. The breakdown by mode and severity are shown in the Table below.

Table 2.1. Casualties in London by mode in 1998.

	Casualties in 1998							
Mode	**Fatal**	*%*	**Serious**	*%*	**Slight**	*%*	**Total**	**%**
Pedestrians	118	*52.2*	1936	*29.2*	6978	*18.0*	9032	19.8
Pedal cycle	12	*5.3*	602	*9.1*	3701	*9.6*	4315	9.4
Motor cycle	36	*15.9*	981	*14.8*	5825	*15.1*	6842	15.0
Car occupants	53	*23.5*	2645	*39.9*	18631	*48.2*	21329	46.8
Taxi occupants	0	*0.0*	40	*0.6*	433	*1.1*	473	1.0
Bus or coach occupants	3	*1.3*	262	*4.0*	1973	*5.1*	2238	4.9
Goods vehicle occupants	3	*1.3*	144	*2.2*	982	*2.5*	1129	2.4
Other vehicle occupants	1	*0.4*	22	*0.3*	166	*0.4*	189	0.4
Total	**226**	*100*	**6632**	*100*	**38689**	*100*	**45547**	

[2] Information on the severity split by class of road (links and junctions) is detailed in DMRB (1997), Section 1, Part 2, Chapters 4 and 5.

%	0.50	14.56	84.94

Source: LRC (1999).

These show that in terms of absolute numbers, most accidents were associated with cars, pedestrians and motor cycles, and that most accidents had a low severity. As a proportion, the percentage of fatalities in the statistics for London (0.5%) is lower than for the UK (1%), though this is expected given the lower average speeds in London. Looking at the London data by mode, it can be seen that severity rates vary by road user. The proportion of fatal and serious accidents is much higher for pedestrians than any other category.

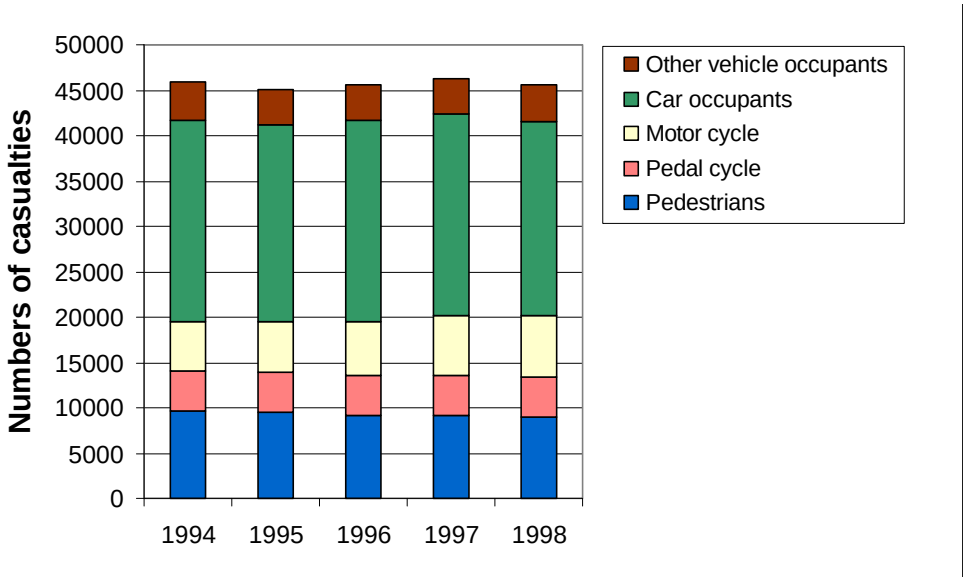
Table 2.2. Severity of Injury (%) in London by mode in 1998.

Mode	**Fatal (%)**	**Serious (%)**	**Slight (%)**
Pedestrians	1.3	21.4	77.3
Pedal cycle	0.3	14.0	85.8
Motor cycle	0.5	14.3	85.1
Car occupants	0.2	12.4	87.4
Taxi occupants	0.0	8.5	91.5
Bus or coach occupants	0.1	11.7	88.2
Goods vehicle occupants	0.3	12.8	87.0
Other vehicle occupants	0.5	11.6	87.8

It is also interesting to examine how these accidents rates have changed over time. The table below shows data over the past five years. Shown in the figure below, it can be seen that overall casualties from road traffic in London have remained fairly constant over time.

Table 2.3. Casualties in London by mode 1994-8.

	Casualties in Greater London					
Mode	**1994**	**1995**	**1996**	**1997**	**1998**	**Average**
Pedestrians	9,618	9,415	9,217	9,174	9,032	9,291
Pedal cycle	4,427	4,539	4,350	4,430	4,315	4,412
Motor cycle	5,531	5,482	5,959	6,548	6,842	6,072
Car occupants	22,066	21,669	22,130	22,214	21,329	21,882
Other vehicle occupants	4,233	3,994	3,944	3,909	4,029	4,022
Total	**45,875**	**45,099**	**45,600**	**46,275**	**45,547**	**45,679**



There was a slight increase in accident levels from 1995 through 1996 and 1997 (as discussed in Glaister et al, 1999) although this trend now seems to be reversing (though note total km travelled also decreased). Even so, the fact that the numbers of casualties in London remain broadly the same, year after year, whilst numbers for the rest of the UK decline is still an issue of major concern.

Data on accident rates per km are given below. Data is also available split by road type (as different roads have different accident rates and severity - this partly a function of speed [at different speeds the numbers and severity of accidents changes] but also a function of road design, surface, etc). Data is also available broken down by local authority area.

Table 2.4 Accident rates per kilometre in London

	Accident rates per km			
	Fatal	**Serious**	**Slight**	**All accidents**
All	0.02	0.47	2.49	2.98
Pedestrian	0.01	0.15	0.52	0.68
Pedal cycle	< 0.01	0.05	0.3	0.34
Motorcycle	< 0.01	0.09	0.45	0.54
Car	0.01	0.39	2.07	2.47
Taxi	< 0.01	0.01	0.06	0.07
Goods vehicle	< 0.01	0.05	0.28	0.33
Bus and coach	< 0.01	0.03	0.18	0.21

Accident rates per km are highest for cars as these values are not adjusted for numbers of vehicles. When this is done (Glaister et al, 1999) the relative risk factors show that the risk of serious injuries or fatalities are greatest for pedestrians, cyclists and motor-cycles.

	Risk by distance	Risk by journey	Risk by Time
Car	1	1	1
Foot	16	13	1
Bicycle	22	6	16
Motor cycle	35	30	31

Source: Glaister et al, 1999.

2.4.2 Non-Road Accidents

Data on rail statistics are given in UK Statistics - Transport Statistics Great Britain (DETR, 1999). There is a difficulty in separating out transport trips that start or finish in London (e.g. intercity trips) from London specific or London commuter journeys and so values are not presented here. Specific statistics for the Underground are held by London Transport.

Transport activity (billion tonne kilometres) on the Thames is recorded in National Statistics (DETR, 1999), though the split by London is not separated. Data on recreational and passenger trips are likely to exist (e.g. from Highways Agency) though were not investigated for this study. Accident statistics are also registered from all marine accidents (and for example include the 'Marchioness' disaster) though data are not dis-aggregated enough to allow a proper comparison by km with other modes of the transport in London.

Accidents for aircraft (for UK registered aircraft in the UK airspace) are given in National Statistics (DETR, 1999). These statistics are not relevant in the London context (as aircraft are not a local transport option). There is, however, another factor which is potentially more important. That of major airline accidents – given London's exceptionally high population density, any aircraft that did crash within Greater London could have a very large impact. The assessment of airline risk is an important one, but one that lies beyond the boundaries of this study.

Methods for looking at accidents with respect to other modes are given in the new DETR Guidance on the Methodology for Multi-Modal Studies (DETR, 2000b)

2.5 Other Issues and Uncertainty

The details above provide a simplified approach to the assessment of transport accidents. In practice there are many issues which are still not well understood. Central to these are the issues of marginal changes in traffic flows. The relationships between traffic volume, traffic speeds, accident rates and accident severity are complex and the evidence is uncertain. Most frameworks assume that as transport volume increase, so do accident rates. However, there are problems: people adjust their driving behaviour with traffic and so may alter speeds (which in turn affect accident rates and severity) or take more care, which may lower rates.

There are other issues for non-motorised road users. Pedestrians and cyclists have higher levels of fatal and serious casualties from collisions with other road users. There are also links between modal choice and both social class and vulnerable groups, particularly children; for example 30% of all pedestrian casualties in London are for 0-16 year olds (LRC, 1998). These issues are discussed in more detail in Chapter 6.

A number of other issues are raised here for completeness. It is generally assumed that transport by public transport is safer than by private car. The statistics bear this out. However, there is another factor to consider. People have to get to central public transport nodes in order to take public transport – usually by walking or cycling. It is important that these additional components of the transport trip are taken into account, especially in light of the different accident rates and severity classes associated with walking and cycling.

There are some questions that remain on how representative accident statistics are, especially for minor accidents which may be unreported – especially for cyclist. Finally, there are some other possible health related effects that we have not been able to consider here, but which may be important. Fear of accidents may reduce the willingness to cycle or walk. There are also possible mental health effects (trauma) from injuries.

2.6 Future Research Recommendations

The research recommendations from accidents are lower than other parts of the study, as the methods of appraisal are widely accepted, and much research effort has been input into this area. Nonetheless there do remain questions about the relationships of certain key assumptions, for example the link between marginal changes in traffic volume and accident rate and severity. There are also health specific areas that warrant more research. It would be interesting to look at the effects of accidents on the NHS, both in terms of numbers (with a break down by impact type) and health costs. It would also be interesting to investigate how potentially important mental health effects (trauma) from injuries might be, as well as further studies into the fear of accidents affecting people's decision to walk or cycle more generally.

2.7 Conclusions

The most obvious and one of the largest effects of transport on health are traffic accidents. Quantification of accidents from transport is already included in current transport policy and scheme assessments and is usually based on historical relationships between levels/types of transport activity and accident rates. These approaches are well established and have agreed methodologies (unlike other categories of impact).

The impacts of transport on health from accidents are obvious; the cause and effect chain is simple and immediate. The assessment of accidents is therefore easier than with other categories considered in this study. There are well-established quantification methodologies for predicting transport accidents and casualties. These methods use historical accident data as a means of predicting future accident rates from new schemes or policies, adjusting for road type and speed, as these influence the numbers and severity of accidents.

The chapter also reports on the current levels of health impacts from road transport accidents in London. In 1998 there were 226 fatalities, 6,600 serious injuries and almost 40,000 slight injuries in Greater London. The relative risk by mode shows that pedestrians, cyclists and motor-cyclists have a greater risk of serious injury or death by distance and by journey (indeed of the 226 fatalities in 1998, more than half were pedestrians). Accident rates in London remain of concern, as they are relatively constant year on year, whilst rates in the rest of the country decline.

The research recommendations from accidents are lower than other parts of the study, as the methods of appraisal are widely accepted, and much research effort has been input into this area. Nonetheless there do remain questions about the relationships of certain key assumptions, for example the link between marginal changes in traffic volume and accident rate and severity. There are also health specific areas that warrant more research. It would be interesting to look at the effects of accidents on the NHS, both in terms of numbers (with a break down by impact type) and health costs. It would also be interesting to investigate how potentially important mental health effects (trauma) from injuries might be, as well as further studies into the fear of accidents affecting people's decision to walk or cycle more generally.

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3 Air Pollution

3.1 Introduction

Studies of pollution episodes (such as the London smog episodes of the 1950s) have shown that very high levels of ambient air pollution are associated with strong increases in adverse health effects. Recent studies also reveal smaller increases in adverse health effects at the current levels of ambient air pollution typically present in urban areas. These health effects include a range of endpoints, such as premature mortality (deaths brought forward), respiratory and cardio-vascular hospital admissions, and possibly exacerbation of asthma, other respiratory symptoms and loss of lung function. The evidence for these effects is strongest for the pollutants PM_{10}, SO_2 and ozone and the relationships are widely accepted as causal. Recent studies also suggest that long-term exposure to these pollutants, especially particles, may also damage health and that these effects may be substantially greater than the acute effects described above.

Transport is a major source of these atmospheric pollutants in urban areas and therefore can be assumed to have adverse health effects. This chapter provides a rapid review of which pollutants and which effects there is strongest evidence for. It reports on major studies which have proposed quantification frameworks for linking health impacts to air pollution concentrations, and summarises how important these effects might be in the context of London.

3.2 The Evidence for the Health Impacts of Transport-Related Air Pollution

It is now widely accepted that transport related emissions are associated with short-term health effects at the concentrations found in UK cities. There is also a broad consensus that the effects of these pollutants on health can be quantified using exposure-response relationships based on epidemiological studies linking pollution concentrations or increments to levels of health effects.

What is more open to debate is which health endpoints should be included in quantification and which exposure-response relationships should be used for quantification. In looking at the large body of literature in this area and in trying to make judgements on which functions to use, a number of important issues need to be considered, including:

- What ambient air pollutants are associated with adverse health effects (acute or chronic), and for what specific endpoints?
- Which of these associations may reasonably be interpreted as causal and are the associations due to correlations with other measured or unmeasured pollutants?
- Where causality is accepted for a pollutant-impact pathway, what individual study or meta-analysis gives a representative value for quantification of that effect?
- How, if at all, should the exposure-response (E-R) functions from individual studies or meta-analyses be adapted for use? and
- What combination or set of quantifiable E-R functions provides a suitable basis for quantifying the public health effects of incremental air pollution from transport?

Judgements at all of these stages are the focus of debate currently among scientists and policy makers concerned with the health effects of air pollution. In this brief review we have tried to identify, though not discuss in detail, the most important aspects of that debate.

Given the remit of this study, we have not aimed to review the underlying epidemiological studies concerning pollutants and endpoints. Instead this work concentrates on existing reviews of the literature which have looked at the underlying studies and made judgements on the above list of question. In doing so, we have looked at the recommendations from two major bodies of work:

- The UK Department of Health's COMEAP group,
- The EC's ExternE Study.

We have also reported on the specific epidemiological studies undertaken in recent years for London.

It should be noted that other review work does exist (e.g. the French Swiss study, WHO guidance, US EPA reviews). The authors have considered this material as part of other work. There is a considerable overlap between the various studies in the sets of impact pathways quantified, the associated E-R functions proposed, and the conventions used in health impact estimation. To some extent this reflects independent assessment of the evidence. However, the influence of the American epidemiologist Bart Ostro on several of these quantification studies should also be acknowledged. It is likely that steps to increase this consistency between studies will be put in place, under the auspices of WHO, in the coming years.

However, in the context of the present study, and bearing in mind the limited resources available to carry it out, the reviews above were considered as giving a sufficient guidance to health impacts, especially when considered along with the results of London specific epidemiological studies.

3.2.1 COMEAP, ExternE and London specific studies

The two studies (COMEAP and ExternE) approach the issue of quantification with very different remits, and so provide very different conclusions.

The UK's COMEAP (Committee on the Medical Effects of Air Pollutants) was asked to review the relationships between air pollution and health and provide relationships for quantification in the UK. The group's objectives (COMEAP, 1998) were to provide 'exposure-response relationships (coefficients) which, in the view of the group, could be applied in the UK with reasonable confidence'.

The functions recommend by COMEAP are based on the results of time-series studies. These are relationships between daily levels of pollutants and the risk of adverse health effects, on the same or subsequent days, adjusting for weather and other factors.

Functions were provided for particles, sulphur dioxide and ozone for deaths brought forward and respiratory hospital admissions. The group did not provide functions of NO_2 in view of the difficulties and doubts about the relationships between exposure to NO_2 and effects on health, though a possible relationship for the effects of the pollutant on respiratory hospital admissions was included. The lack of UK studies and uncertainties about the independent effect of CO led COMEAP not to estimate the effects of CO.

In contrast, the aim of ExternE was to quantify as fully as practicable, while acknowledging that (sometimes major) uncertainties existed. A set of exposure-response (E-R) functions for acute and for chronic health effects of air pollution was provided, based on a review of the extensive epidemiological literature on the health effects of ambient air pollutants, and other evidence (e.g. toxicology, human chamber studies). Functions for estimating increased mortality due to primary particles, nitrates, sulphates, SO_2, ozone, benzene and diesel particles were included. For particles this also extended to chronic (long-term) effects. Morbidity effects considered include endpoints such as congestive heart failure due to particles and CO, respiratory hospital admissions due to particles, SO_2 and ozone, non-fatal cancer due to benzene and diesel particles.

The ExternE set of E-R functions draws heavily on European studies, but also includes US studies where good evidence of a real effect exists and there are not suitable European functions.

The two reviews form similar judgements on which pollutants are causal. They also agree closely on some functions (e.g. deaths brought forward [acute mortality], respiratory hospital admissions) for which the evidence is strongest. However, because of the additional endpoints considered in ExternE, overall the two reviews provide very different sets of functions. In effect, the COMEAP functions provide a sub-total of health effects, by only recommending quantification of two endpoints, though the confidence attached to the quantification is higher. The ExternE functions also include these effects (with slightly different functions) but they also attempt to quantify other effects where there is good evidence of a real link, though of course this means uncertainty increases. ExternE assumes greater transferability of data than COMEAP. Note, even within the approach recommended by ExternE, there may remain unmeasured endpoints.

There is also another approach that is relevant in the context of this study. That is to use results from the specific London epidemiological research studies (especially the studies by Anderson and colleagues at St. George's Hospital Medical School). These functions include many of the same endpoints as the two groups above, but also others such as cardiovascular admissions and GP consultations. These functions have the advantage of being based on London specific data, though as with any individual city, estimates might be misleading due to random variations and various sources of confounding and bias.

Which is the more correct approach? To a degree this is a question for the policy makers. To omit effects (as with COMEAP) because quantification is more uncertain will mean that these are ignored (i.e. it effectively attributes no or zero effect to them which introduces a very large error). However, if more uncertain results are to be used (as with ExternE), then this uncertainty must be taken into account and presented to the policy maker or user.

The use of London-specific studies has clear advantages of relevance, in that these should reflect the local situation more accurately with respect to the specific local pollution mix, the background incidence and health status, etc. However, even with a city as big as London, there are some advantages in embedding the London-specific results in the wider context of the international literature, thereby limiting the effect of random variations and of any sources of confounding which may have been difficult to adjust for within a single study. Also, there are no London-specific results for some pathways, including chronic mortality; and so use of the wider literature is then essential for quantification.

The discussion by pollutant is provided below. Note for more information the reader should refer to the original reports (COMEAP, 1993, 1998: the ExternE study – EC, 1995: 1999: 2000, plus individual London specific references). In practice, the present write-up draws heavily on text prepared for COMEAP and ExternE; though with substantially less detail included here.

In our view, it is helpful to have results from more than one approach, because this reduces any appearances that any one approach is best in all respects, and so leads to some evaluation of the basis for similarities and differences.

3.2.2 Particles

There is substantial epidemiological evidence of adverse acute health effects of particulate air pollution; and strong, but much less widespread, epidemiological evidence of chronic health effects.

Because particulate air pollution is a complex mixture rather than a single substance, there is a lot of diversity in how particulate air pollution is characterised in various epidemiological studies. This leads to E-R functions for particles in terms of total suspended particles (TSP); PM_{10} (inhalable particles); black smoke (BS); finer fractions such as $PM_{2.5}$ or sulphates; and other indices. It is also possible to `convert' E-R functions from one index of particles to another, using appropriate conversion factors. Such conversion is of course an approximation only. From these possibilities, E-R functions for ambient particles are generally expressed in terms of PM_{10} (see e.g. EC, 1995; COMEAP, 1998). This is because PM_{10} has been or is the basis of standards (and so of routine monitoring) in many countries and many epidemiological studies are now available in that metric.

However, the particles of interest in the present project are not typical of ambient PM_{10} generally. Rather, they arise as primary emissions from transport; or as secondary particles generated subsequently (i.e. as sulphates from SO_2, or as nitrates from NO_x). In terms of particle size, particles from primary particles and sulphates[3] lie principally, if not exclusively, within the finer, $PM_{2.5}$, fraction of PM_{10}.

Causality of acute health effects is now widely accepted, and there is a growing consensus on chronic health effects though there is no well-established mechanism of action of particulate air pollution. Furthermore, there is little evidence about the relative toxicity of various kinds of inhalable (PM_{10}) particles. There is some conjecture that, per $\mu g/m^3$, the relatively fine fractions (e.g. $PM_{2.5}$) are associated with greater risks than PM_{10} generally.

[3] Nitrates are a more complex issue. The relevant species are usually categorised in modelling studies into non-specific nitrate aerosols and ammonium nitrate. The formation of non-specific nitrate aerosols often occurs through adsorption onto existing particles. It may therefore not create incremental particle numbers. Moreover, a large proportion of the particles it adsorbs onto are in the coarse fraction (i.e. $>PM_{2.5}$ and even PM_{10}). Finally, the chemistry of these particles is likely to be different to other secondary species. This raises important questions concerning potential causality, though because it is not known if the numbers, mass, or chemistry of particles is the main reason for their health impacts, it is currently impossible to answer these . The issue is discussed in an ExternE working paper ‘atmospheric modelling note on secondary particulates, Lee, D and Watkiss, P (1999)

Other aspects of particles may also affect their toxicity. There is conjecture that insoluble particles may be more toxic (other things being equal) than soluble ones, and toxicity may be increased by, or even largely due to, transition metals adsorbed on to the particle surface.

Evidence to date on these issues is far from conclusive. However, they suggest that, per $\mu g/m^3$ increment in ambient concentrations, the most severe health effects are associated with primary particulate emissions and sulphates, and the least severe with nitrates. (Note, it may be that health effects associated with nitrates are negligible; there is little or no direct epidemiological evidence about this.) Many epidemiological studies have shown associations of sulphates with adverse health; but they do not show if the effects are a consequence of sulphates as such, whether sulphates in these studies are a surrogate for particles or other pollutants, or whether sulphates are associated with other toxic compounds.

COMEAP (1998) used PM_{10} as the metric for quantification, irrespective of the source, the physical characteristics or the composition of particles within the PM_{10} size range. This was a pragmatic decision, intended to be applied to ambient particles in the UK as a whole (or to urban areas of it), and not to particles from any specific source.

The ExternE study attempted to discriminate toxicity according to source or nature of the ambient particles, and so it adopted the following working conventions:

- Each incremental $\mu g/m^3$ of primary particles emitted during transport is treated as if had the toxicity of a $\mu g/m^3$ of $PM_{2.5}$;
- Each incremental $\mu g/m^3$ of sulphates is also treated as if it had the toxicity of $PM_{2.5}$;
- Each incremental $\mu g/m^3$ of nitrates is treated as is it had the (lower) toxicity of PM_{10}.

Discussion on developing functions as $PM_{2.5}$, rather than PM_{10}, can be found in the ExternE report (EC, 2000). In summary, the study has not used the available E-R functions for $PM_{2.5}$. Mostly, these are from North American studies, and are available for selected impact pathways only. Instead ExternE directly converts from the exposure-response functions for PM_{10}, assuming that in mass terms, $PM_{2.5}$ is about 0.6 of PM_{10}

3.2.3 Ozone

The results of the EC APHEA study have confirmed a relationship of ambient ozone with acute mortality and hospital admissions in Europe. The overall evidence strongly supports the view that the acute health effects of ozone can and should be quantified; and that the estimated health impacts from ozone should be added to those of particles.

Epidemiological studies represent the effects of daily ozone characterised in various ways; e.g. as 1-hr daily max.; as 5-hr daily average; as 8-hr. daily average; as 24-hr daily average. COMEAP presents functions in terms of the 8 hour mean ozone concentrations. ExternE expresses functions in terms of 6-hr daily average, in order to link with pollution increments expressed in that metric. Both sets of functions involved applying conversion factors to functions derived originally using different indices, based on data from some of the European APHEA-study cities (COMEAP) and other studies also (ExternE).

3.2.4 Sulphur dioxide

In earlier studies within the ExternE programme (EC, 1995), any health effects associated with SO_2 as a gas were not quantified; the evidence at that time was ambiguous. The APHEA

results have however established an association of SO_2 with acute mortality, and probably with hospital admissions also, from which E-R functions can be derived. The evidence from epidemiological studies carried out in the USA is less convincing.

It is however still unclear if these associations with SO_2 are causal. It may be that SO_2 is acting as a surrogate for other pollutants, especially fine particles (e.g. sulphates) not well quantified in the particle measurements available for study (usually, black smoke). However, in the APHEA studies the size of the apparent SO_2 effect does not depend on the background concentrations of ambient particles, suggesting that it is not a surrogate for PM. ExternE therefore uses E-R functions for SO_2 as additive to those for particles and of ozone. This is the viewpoint also taken by COMEAP (1998).

3.2.5 Carbon monoxide

Carbon monoxide is the most obvious marker in the air pollution mixture related to vehicle emissions. There is relatively little epidemiological evidence concerning CO, so that it is difficult to place in context the results from a few (well-conducted) studies that report positive associations. Those studies do provide the basis for E-R functions, but they do not give strong guidance on how representative or transferable these functions are. Specifically, whereas in many studies CO is not examined as a possibly causative pollutant, there are also well-conducted studies which do consider CO and yet do not find a CO-related effect (as the effect of CO is explained by its correlations with other primary emissions).

On present evidence, ExternE recommends that the functions for CO and acute hospital admissions for congestive heart failure be used; but that functions for CO and acute hospital admissions for ischaemic heart disease and acute mortality are only used for sensitivity analyses. In epidemiological studies the effects of CO are represented as 1-hr daily maximum and 8-hr daily average. Consequently, E-R functions expressed in terms of 1-hr max CO and 8-hr daily average have been scaled up by a factor of 2 and 1.5 respectively.

COMEAP quoted studies that showed associations between CO and deaths brought forward and cardio-vascular admissions, but did not quantify because of the problems separating CO from other components of the air pollution mixture, and because of the lack of UK studies (though there are now London specific studies). The quantification report did however acknowledge that information on CO is accumulating, that assessment may be possible comparatively soon, and that this would be likely to include hospital admissions for cardio-vascular disease and deaths brought forward.

3.2.6 Nitrogen dioxide

In early ExternE work, the epidemiological evidence regarding NO_2 was assessed. Some studies reported NO_2 effects. However, the broad thrust of the evidence then was that apparent NO_2 effects were best understood not as causal, but as NO_2 being a surrogate for some mixture of (traffic-related) pollution. We concluded that a direct effect of NO_2 should not be quantified. (Indirectly, NO_x did contribute, as a precursor to nitrates and to ozone).

The APHEA results reported positive associations between NO_2 and daily mortality or respiratory hospital admissions in several European cities. Consequently, it is possible to propose E-R functions. APHEA however also supports the view that the apparent NO_2 effects may be due to particles; or at least, are highly dependent on background particle levels.

Against this background, ExternE recommends that the E-R relationships for NO_2 are not used, except for sensitivity analyses. In the epidemiological studies from APHEA the impacts of NO_2 are described in terms of 1-hr maximum. Consequently, the E-R functions in terms of 1-hr maximum have been scaled up by a factor of 1.67 to express results in the metric of 24-hr average.

COMEAP also did not quantify NO_2 effects in view of the difficulties and doubts about the relationships between exposure to NO_2 and effects on health, though a possible relationship for the possible effects of the pollutant on respiratory hospital admissions was included.

3.2.7 Chronic effects

There are differences of views about whether longer-term exposure to ambient particles causes premature mortality in ways that are not captured by the time series studies of acute mortality. COMEAP (1995) concluded that it would be prudent to assume that the associations shown in various studies are causal. These is also some debate whether and how the impacts can be estimated using results from a small number of cohort studies carried out in the USA; and on this, COMEAP (1995, 1998) was less confident. However the effects, if real, are the dominant ones among all health endpoints considered in this study, and there have been methodological developments on using the cohort study results to estimate impacts; and so we have included estimates based on life-table methods.

With regard to the underlying epidemiological evidence, mortality effects of long-term exposure to air pollution have frequently been studied by examining relationships cross-sectionally between death rates and air pollution in cities. These studies generally reported positive associations for particles. Their major limitation lies in their ecological design; that is, aggregated measures are used for groups of individuals. This limits adjustment for confounding factors, such as smoking habits, and in other ways also complicates interpretation. Nevertheless, the ecological cross-sectional studies showed consistently positive associations between ambient particles and mortality at different times and places (Lipfert, 1994).

More recently, the relationship of mortality to air pollution has also been studied in prospective cohort studies (Abbey et al, 1991; Dockery et al, 1993; Pope et al, 1995). These studies allow adjustment for possible confounding factors (e.g. smoking habit, gender, race, socio-economic status) on an individual basis. Both Dockery et al, (1993) and Pope et al, (1995) found associations between average particle concentrations and mortality from cardiopulmonary disease, and possibly lung cancer, but not deaths from other causes. More recent results from Abbey et al. also suggest some particulate effects. Together with other evidence, these studies do suggest that longer-term exposure to ambient particles does influence premature mortality in ways unaccounted for by time series studies.

3.2.8 Discussion by health endpoint

The health endpoints and E-R functions recommended in COMEAP and ExternE are discussed below. As noted earlier, the present text draws strongly on that written for ExternE and/or COMEAP. The ExternE sets of functions are somewhat dated, with the main ExternE focus in recent years being on estimating chronic effects. However, we think that use of E-R functions from more recent studies would not lead to major differences in results.

Acute mortality
The COMEAP function provided for PM_{10} is based on the WHO recommendations. This considered 17 studies including 12 in the US, three in Europe and 2 in Latin America and provides a higher value than the European or UK studies. For SO_2, COMEAP uses a meta-analysis from the results of the APHEA study (Katsouyanni et al, 1997). For ozone, the results of APHEA (Touloumi et al, 1997) are used.

The ExternE E-R function for PM_{10} and acute mortality was calculated as the mean of coefficients from Amsterdam (Verhoeff et al, 1996) and Köln (Spix and Wichmann, 1996) which were similar to high and low estimates respectively from the APHEA study. Estimates for ozone were derived from Barcelona (Sunyer et al, 1996); for SO_2 as the mean of Athens (Touloumi et al, 1996) and London (Anderson et al, 1996). For sensitivity analyses the E-R function for NO_2 was obtained from the mean of Barcelona (Sunyer et al, 1996) and London (Anderson et al, 1996); for CO from Touloumi et al (1994) in Athens.

Note both COMEAP and ExternE highlight that although quantification of deaths brought forward is possible, they do not allow us to estimate by how long. Moreover, many of the deaths associated with pollution are probably in the elderly and the sick, hastening death, and so the period of life lost may be small.

Chronic mortality
As only ExternE provides quantification for chronic mortality, the discussion below only relates to that study. The ExternE work drew on a number of US studies for the analysis (as these are the only studies available).

The first of these, by Pope et al (1995) involved about 500,000 individuals in 151 US cities, compared with approximately 8,000 people in six cities studied by Dockery et al (1993). This difference in scale reduces chances of confounding city differences in air pollution with other between-city differences. Also, Pope et al give results that are intermediate between the higher estimates of Dockery et al and the lower risks identified by Abbey and co-workers; and so we based risk estimates on Pope et al., despite the better pollution measurements in Dockery et al.

Pope et al (1995) found that, after taking account of confounders, the adjusted mortality rate was 15%-17% higher in the most polluted areas compared to the least polluted. Risks are expressed as the % change in hazard (age-specific death rates) per $\mu g/m^3$ and are given in terms of $PM_{2.5}$ and of sulphates. These can in turn be re-expressed in terms of PM_{10} using the usual conversion factors (e.g. Dockery and Pope, 1994).

When converted to PM_{10} equivalent, i.e. expressed per $\mu g/m^3$ PM_{10}, the coefficient derived from $PM_{2.5}$ is about twice as high as that derived from sulphates. The view of ExternE was that $PM_{2.5}$ is more likely than sulphates to capture the fractions of ambient PM which are most detrimental to health; and so risk estimates derived in terms of $PM_{2.5}$ are more likely to transfer well than estimates in terms of sulphates. Consequently, the study bases quantification on Pope et al.'s E-R function in terms of $PM_{2.5}$, and subsequently converted to PM_{10} equivalent. The functions were however scaled downwards by a factor of three, to take account both of possible over-estimation of risk estimates in the original study through using recent rather than historical estimates of exposure and the apparently higher particle effects in time series studies in the USA compared with Europe. (Very recent results on acute mortality

in the 20 largest US cities have however given results on average very similar to those from APHEA in Europe).

In order to estimate the years of life lost (YOLL) attributable to air pollution, regression estimates from Pope et al (1995) were linked with the populations-at-risk and age-specific death rates in four European countries (Germany, Italy, Netherlands, UK), using life table methods. Effects were estimated separately for women and for men, for the currently-alive population in each of the four countries, using recent (c.1995) demographic data (numbers alive, annual death rates) in 1-year age-groups, up to age 94 inclusive, the latest years being extrapolated where necessary. The aim was to evaluate the effect on mortality of a 1-yr. increment or reduction of 10 $\mu g/m^3$ PM_{10}; i.e. with concentrations reverting to their original values after one year. This reversion is an unrealistic scenario in practice; but it permits comparability with other effects evaluated in the present study.

The life table implementation, developed and carried out by Brian Miller at the IOM, involved following the 1995 population right through until everybody now alive had reached 94 years, under two sorts of scenario:

i. a baseline scenario assuming current hazard rates remain unchanged in future;
ii. a changed scenario whereby future hazards were modified to take account of a pollution change.

Differences in results between scenarios i. and ii. were considered to be the pollution effect. This was summarised as life-years lost, scaled per 100,000 current live population, per 10 $\mu g/m^3$ PM_{10} reduction. Because the pollution change was for one year only, there are no effects on as yet unborn cohorts.

Results varied according to the degree of latency assumed between the pollution change and the resultant change in age-specific death rates. The cohort studies are, strictly, uninformative about this latency. We examined various assumptions, including immediate effects, and delays variously of 10, 20 and 30 years. (The estimated impacts reduce with degree of latency). For implementation, we chose results which close to an average between zero and 20-year latency.

Results from all four countries were similar. Consequently, a single set of values was used and applied in all countries studied. Without any scaling-down of the estimated PM2.5 effect, this amounted to an estimated 470 YOLLs per 100,000 current population (all ages) per 1-yr. increase of 10 $\mu g/m^3$ PM_{10}. After scaling, these results were reduced by a factor of three.

Respiratory hospital admissions

The relationship for respiratory hospital admissions for PM_{10} in COMEAP is taken from WHO, though based on six cities, four in the US, one in Canada and Paris. For SO_2, COMEAP uses a meta-analysis from the results of the APHEA study (Katsouyanni et al, 1997). For ozone, the results of APHEA (Touloumi et al, 1997) are used.

The ExternE E-R relationship between PM_{10} and respiratory hospital admissions was derived from data from Paris, Dab et al (1996) within the APHEA study. Data from London (Ponce de Leon et al, 1996) from APHEA provided E-R for ozone, SO_2 and NO_2, the latter only included in sensitivity analyses.

Cardiovascular hospital admissions

ExternE includes direct evidence of PM effects on cardiovascular hospital admissions as consistent with the longer-established mortality effects. The ExternE E-R function for PM_{10} and cerebrovascular hospital admissions comes from Wordley et al (1997) in Birmingham, UK. Schwartz and Morris (1995) reported results for Detroit on the relationship between cardiovascular admissions for congestive heart failure and ischaemic heart disease in those aged 65 or more. E-R functions for PM_{10} and CO in relation to admissions for congestive heart failure were derived from this study.

Emergency room visits (ERVs)
Because of differences in health care systems, ERVs apply much more in North America than to Europe, and so impacts are usually estimated for sensitivity analyses only. ExternE includes a function for COPD and PM_{10} though this was derived from a European study: Sunyer et al (1993) in Barcelona. A function for PM_{10} and ERVs for asthma was derived in ExternE from Schwartz et al (1993) and Bates (1990), while that for PM_{10} and ERVs for childhood croup was also European (Schwartz et al, 1991). Relationships for ERVs and ozone are from Cody et al (1992), with baseline values from Bates et al (1992).

The London specific studies also include GP consultations.

Restricted Activity Days (RADs)
Only ExternE includes RADs in quantification. As noted in ExternE (EC, 1995), the evidence linking RADs quantitatively with air pollution is among the weakest of all endpoints, because the underlying studies are cross-sectional rather than longitudinal in nature. However no better studies had been published in the meantime. The ExternE E-R function for PM_{10} and RADs was therefore based as before on Ostro (1987). Because particle effects estimated in the US may be higher than in Europe, the Ostro estimates were reduced to one half of their original value. Minor restricted activity days in relation to ozone were derived from Ostro and Rothschild (1989).

Acute effects in asthmatics
The health impacts on asthmatics of increased use of medication (bronchodilator usage), increase in respiratory symptoms, and cough were considered in two European studies (Dusseldorp et al 1995, Roemer et al 1993) and one US study, Pope and Dockery (1992). ExternE includes these relationships but the estimate derived from the US has been adjusted downwards to a half of its original value. The ExternE exposure-response function for O_3 and asthma attacks is from Whittemore and Korn (1980).

Respiratory symptoms in the general population
ExternE uses results from a large-scale study in California, USA (Krupnick et al., 1990).

Chronic morbidity in adults
ExternE, following Ostro, uses an E-R function from Abbey et al (1995) for PM on chronic bronchitis in adults. This gives new cases per year of chronic bronchitis per $\mu g/m^3$ for PM_{10}, though it is noted that the study is in a community of Seventh Day Adventists, with a distinctive lifestyle, and so results may not transfer well. Because of evidence suggesting that the estimated risks for particles (per $\mu g/m^3$ PM_{10}) are higher in the USA than in Europe, ExternE scales down by a factor of two the risks as estimated by Abbey et al., for use in the present study. ExternE highlights there are important questions of transferability with this particular function.

Chronic morbidity in children
ExternE uses exposure-response functions for chronic morbidity in children have been derived from Dockery et al (1989). Note however that these are best understood as additional episodes of illness, and not the development of a chronic condition.

3.2.9 Other Pollutants, Especially Carcinogens

As well as the 'classical' pollutants considered above, ExternE also considered other substances released into the atmosphere from transport sources.

These include aldehydes (including formaldehyde and acrolein); ethene and ethylene oxide; MTBE (methyl tertiary-butyl ether); VOCs (volatile organic compounds) in general, and benzene and 1,3 butadiene in particular; polycyclic aromatic hydrocarbons (PAHs); diesel exhaust particulates; and lead. The ExternE analysis is summarised below.

Note the effects of, and evidence for, these other pollutants are markedly different from those concerned with particles, SO_2, NO_x, CO and ozone. Firstly, we are dealing principally with chronic rather than with acute health effects (though rarely, there may also be identifiable acute effects at ambient outdoor concentrations). Secondly, the basis of epidemiological evidence is generally much weaker and usually involves extrapolation.

Results, guided by existing recent reviews, are provided in ExternE (EC, 1995: 1999; Pilkington and Hurley, 1996, 1997) and are summarised below.

Unit risk factors for the occurrence of cancer
A unit risk factor (URF) is the estimated probability that a person of 'standard' weight of 70 kg will develop cancer due to exposure (by inhalation) to a concentration of 1µg/m^3 of a pollutant over a 70-year lifetime. These were used as the basis for ExternE for evaluation of the effect of a 1-year increment in exposure. This assumes that a 1-year increment increases the lifetime risk of cancer by 1/70 of the URF, i.e. by 0.014 of the URF.

The scientific evidence for URFs usually consists of animal studies and some epidemiological studies of workers exposed to high concentrations. There are major methodological issues when using either occupational and/or animal studies for quantitative human risk assessment; see, e.g., US EPA (1996); or HEI (1995) with regard to diesels specifically. The overview by Nauss et al in HEI (1995) is particularly useful. Issues to be considered include that:

- The reliability of risk estimates in occupational studies depends crucially on the reliability of estimated long-term exposures of the study subjects, and this can vary considerably;
- Use for public health risk estimation requires also extrapolation both to low concentrations and to possibly more susceptible individuals;
- Quantitative use of risk estimates from animal studies may also involve low-dose extrapolation, and quantitative animal-to-human scaling.

It follows that unit risk estimates depend not only on the availability and reliability of human and animal evidence on the carcinogenicity of the pollutant, but also on the models and assumptions used in extrapolating from that evidence. It also depends more generally on the judgement of the group of experts making the classification. These difficulties have led to substantial diversity in the acceptance of quantified risk estimates for development of cancer. As a broad generalisation, quantified risk estimation for carcinogens is much more strongly established in the USA than in Europe. For example, the UK the Department of Health's

Expert Committee on Cancer has chosen **not** to use quantified risk assessment in its evaluations.

The relative importance of human data compared to animal data varies considerably in the estimation of unit risk for the pollutants with which we are concerned. Very briefly, the most reliable human data available are for benzene. There are reasonable human data available for diesel particulates, PAHs and 1,3 butadiene, though quantification of diesel risks based on the human studies is seriously limited by limitations in the exposure data of the principal studies.

The approach of the US Environmental Protection Agency (EPA)

Among the URFs proposed by various expert groups, those proposed by the US Environmental Protection Agency (EPA) are commonly quoted. These have also been used in ExternE to ensure consistency. In extrapolating from high to low exposures, the EPA has traditionally used the linearized multistage model, which assumes no threshold, and (as its name suggests) a linear dose-response relationship in the low-dose region. It may therefore over-estimate risks at low exposures. However, the risks for a baseline exposure other than 1µg/m^3 should simply be a scaling of the risks for 1µg/m^3; and it does not matter whether the pollution to be evaluated is background (as in the definition of URF) or an increment. This is a great simplification.

Benzene

Regarding carcinogenicity, benzene has been shown to be clearly genotoxic (able to damage DNA) in both in vivo and in vitro experiments. Benzene is classified by IARC as Category 1, a known human carcinogen. There is no convincing evidence of chronic non-cancer effects at ambient concentrations. There are many occupational studies investigating exposure to benzene and development of cancer, especially leukaemia. Risk quantification is complicated and many different assumptions can be used, i.e. for function shape, exposure pattern (see EC, 2000). The US Environmental Protection Agency (EPA) risk assessment for benzene gave a unit risk factor of 8x10-6 per µg/m^3 (US EPA, 1990) which is the value used for ExternE.

1,3 butadiene

1,3 butadiene is potentially carcinogenic to both the white and red cell systems. Animal studies have shown that it is carcinogenic both in rodents and in mice but there is no evidence available on cancer risks to the general population from ambient exposures. As a result, 1,3-butadiene is classified by IARC as Category 2a - Probable human carcinogen. Irritant effects also occur, but only at concentrations much higher than those relevant to Transport. The epidemiological evidence consists mostly of mortality studies that use qualitative estimates or exposure categories rather than estimates of actual lifetime exposures, and with limited consideration of other workplace exposures. The human studies cannot be used directly in quantified risk assessment because sufficiently reliable estimates of past exposures are not available. Thus, the US EPA (1990) unit risk factor (URF) of 3 (or 2.8) x 10-4 per µg/m^3 lifetime exposure is based on multi-stage modelling of animal (mice) experimental data.

Polycyclic aromatic hydrocarbons (PAHs)

PAHs include a wide range of substances including benzo[a]pyrene (BaP). The relationship between BaP and other PAHs differs for various types of emission, but has been shown to be relatively similar in the ambient air of several towns and cities. There is strong evidence, including from epidemiological studies (e.g. Redmond et al., 1972; Hurley et al., 1983; Armstrong et al., 1994) to suggest that certain components of PAHs, and specifically benzo[a]pyrene, are carcinogenic in humans; and that nitroaromatics as a group pose a hazard

to health. In 1986 IARC and the US National Cancer Institute concluded that PAHs were a risk factor for lung cancer in humans. Benzo[a]pyrene specifically, rather than PAHs as a group, is labelled as a probable human carcinogen. As these compounds form complex mixtures and are also absorbed onto particulates, it is difficult to quantify levels of human exposure and so is difficult to estimate risks reliably. Benzo[a]pyrene is the only PAH for which a suitable database is available, allowing quantitative risk assessment. The EPA unit risk factor of lung cancer for BaP is 1x10-4 per ng/m^3 (US EPA, 1990). Limitations in the use of benzo[a]pyrene as an indicator of PAH toxicity in air pollution are that some PAH is bound to particulates, and that some of the gaseous components are not included. WHO (1987) estimated a URF of 8.7 x 10-5 per ng/m^3; i.e. almost identical to that used by US EPA.

Diesel exhaust particulates

Diesel exhaust contains a complex mix of gases and particles. The composition varies considerably and has changed dramatically over the last 30 years. The carbon core of diesel particles serves as a nucleus for the condensation of organic compounds, for example PAHs. Rats develop an increased incidence of lung tumours when exposed chronically to high concentrations of diesel exhaust that overload particle clearance defences. There is growing evidence that the tumours may be because of the particle core rather than the organic constituents. Diesel soot has been shown to be a direct acting mutagen, i.e. able to cause changes in genetic material without being activated by the body's metabolism. The available human (occupational) studies are limited both by poor exposure data and limited consideration of confounders, for example environmental tobacco smoke, diet and socio-economic factors. However, the results of studies published over the past 15 years consistently show a weak association between exposure to diesel exhaust and lung cancer. There is some evidence of excess bladder cancer also.

Diesel emissions are currently classified by IARC as Category IIa; probably carcinogenic to humans. For diesel particulates, the EPA (1994) unit risk estimate derived using a linearized multistage model is 3.4 x 10-5 based on a lifetime exposure to 1 µg/m^3 diesel particulate matter. As discussed by Nauss et al (1995), the US EPA in 1990 based its risk calculations on the risk of benzo[a]pyrene (BaP), because scientific opinion at that time considered that organic constituents of diesel exhaust were responsible for observed carcinogenicity in rats. The EPA has since decided to use the non-organic carbon core of particles. A range of risk estimates is summarised by Nauss et al (1995), showing that estimates based on animal data vary by as much as two orders of magnitude, depending on the assumptions made about exposure and the extrapolation models selected. Respiratory irritation and airway hyper-reactivity can also be caused by several of the components of diesel exhaust. There are however no usable E-R functions, due to the variable and complex mixture of substances. There is very limited evidence of an association with asthma, and little evidence for other (non-pulmonary) health effects.

Formaldehyde

Formaldehyde is a potent irritant, and no clear threshold has been defined for these effects. Available evidence suggests that ambient levels of formaldehyde could produce irritant symptoms to the eyes and respiratory tract in a sub-group of the general population. It is unlikely to cause asthmatic symptoms in healthy subjects at exposures encountered in environmental settings, but could potentially exacerbate symptoms. Thus, an occasional mild effect (e.g. symptom day) among sensitive people cannot be ruled out where incremental formaldehyde from transport adds to existing relatively high background levels. However,

effects are likely to be small and are difficult to quantify; and we do not propose E-R functions.

Formaldehyde is classified as IARC Category 2A, probable human carcinogen (IARC Monographs, Volume 62, 1995). There is however no convincing evidence of an effect at low ambient exposures; and possible mechanisms suggest that in the absence of damage to the respiratory tract tissue, any cancer risks at low ambient concentrations are negligible (WHO, 1999). The Environmental Protection Agency (EPA) risk assessment for formaldehyde gave a unit risk factor of 1x10-5 per $\mu g/m^3$ (US EPA, 1990). This URF may substantially over-estimate the true risks from ambient pollution; we recommend that it be used for sensitivity analyses only.

Acetaldehyde and other aldehydes

Irritant effects are reported as for formaldehyde, although the threshold for onset and incremental effects is less well documented than for formaldehyde. Acetaldehyde is classified as IARC category 2B (possible human carcinogen), and acrolein as Category 3, due to inadequate evidence for carcinogenicity in humans and animals at the present time. The Environmental Protection Agency (EPA) risk assessment for acetaldehyde gives a unit risk factor of 2x10-6 per $\mu g/m^3$ (US EPA, 1990). Given the limited available evidence, these functions were not used.

Volatile organic compounds (VOCs), as a mixture

This is the name given to a wide range of volatile hydrocarbons, for example alkylbenzene, toluene, alkenes, formaldehyde and trichloroethane. VOCs are categorised by the World Health Organisation into four categories on the basis of their boiling point range, with no sharp cut off between the categories. Several of these have been considered individually above; we now briefly consider VOCs as a mixture.

At present there is no standardised way to summarise the combined effects of the many different compounds in the atmosphere. A tentative dose-response relationship has been suggested by Mølhave (1991), based on studies of indoor environments. However, nasal and eye irritation have been reported during episodes of photochemical pollution, and it is possible that certain VOCs contribute. It is unlikely that the acute health effects reported to be associated with VOCs would occur due to sole exposure to ambient levels of VOCs within the normal range. Their presence could have some effect if additive to the effects of other irritants. The consequences are likely to be small, and are not quantifiable.

Some VOCs are categorised as probable human carcinogens by IARC; see the separate evaluations, above. Due to the varying composition of VOCs and current limitations on monitoring, there is insufficient data to provide a reliable estimate of the risk of cancer from exposure to a mixed airborne level of VOCs. Consequently, risk estimates have not been calculated for VOCs as a group.

Lead

There is no consistent evidence that lead is a carcinogen. Inorganic lead is currently classified by IARC as a possible human carcinogen (Category 2B). For organic lead there is as yet inadequate evidence for categorisation as a human carcinogen (Category 3). We have not attempted to quantify a carcinogenic effect.

However, several non-cancer health effects have been proposed or examined. The main evidence concerns lead in air and childhood IQ in children. The pathway is in two parts, which need to be considered together:
a. the relationship between lead in blood and childhood IQ; and
b. the relationship between lead in air and lead in blood.

IQ as an endpoint is a very non-specific measure, and there is controversy about what it really represents. However, relationships with blood lead are better established with IQ, and are less variable across studies, than with more specific endpoints. Functions were used in ExternE, based on Schwartz (1994) of: 2.57 IQ points per 10 µg/dl blood lead in school-age children, though given the introduction of lead free petrol, this is no longer a major issue for London.

Platinum and related compounds
Platinum is used in vehicle exhaust catalytic converters to control air pollution. There is no body of evidence that platinum is a carcinogen. Variable results have been shown on skin irritancy tests in animals dependent on the type of platinum salt used. Allergic effects in humans as a result of inhalation of platinum salts were first documented in 1804. The main evidence is from occupational studies (e.g. Hunter et al., 1945; Roberts, 1951; Levene, 1971). It is unclear what if any of these effects might occur at ambient concentrations.

Years of life lost and latency
ExternE considered many of the above substances as initiators of cancer. For those who die from cancer as principal cause of death, the average YOLL are estimated as the difference between the expected age of death in the absence of cancer, conditional on having survived to age of diagnosis of cancer and the average age of death among those who die from the cancer. For each type of cancer endpoint identified in the review of transport pollutants, we give average YOLL accounting for age of peak incidence of the cancer in the general population, average latency and average survival rate.

3.3 Functions

The recommended sets of E-R functions from COMEAP, from ExternE and from London specific studies are shown below. Within endpoint, effects are considered as being additive across pollutants (particles, ozone, SO_2) unless otherwise stated. Note however, that different endpoints have different levels of uncertainty. Care must therefore be taken in comparing the overall effects when using the functions. As described above, estimated mortality impacts from cohort studies also include acute mortality effects. Avoidance of double-counting is best achieved by not including time series acute mortality estimates when cohort study estimates are included. This has a much greater impact on final estimates than any adjustment to RADs.

The final set of functions concerns London specific studies (Atkinson et al, 1997; Atkinson et al, 1999a, 1999b; Bremner et al, 1999; Hajat et al, 1999). The first phase of time series studies to be carried out in recent years was associated in part with the APHEA (Air Pollution and Health, a European Approach) study. This was the first to adopt a co-ordinated multi-city approach to time series studies. The underlying premise was that a multi-city approach could provide better overall estimates of effects, since individual city estimates might be misleading due to random variations and various sources of confounding and bias. Where there was inconsistency, a multi-city approach might enable this to be investigated. The implication for London is firstly that the results are methodologically comparable with the other European cities of APHEA, and secondly, that in some cases it might be preferable to

choose a European wide summary estimate rather than a London one. Reports on daily mortality, and hospital admissions for respiratory disease, cardiovascular disease and asthma for the years 1987-92 have been published (Anderson, 1996; Ponce de Leon, 1996; Poloniecki, 1996) as well as the APHEA reports, all of which incorporate London data.

The next phase used data for the years 1992-96 and incorporated additional measures (PM_{10} and CO), and health outcomes (accident and emergency attendances and general practitioner consultations), and the results have all been published. Some of these data are contributing to APHEA 2 but results will not be available until the autumn of 2000. It can be said however that the London results are not out of line with the summary estimates based on all European cities. The approach used was that of Poisson regression allowing for seasonal patterns using parametric models, long term trends, temperature, humidity, holidays, day of the week, influenza epidemics and auto-regression. Various single day and cumulative lags were used and the estimate with the strongest statistical significance was selected.

Table 3.1. Exposure-Response Functions recommended by COMEAP.

Impact Category	Pollutant	E-R function
Deaths brought forward (all cause)	PM_{10}	0.75 % per 10 µg/m^3 (24 hr mean)
	SO_2	0.60 % per 10 µg/m^3 (24 hr mean)
	Ozone	3.0 % per 50 µg/m^3 (8 hr mean)
Respiratory Hospital Admissions (RHA)	PM_{10}	0.80 % per 10 µg/m^3 (24 hr mean)
	NO_2	2.5 % per 50 µg/m^3 (24 hr mean)
	SO_2	0.50 % per 10 µg/m^3 (24 hr mean)
	Ozone	3.5 % per 50 µg/m^3 (8 hr mean)

Source: COMEAP, (1998). Quantification of the Effects of Air Pollution on Health in the UK.

Table 3.2 Exposure-Response Functions recommended by ExternE. The exposure response slope, f_{er}, has units of case events per year per person per µg/m^3, except for mortality which is expressed as percentage increase per µg/m^3

Receptor	Impact	Reference	Pollutant[1]	f_{er} [1]
ASTHMATICS				
Adults	Bronchodilator Usage	Dusseldorp et al, 1995	PM_{10} $PM_{2.5}$	0.163 0.272
	Cough	Dusseldorp et al, 1995	PM_{10} $PM_{2.5}$	0.168 0.280
	Lower respiratory symptoms (wheeze)	Dusseldorp et al, 1995	PM_{10} $PM_{2.5}$	0.061 0.101
Children	Bronchodilator usage	Roemer et al, 1993	PM_{10} $PM_{2.5}$	0.078 0.129
	Cough	Pope and Dockery, 1992	PM_{10} $PM_{2.5}$	0.133 0.223
	Lower respiratory symptoms (wheeze)	Roemer et al, 1993	PM_{10} $PM_{2.5}$	0.103 0.172
All	Asthma attacks (AA)	Whittemore & Korn, 1980	O_3	4.29 E-03
ELDERLY 65+				
	Congestive heart failure	Schwartz and Morris, 1995	PM_{10} $PM_{2.5}$ CO	1.85 E-05 3.09 E-05 5.55 E-07
CHILDREN				
	Chronic cough	Dockery et al, 1989	PM_{10} $PM_{2.5}$	2.07 E-03 3.46 E-03
ADULTS				
	Restricted activity days (RAD)[2] Minor RAD [3]	Ostro, 1987 Ostro and Rothschild, 1989	PM_{10} $PM_{2.5}$ O_3	0.025 0.042 9.76 E-03
	Chronic bronchitis	Abbey et al, 1995 (after scaling)	PM_{10} $PM_{2.5}$	2.45 E-05 3.90 E-05
ENTIRE POPULATION				
	Chronic Mortality(CM)	Pope et al, 1995 (after scaling	PM_{10} $PM_{2.5}$	0.129% 0.214%
	Respiratory hospital admissions (RHA)	Dab et al, 1996 Ponce de Leon, 1996	PM_{10} $PM_{2.5}$ SO_2 O_3	2.07 E-06 3.46 E-06 2.04 E-06 3.54 E-06
	Cerebrovascular hospital admissions	Wordley et al, 1997	PM_{10} $PM_{2.5}$	5.04 E-06 8.42 E-06
	Symptom days	Krupnick et al,	O_3	0.033
	Cancer risk estimates	Pilkington et al, 1997; based on US EPA	Benzene 1,3-butadiene	1.14 E-07 4.29 E-06
	Acute Mortality (AM)	Spix et al / Verhoeff et al, 1996 Anderson et al / Touloumi et al, 1996 Sunyer et al, 1996	PM_{10} $PM_{2.5}$ SO_2 O_3	0.040% 0.068% 0.072% 0.059%

[1] Sources: (EC, 1995) and (Hurley et al., 1997). Within ExternE, sulphates are treated as $PM_{2.5}$ and nitrates as PM_{10}.
[2] Assume that all days in hospital for respiratory admissions (RHA), congestive heart failure (CHF) and cerebrovascular conditions (CVA) are also restricted activity days (RAD). Also assume that the average stay for each is 10, 7 and 45 days respectively. Thus, net RAD = RAD - (RHA*10) - (CHF*7) - (CVA*45).
[3] Assume asthma attacks (AA) are also minor restricted activity days (MRAD), and that 3.5% of the adult population (80% of the total population) are asthmatic. Thus, net MRAD = MRAD - (AA*0.8*0.035).

Table 3.3 E-R relationships obtained from time series analyses for London 1992-94.

Outcome	Pollutant	Lag	% change (10-90th)[4]	(95% CI)	Beta coefficient	Standard error	Baseline (daily)
All cause mortality[6] All ages	PM_{10}[5]	1	0.8	-0.6, 2.2	0.000260	0.000230	169/day
	O_3[7]	2	-0.7	-2.3, 0.9	-0.000283	0.000321	
	SO_2[8]	1	1.0	-0.3, 2.3	0.000561	0.000367	
	NO_2[9]	1	1.1	0.0, 2.3	0.000313	0.000164	
	CO[10]	1	0.9	-0.2, 2.0	0.011085	0.006648	
Respiratory Mortality[6] All ages	PM_{10}	3	4.0	0.9, 7.3	0.001282	0.000513	27/day
	O_3	2	-3.6	-7.7, 0.8	-0.001407	0.000871	
	SO_2	2	3.0	-0.1, 6.2	0.001653	0.000868	
	NO_2	3	2.3	-0.4, 5.0	0.000634	0.000373	
	CO	3	2.0	-0.3, 4.5	0.025261	0.015014	
Cardio-Vascular Mortality[6] All ages	PM_{10}	1	1.7	-0.2, 3.7	0.000548	0.000320	73
	O_3	2	3.5	0.5, 6.7	0.001346	0.000591	
	SO_2	1	0.8	-1.0, 2.7	0.000452	0.000523	
	NO_2	1	2.3	0.7, 3.9	0.000622	0.000226	
	CO	1	1.4	-0.1, 3.0	0.017878	0.009426	
Respiratory Admissions[11] All ages	PM_{10}	1	3.0	1.06, 4.94	0.000956	0.000313	151
	O_3	1	1.21	-1.01, 3.48	0.000469	0.000440	
	SO_2	1	2.01	0.29, 3.76	0.001105	0.000482	
	NO_2	1	1.64	0.14, 3.15	0.000451	0.000210	
	CO	1	0.74	-0.57, 2.07	0.009243	0.008378	
Cardio Vascular Admissions[11] All ages	PM_{10}	0	1.94	0.56, 3.33	0.000624	0.000226	173
	O_3	2	2.34	0.19, 4.55	0.000902	0.000423	
	SO_2	0	1.57	0.22, 2.93	0.000864	0.000379	
	NO_2	0	1.35	0.22, 2.49	0.000372	0.000158	
	CO	0	1.27	0.20, 2.36	0.015831	0.006773	
GP Consultations[12] All lower respiratory All ages	PM_{10}	2	1.5	0.1, 3.0	0.000490	0.000238	4370
	O_3	0	-2.3	-4.3, -0.3	-0.000907	0.000412	
	SO_2	2	2.2	0.8, 3.6	0.001188	0.000386	
	NO_2	2	1.0	-0.3, 2.4	0.000285	0.000194	
	CO	2	1.4	0.2, 2.7	0.017794	0.007858	
GP Consultations[12] Upper respir. Disease All ages	PM_{10}	1	2.8	0.8, 4.9	0.000901	0.000305	4761
	O_3	3	2.1	-0.1, 4.3	0.000788	0.000426	
	SO_2	1	2.7	0.8, 4.6	0.001473	0.000521	
	NO_2	2	1.4	-0.3, 3.2	0.000392	0.000243	
	CO	1	0.8	-0.8, 2.4	0.009698	0.009948	
Accident & emergency Visits[13] All ages	PM_{10}	1	2.97	0.83, 5.16	0.000954	0.000350	243
	O_3	1	1.65	-0.92, 4.30	0.000638	0.000510	
	SO_2	1	2.81	0.72, 4.93	0.001538	0.000580	
	NO_2	1	1.20	-0.57, 3.0	0.000332	0.000250	
	CO	1	0.76	-0.83, 2.38	0.009495	0.010130	

Source: (Hajat. S et al, 1999; Bremner et al, 1999; Atkinson et al, 1999a; Atkinson et al, 1999b).

[4] Most significant lag
[5] 24 hr average 10-90th 31 mcg/m3
[6] Bremner et al, 1999
[7] 8 hr moving average 10-90th 26 ppb
[8] 24 hr average 10-90th 18 mcg/m3
[9] Max 1 hr average 10-90th 36 ppb
[10] 24 hr average 10-90th 0.8 ppm
[11] Atkinson et al, 1999
[12] Hajat et al, 1999
[13] Atkinson et al, 1999

3.4 Quantification Frameworks

3.4.1 How to estimate emissions, air pollution concentrations and population weighted pollution levels from transport

To use these relationships in a quantification framework for transport, a series of steps are undertaken. The level of detail is, to a large extent, determined by the application. However, most studies use the same generic approach, using a series of steps to:

- Quantify the emissions from the pollutant source;
- Assess the resulting air pollution concentrations in the surrounding area from these emissions (for example with air pollution dispersion models);
- Assess the population weighted pollution increases;
- Use exposure-response functions that link population weighted pollution increments to health endpoints.

Such an approach is known as a bottom up approach (also known as the impact pathway or dose-response approach). This method has been developed through studies such as the EC's ExternE project. Details by step are provided below.

3.4.2 Emissions

Emissions from vehicles vary with a number of factors including:

- Vehicle type and size
- Speed
- Technology (e.g. Euro standard)
- Fuel
- Other factors (including gradient, engine tuning, climatic conditions, vehicle condition, trip length, traffic conditions, driver behaviour, etc).

Measurement data on vehicle emissions are usually presented as emission factors. These can be based on the 'average-speed' model (see Samaras et al., 1998), the 'instantaneous emission approach' (UBA handbook, see INFRAS, 1999), drive-cycle averages or reconstruction (ESTEEM project - DG12-JOULE: see European Commission, 1998).

The level of detail required in a study will determine the accuracy of the emission factors used. At the transport scheme appraisal level, dis-aggregated factors are often used, usually with the average speed approach in the UK. Separate factors for different vehicle types, at different speeds are multiplied by the data on the vehicle stock (vehicle fuel, size and technology) and trip characteristics. Calculations to account for cold start effects are also included.

Emission factors can be found readily in the literature. These can be combined with specific data for London, for example DETR have data on the vehicle stock in London (e.g. London Traffic Monitoring Report, vehicle registrations), as well as average speeds on London's roads. Calculation of emissions in this way is not a small task, as the vehicle fleet varies with year (because of the introduction of newer vehicles with tighter emissions standards).

Average data is also available where less detail is required, and is available for example at the UK Emission Factors Database (http://www.rsk.co.uk/ukefd/index.htm) in the form below.

Table 3.4 Emission data for petrol cars in the UK (UK Emissions Factor Database)

Petrol cars	NO_x g/km								
Year	1997	1998	1999	2000	2001	2002	2003	2004	2005
Urban	0.97	0.86	0.76	0.65	0.56	0.48	0.42	0.36	0.3
Rural single c/way	1.12	0.98	0.86	0.74	0.63	0.53	0.45	0.39	0.32
Rural dual c/way	1.4	1.24	1.08	0.92	0.78	0.66	0.56	0.48	0.39
Motorway	2.07	1.84	1.63	1.4	1.21	1.04	0.89	0.77	0.64
Cold start (g/trip)	0.97	1.05	1.12	1.2	1.22	1.22	1.21	1.19	1.14

Source: UK emissions factor database. Data for other vehicles and other modes is also available at this site.

The only caveat with average data is that London is different to other UK cities. Average speeds in London are particularly low - average speeds (DETR, 1999) are shown below (for 1994-1996) and have declined steadily over the past 30 year.

Table 3.5 Average Speeds in London.

	Miles per hour			
Time	Central area	Inner area	Outer area	All
Morning peak	10.9	13.4	17	15.6
Daytime off peak	10.9	15.0	22.7	19.1
Evening peak	10.8	12.8	19.0	16.6

Source: DETR, 1999.

Most local authorities have collected emissions data as part of the National Air Quality Strategy review (i.e. stage I, II and III review and assessment). The 1995 Environment Act placed a duty on local authorities to review and assess air quality to determine if the national air quality objectives were likely to be met by the end of 2005. The first stage review requires collation of information, including details of any significant transport related sources. The aim of a second stage review and assessment is to provide a further screening of pollutant concentrations in local authority areas. Where potential exceedances are identified, the third stage is required which entails an accurate and detailed review and assessment of current and future likely air quality, which includes the compilation of emissions inventories.

3.4.3 Air Dispersion Modelling

There is a huge body of literature on the second step, air pollution dispersion modelling. The local scale analysis focuses on distances up to (typically) 20 km from the emission source. Gaussian plume models are commonly used for the estimation of local scale atmospheric dispersion of primary pollutants as they combine relatively accurate results and low computing time, though more advanced models (ADMS) have become more widely used over the last couple of years.

Air quality modelling is currently being undertaken by many local authorities as part of the NAQS review. The Department of Transport's *Design Manual for Roads and Bridges* provide appropriate air quality screening tools for road sources. If potential exceedance areas are identified, then more detailed modelling is required, using local dispersion models. The UK Department of Environment, Transport and the Regions (DETR) has a new advisory service for the local authorities concerning the use of computerised air quality models. The

service offers local authorities guidance on using dispersion models for assessing air quality. As well as guidance (Selection and Use of Dispersion Models LAQM.TG3(98)) there is also a DETR AQ Modelling Helpline.

However, air pollution dispersion extends beyond the immediate range of emissions, both from primary pollutant dispersion and secondary pollutant formation of ozone and secondary particles[14]. In practice emissions from London will contribute to background air pollution concentrations across the rest of the UK and Europe. Studies (such as ExternE, 2000) indicate that for typical urban areas, the levels of health impacts from transport may be as large outside of the source area as within it (though note the proportion of local impacts is higher for London because of the extremely high local population density).

Regional scale modelling of primary pollutants and formation of secondary species is possible and has been undertaken for health assessments in ExternE. However, it is not typically included in local air quality and impact assessments. The assessment of the effect of NO_x and VOC emissions on ozone concentrations also needs to be considered. Ozone is a secondary pollutant though modelling its formation at the local and regional scale is difficult, and is complicated by the detailed reactions - local emissions of NO from transport can actually reduce ozone levels locally (even though they contribute to ozone formation at the regional scale).

Studies such as ExternE have developed approaches for quantifying regional and secondary pollutants and impacts, but it is difficult to know how these can be translated to the framework proposed here as they rely on specific models.

3.4.4 Implementation – population weighted pollution levels.

The actual implementation of this approach relies on linking the pollution concentrations and dose-response functions to population data - to provide the likely increments in health events. Thus, for implementation, the geographical area to be considered is sub-divided into smaller regions using a regular grid system (for example a 1 km^2 grid across London with the option of higher resolution close to the source itself).

Each grid-cell is considered as a micro-environment with a homogeneous pollutant concentration. For ease of implementation, the associated population at risk is the population resident in that grid-cell. As in the epidemiological studies, daily movements of people between grid-cells are ignored. Thus, the approach requires data on the population distribution. The level of resolution of the population data can range from generic population density types (e.g. city centre, town, rural, etc.) to detailed census data.

The use of GIS allows the simple combination of population data and pollution data, and can quickly and accurate calculate health effects from pollution by simply multiplying population

[14] The health impacts of secondary particulates formed from transport emissions are likely to be low in the immediate local area of the emission. Sulphate aerosols generally have low formation rates, so the error from ignoring their impacts on local scale is considered to be small. Nitrate aerosols are considered a potentially greater problem due to the high NO_x emission levels from vehicles in comparison to other sources. However, an analysis of health impacts from nitrates revealed that effects arising within the local scale area are less than 10% of total effects, even in large urban areas (ExternE, 1998). On the basis of these results, secondary particulate formation is unlikely to be a dominant effect within the local scale area. However this does mean regional impacts are likely to be important and should be assessed. The impact of secondary particulates on health is highlighted as an area warranting further investigation.

weighted exposure by the functions. Of course, using above approach takes time and effort. At the wider policy level, a simplified approach can be used, taking values (e.g. impacts per tonne of pollutants) derived from bottom-up studies.

3.4.5 Exposure Response Functions - % change vs events

A quick look at the Tables of functions above shows there are two forms of exposure response functions, either:

1) % change in the number of events (relative risk) of any given type, normalised by unit pollution. This is applied to a baseline rate.
2) Absolute change in the number of events of any given type, normalised by unit pollution and size of population.

Both involve important issues with respect to transferability.

The second is easier to implement and must be used where data on the number of events are unavailable, or where concern exists as to whether the endpoint definition used is identical to that adopted in the original epidemiological work. The first is perhaps more accurate (as potentially it does not assume transferability of background rates, though as the uncertainty section states, this is not necessarily the case).

The extent to which the available data correctly quantify the impact referred to by the exposure-response functions identified is an important issue. At this point it should be noted that some inconsistencies are present, making estimates of some impacts from functions expressed as % change in number of events per unit pollution unreliable. Further discussion on the uncertainty this introduces is presented in the next section.

3.5 Uncertainty

The above discussion shows that there are alternative approaches with respect which pollutants, which endpoints and which functions can or should be used in quantification. This is related to the wider category of the uncertainty with such quantification methodologies.

Quantifying the sources of uncertainty is problematic because of a general lack of information. Uncertainties can be grouped into different categories, even though there may be some overlap:

- Data uncertainty (e.g. slope of a exposure-response function, deposition velocity of a pollutant);
- Model uncertainty (e.g. assumptions about causal links between a pollutant and a health impact, assumptions about form of a exposure-response function (e.g. with or without threshold), and choice of models for atmospheric dispersion and chemistry);
- Idiosyncrasies of the analyst (e.g. interpretation of ambiguous or incomplete information).

The first category (data uncertainties) is of a scientific nature. It is amenable to analysis by statistical methods, combining the component uncertainties over the steps of the pathway, to obtain formal confidence intervals around a mid estimate. The other areas are different and invariably bring the greatest source of uncertainty, as they centre on the choice of pollutants, endpoints and functions themselves. The functions or endpoints included invariably fall back on subjective judgement by experts.

There are a number of issues that are commonly cited as of concern. Given that particles dominate the health effects, the fact that the mechanism of effect for most endpoints is not well understood is a problem. There are also other issues involved. PM_{10} is a complex mixture (with respect to size and composition) and it is not known which (all or part) of the mixture is causal. There is evidence that particles (primary and secondary) from combustion are more strongly associated with effects, as are the smaller size fractions ($PM_{2.5}$, and possibly even PM_1). Note however, that any changes in the causality of different fractions has implications in wider implementation – if some fractions of the mixture are not causal, it means the levels of impacts from the remaining fraction is higher per unit value than currently estimated.

There are also issues with implementation regarding a number of areas, especially

- Can the functions be implemented linearly without threshold.
- Are the functions (and background data) transferable.

For many of these pollutants, there clearly is a threshold at the individual level; e.g. daily pollution will not lead to sudden death in healthy individuals. There is not however good evidence of a threshold at the population level; i.e. it appears that, for a large population even at low background concentrations, some vulnerable people are exposed some of the time to concentrations which do have an adverse effect. This understanding first grew in the context of ambient particles, where the 'no threshold' concept is now well established (though it is however difficult to demonstrate no threshold by epidemiological methods because of the small signal and sparse data points).

The issue of transferability is a more serious one. Results in Europe from APHEA show important differences between estimated effects of particles in ‘Western’ and ‘Eastern’ Europe. Some differences also exist between epidemiological studies in North America and Europe. These may be due to of differences in the nature of the pollution mix, or the concentration levels. They may also vary for other reasons.

Consideration of the transferability of exposure-response data from specific locations in the UK, the rest of Europe, or the USA to the UK is complex. The issues here of most importance to the implementation of the functions divide into three categories. The first concerns the scheme adopted for collection of air quality data;

- Type of monitoring equipment used
- Number of monitors/spatial resolution of system

The next category concerns the link between air quality and the dose of pollutant inhaled;

- Behaviour of population (modes of transport used, time spent outdoors in heavy traffic areas, or in buildings with windows open overlooking busy streets)

It is necessarily assumed that there is no variation in behaviour, though this is clearly a significant simplification when comparing personal exposure regimes between countries with very different climates, or different standards for vehicle emissions. Ideally therefore, some account needs to be taken of these issues when selecting functions.

Finally, a category that reflects the responsiveness of the population;

- Health state of population;
- Age structures of population;
- Access to medical services.

These issues are accounted for by the use of the set of functions given below based on % change in the number of events of morbidity and mortality. There remains a problem, however, with the consistency of disease reporting between countries. This is most problematic for minor effects, such as 'restricted activity days'.

Functions are described as 'acute' (short term) or 'chronic' (long term) relating to the period of exposure to air pollution required for the effects identified to arise in association with air pollution.

Finally, the functions in the report are generally intended for use with pollution increments, as would be the case for looking at incremental pollution changes from transport schemes, policies or options. There are some concerns about applying them to look at total air pollution, or as in the example below with all transport-related pollution, as these are non-marginal changes.

In conclusion, there are significant uncertainties for the types of health frameworks presented here. It is frequently argued that in cases where uncertainties are potentially large, as here, it is not appropriate to conduct quantification. We believe that this is wrong. By reviewing uncertainties it is possible to develop an understanding of the potential risks of different policies to society in terms of, on the one hand, potentially unnecessary expenditure on pollution prevention schemes, and on the other, potentially excessive health damages. Details of uncertainty ratings for different endpoints are presented in a recent report (Hurley et al, 2000) on quantifying the health effects of particles in the UK.

3.6 Impacts from Transport Related Air Pollution in London

This section uses the three sets of functions to quantify health impacts in London. Data on air pollution levels have been taken from mapping data from NETCEN (based on 1997 pollution maps, except for PM_{10} and NO_2, which are based on 1998, split by central, inner and outer London). To calculate levels of health impacts from air pollution concentrations, this information has been combined with GIS population for London. The population database used for this study was from the 1991 census. Data were disaggregated by sex and age (in 5 year classes). The original data were disaggregated from postcode sectors to the 1x1 km grid adopted for most of the analysis, using the GIS package ArcView 3.0. To separate out the contribution of transport, data from the National Atmospheric Emissions Inventory on emissions (from transport and all sources) has been used. It is necessary to define the distribution of several sub-groups of the population for application of some of the exposure-response functions. The following assumptions (CSO, 1999) have been used:

- Adults: those over 15 years of age (~19%);
- Adult asthmatics: 5% of adults;
- Children: those of 15 years or under;
- Child asthmatics: 10% of children;
- Elderly: those aged more than 65 years (~16%).

Mortality and morbidity data was taken from Table 3.3. Information is also available at a more detailed level (e.g. at health authority level)

These data were used to calculate the health impacts from air pollution concentrations in London. However, for the purposes of this study, what we are interested in is the health

impacts from transport emissions. Therefore the results were adjusted in line with emission estimates as a proportion of all emissions. Note for London, the contribution of transport to total emissions is much higher than for other cities. Thus, for CO, benzene, and 1,3-butadiene transport is responsible for >95% of all emissions and for PM_{10} and NOx, transport is responsible for around 75% of total emissions. For PM_{10}, however, an adjustment must be made as London has high levels of background particulates. Therefore to extrapolate this data to attribute damage to transport would severely overestimate its contribution. Evidence from APEG (Airborne Particles Expert Group) suggests that only around a third of PM_{10} arise from direct emissions in London. Note the values for SO2 are based on 1997 fuel – values will be very much lower now due to the introduction of city-diesel (low sulphur diesel).

Within this study we have not quantified the effects of transport emissions in London on ozone. Ozone formation is complex and formation must be assessed at both a local and regional level. The assessment of the effect of emission reductions on ozone concentrations at the *local* scale is difficult due to strong non-linearities associated with ozone formation, particularly concerning NO_x emissions. A release of NO_x within an urban area such as London may well cause an immediate *decrease* in ozone levels near the source because of the reaction of the direct emissions of NO with ozone to form NO_2. However, as the plume moves along, this NO_x may enhance ozone levels downwind, i.e. outside London. The effects of VOC emissions on ozone are more straightforward, although still rather variable in magnitude. Increased VOC emissions almost always give rise to increased ozone levels. In high-NO_x areas, ozone levels are strongly affected by VOC emissions. In low-NO_x areas, ozone levels are less sensitive to VOC emissions. Whether ozone increases or decreases as a result of NO_x and VOC emission changes depends heavily on a range of factors, including the distribution of emissions near the source, the ambient NO_x level, and the meteorological conditions. The same release may even cause an increase in ozone one day and a decrease in ozone on the next day. As a result, data on the role of transport in London in formation of ozone within the city and outside it are less well characterised than for other pollutants. For this reason, we have not quantified the effects of transport on ozone though stress this may not underestimate actual impacts - it may be that transport emissions in London lead to lower ozone levels within the capital, but to increases outside.

The results, for all pollution in London, and for pollution from transport are shown in the Tables below for the different sets of functions.

Table 3.6. Predicted impacts from Air Pollution for Transport in London based on recommendations from COMEAP.

Impact Category	Pollutant	Numbers of events/year
Deaths brought forward (all cause)	PM_{10}	198
	SO_2	181
	Ozone	n.q
Respiratory Hospital Admissions	PM_{10}	188
	NO_2	690
	SO_2	162
	Ozone	n.q.

n.q. = not quantified. For this analysis it is not possible to quantify ozone related effects from transport without more detailed studies.

It must be stressed that different health endpoints have very different severity. For example, the endpoints associated with symptom days and with bronchodilator use, cough and lower respiratory symptoms are minor - those associated with hospital admissions clearly are not. This is reflected in the all cause annual rates (see above) – for example in London there are some 55,000 respiratory hospital admissions per year, but some 1.5 million GP consultations for each of lower and upper respiratory symptoms.

Table 3.7. Predicted impacts from Air Pollution from Transport in London based on recommendations from ExternE

Receptor	Impact	Pollutant	Events/year
Adults	Bronchodilator use	PM_{10}	176,000
	Cough	PM_{10}	181,500
	Lower Respiratory Symptom (wheeze)	PM_{10}	65,900
Children	Bronchodilator use	PM_{10}	40,000
	Cough	PM_{10}	68,300
	Lower Respiratory Symptom (wheeze)	PM_{10}	52,900
All	Asthma attacks (AA)	O_3	n.q.
	Congestive heart failure	PM_{10}	400
		CO	280
	Chronic cough	PM_{10}	10,600
	Restricted Activity Day (RAD)	PM_{10}	540,000
	Minor RAD	O_3	n.q.
	Chronic bronchitis	PM_{10}	500
	Chronic Mortality (YOLL)	PM_{10}	33,700
	Respiratory Hospital Admission	PM_{10}	55
		SO_2	57
		O_3	n.q.
	Cerebro-vascular Hospital Admission	PM_{10}	135
	Symptom days	O_3	n.q.
	Cancer	Benzene	3
	(incidence)	1,3 butadiene	24
	Acute Mortality	PM_{10}	105
		SO_2	217
		O_3	n.q.

n.q. = not quantified. For this analysis it is not possible to quantify ozone related effects from transport without more detailed studies.

Table 3.8. Predicted impacts from Air Pollution from Transport in London based on exposure response relationships from London specific studies.

	Events/year	
Impact	**PM_{10}**	**SO_2**
All cause mortality	69	170
Respiratory mortality	55	81
Cardio- vascular mortality	63	59
Respiratory admissions	227	300
Cardio vascular admissions	169	269
GP consultations (all lower respiratory)	3,354	9355
GP consultations (upper respiratory)	293	551
Accident & emergency Visits	365	675

Note for the London specific functions, only PM_{10} and SO_2 have been assessed. This is because the functions in Table 3.3 for other pollutant (BS, NO_2, CO) show a strong correlation with PM_{10} as they represent primary pollutants from (mostly) the same source[15]. To add them to the values above would therefore be likely to double-count effects. The exception is for ozone effects, though within this study, we have not assessed transport's contribution to ozone formation in London.

In all cases we have not included secondary particulates or regional pollution impacts (i.e. outside of London) from transport. The results are therefore likely to be an underestimate of health effects.

The tables above are alternative estimates. It is stressed that the uncertainty is different for different pollutants and different endpoints. Therefore care must be taken in interpreting the results. For this reason, many studies group endpoints by uncertainty rating, for extra information in policy decisions.

We have combined the values above to do this in this study for our recommended approach. For acute mortality and respiratory hospital admissions, functions as recommended by the COMEAP have been used. For additional, more uncertain impacts, functions as recommended by ExternE have been used. The uncertainty rating relates to the available evidence and the reliability of quantification. Band I have the highest confidence – acute mortality and RHA are widely reported in the International literature. Band II endpoints are also widely reported, though there are less specific UK studies, hence they are given a lower rating. Band III (chronic mortality) is based on US studies – the higher uncertainty band reflects the additional problems with quantification of this effect. Finally, band IV endpoints are based on US studies and also include chronic morbidity effects.

Table 3.9. Predicted impacts from Air Pollution from Transport in London based on exposure response relationships by uncertainty band.

Uncertainty Band	Impact	Events / year from transport in London				
		PM_{10}	SO_2	CO	O_3	Benzene/ butadiene
I	Deaths brought forward[1]	198	181		n.q.	
	Respiratory hospital admissions[1]	188	162		n.q.	
II	Bronchodilator use[2]	216,000				
	Cough[2]	249,800				
	Lower respiratory symptom [2]	118,800				
	Cerebro-vascular hospital admission[2]	135				
	Congestive heart failure[2]	400		280		
III	Chronic Mortality (YOLL) [2]	33,700				
IV	Chronic cough[2]	10,600				
	Restricted Activity Day (RAD) [2]	540,000				
	Minor RAD[2]				n.q.	
	Chronic bronchitis[2]	500				
	Asthma attacks (AA)				n.q.	
	Symptom days				n.q.	
	Cancer[2]					27

[15] Though this assumes that PM_{10} is the primary causal agent in the air pollution mixture.

n.q. = not quantified.
[1] Based on COMEAP. [2] Based on ExternE. [3] Based on ExternE. [4] Based on ExternE.

3.7 Future Research Recommendations

As the above discussion indicates, this area is still the subject of continued research (and debate). There are therefore a large numbers of possible research areas that warrant further research. Any research recommendations here should be compared to other ongoing programmes, however, we highlight the issue of ranking/uncertainty analysis as a priority. In addition we recommend the use of the approach to look at specific scheme and policy measures, particularly in the context of current legislation with respect to health based local air quality standards (as part of the National Air Quality Strategy).

3.8 Conclusions

Studies of pollution episodes (such as the London smog episodes of the 1950s) have shown that very high levels of ambient air pollution are associated with strong increases in adverse health effects. Recent studies also reveal smaller increases in adverse health effects at the current levels of ambient air pollution typically present in urban areas. These health effects include a range of endpoints, such as premature mortality (deaths brought forward), respiratory and cardio-vascular hospital admissions, and possibly exacerbation of asthma, other respiratory symptoms and loss of lung function. The evidence for these effects is strongest for the pollutants PM_{10}, SO_2 and ozone and the relationships are widely accepted as causal. Recent studies also suggest that long-term exposure to these pollutants, especially particles, may also damage health and that these effects may be substantially greater than the acute effects described above.

Transport is a major source of these atmospheric pollutants in urban areas and therefore can be assumed to have adverse health effects. This chapter provides a rapid review of which pollutants and which effects there is strongest evidence for. It reports on major studies which have proposed quantification frameworks for linking health impacts to air pollution concentrations, and summarises how important these effects might be in the context of London.

Frameworks for quantifying the health impacts of transport related air pollution do exist. The frameworks require a series of steps and involve additional analysis to that typically found in transport model outputs. Such approaches first quantify emissions from transport vehicles (taking into account that emissions vary with vehicle type, fuel type, technology and speed). They then proceed to assess the effects of these emissions on local air pollution concentrations, usually with the use of dispersion models. The pollution data is then combined with data on population to estimate the population weighted air pollution increase. The final step is to quantify health impacts with the use of exposure-response functions from epidemiological studies, which link ambient air quality to health endpoints. There is however debate on the exact exposure-response functions and health endpoints that should be included in any assessment framework.

For this study we have compared the results from three sets of exposure-response functions: from the Department of Health's COMEAP sub-group, from the EC's ExternE Project and from functions from specific London epidemiological research studies. We have combined

the first two of these to provide a framework for quantification for health damages from transport in London. This uses COMEAP recommendations to quantify those effects for which there is the greatest confidence (least uncertainty) and ExternE recommendations to estimate additional health impacts where the evidence is strong but where quantification is more uncertain. Impacts have been classified into uncertainty bands, to reflect the different confidence levels attached to different health endpoints and quantification approaches.

The approach is applicable for looking at marginal changes from transport policies or schemes. We have however demonstrated it here by estimating the current levels of air pollution related effects from all transport in London, in order to compare the potential importance of air pollution relative to other categories of impact. We estimate 380 fatalities (deaths brought forward) and 350 respiratory hospital admissions per year occur in London from transport related pollution (excluding ozone). Interestingly the number of fatalities is of a similar magnitude to the numbers of deaths in London from traffic accidents, though it is stressed there are important differences in the age and health-state of people affected by the two impacts. Many of the deaths associated with pollution are probably in the elderly and the sick and the period of life lost may be small. The attribution of causality is also far more certain for accidents than for air pollution. In addition to these impacts, there are also estimated to be an additional 815 cardio-vascular/cerebro-vascular hospital admissions and half a million minor respiratory symptoms from transport related air pollution, though a higher uncertainty rating is attached to these values. Air pollution in London is also thought to lead to changes in life expectancy (chronic mortality). We estimate that transport related air pollution leads to the loss of around 34 thousand years of life per year, though stress there is a higher uncertainty in this value than for the endpoints above. Finally, there are a number of other possible impacts that have been reported in US studies. The use of these estimates leads to additional health impacts from transport emissions in London including a small number of deaths (less than 30 per year) from carcinogenic emissions, and a very large number (half a million) respiratory symptoms. These effects are given the highest uncertainty rating.

These numbers leads us to conclude that air pollution related health impacts from transport may be equivalent to, if not greater than transport accidents for London, though the age and health-state of the people affected are different.

Finally, there are a large numbers of possible research areas that warrant further research. Any research recommendations here should be compared to other ongoing programmes though we highlight the issue of uncertainty analysis as a priority, as well as further work to improve the estimates of chronic effects.

3.9 References

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4 Noise

4.1 Introduction

Noise is a major nuisance and is widely recognised as a dis-benefit affecting human amenity. It may also lead to a number of health impacts through a variety of direct and indirect effects, though there is considerable debate on the reliability of the evidence. These potential impacts include temporary and permanent hearing loss, interference with sleep and communication, tension, fatigue, headaches and other physical and psychological problems.

In general, current noise limits are based on primary research of industrial noise sources, those used as benchmarks for similar industry, or where historical or socio-economic or political factors have been relevant. Few studies have been designed to principally consider community aspects of noise exposure.

The EC Green Paper 'Future noise policy' (1996) shows that the EC is moving towards noise control action as a response to increased concerns about possible health effects. A framework approach is currently being developed, using 3 possible outcomes ; Zero or negative impact, intermediate impact, unacceptable noise impact at increasing noise levels.

Transport is a major source of ambient noise levels and therefore may have important health impacts. This chapter provides a rapid review of the evidence relating to noise levels, especially from transport, and discusses how any evidence could by used in quantification frameworks. Finally, it evaluates the potential effects relating to the impacts of transport noise in London.

4.2 Noise Units and Weightings

Noise levels are physically measured in terms of air pressure fluctuations. However, because of the wide range of common noise levels, a logarithmic scale is used, relative to the lowest audible sound. A logarithmic scale also has advantages in that human hearing responses to noise levels are logarithmic rather than linear in behaviour. The units of this scale are the decibel (dB).

The sensitivity of the human ear varies according to the frequency of noise. This is reflected by the mathematical weighting of particular frequency ranges during measurement. The 'A' (audio) weighting (dB(A)) is most commonly used in studies detailing the human impacts of noise, as values are weighted in accordance with a simplified representation of the sensitivity of the human ear to different frequencies. The relative importance of low-frequency sound is lessened by this weighting - this is important when considering the effects of heavy road vehicles, which tend to produce more low frequency noise and vibration.

As noise levels vary greatly over different times, some means of describing variation, or highlighting particular events is required. The common measures in use are summarised below.

The different weightings and metrics used in noise assessment complicate the assessment of the health impacts and any subsequent derivation of quantification frameworks. It is important to

consider what sound weighting scales and what time periods have been used when assessing underlying studies (and how these relate specifically to transport noise).

Measure dB(A)	Definition
L_{10}	The noise level exceeded for 10% of the sampling period.
L_{90}	The noise level exceeded for 90% of the sampling period.
L_{eq}	The continuous noise level equivalent to the energy of the measured variable noise[16].
L_{max}	The absolute maximum level experienced in the sampling period.

The standard used in UK traffic noise assessments is $L_{10,\ 18hour}$, measured between 6am and midnight (DoT, 1993). A reasonable correlation has been shown to exist between this measure and local residents' dissatisfaction with existing traffic noise. Other countries (and the OECD) use L_{eq} measures, over varying time periods. It has been shown that an approximate linear relationship exists between the L_{10} and L_{eq} scales (A-weighted), with the former generally exceeding the latter by 3 dB (Noise Advisory Council, 1978).

4.3 The Evidence for the Health Impacts of Transport Noise

4.3.1 Introduction

It is widely accepted that transport noise is a major source of nuisance. Indeed, surveys show that 23% of the UK population are bothered by noise from road traffic (NSCA, 2000).

However, there is also considerable conjecture that it also has a casual role with respect to human health impacts. This review has set out to consider these potential impacts, covering both the auditory and non-auditory effects of transport noise. Non- auditory effects of noise are defined as 'all those effects on health and well-being that are caused by exposure to noise, with the exclusion of effects on the hearing organ, and the effects which are due to masking of auditory information i.e. communication problems (Smith and Broadbent, 1992).

There are numerous references in the literature showing associations between noise and health impacts. The following sections discuss this literature, though given the time-scales of the project the review has had to be rapid, and has not been able to consider the underlying studies.

Instead we have focused on a number of major reviews on this subject that have been published over the last five years. Four main studies have been used for the review, summarised below, and their conclusions by impact endpoint compared in the following sections.

DETR (1997) (IEH)

This document is a review of papers considered within a workshop convened by DoH and DETR to assess the strength of evidence, and identify gaps in knowledge concerning the non-auditory effects of noise. The evidence for health effects was considered to be strongest in relation to annoyance, sleep disturbance, IHD, and performance in school children, although

[16] Can be expressed as '**LEP,d**' which refers to the level of daily personal noise exposure.

there was consensus only for annoyance effects. The evidence for the other factors was considered weak and only a small minority of people were considered to be affected.

DETR (1999)

The document reviews existing information on health effects and seeks to establish levels at which there might be particular effects on the population and to provide advice on the feasibility of effects-based standards. The authors found that the available literature is contradictory, with only the most poorly designed and executed studies showing significant effects. It was considered that other than noise annoyance and some indicators of sleep disturbance the evidence for other health effects was quite weak. Significant effects among the most susceptible individuals were considered to be scientifically plausible if unproven.

WHO (1999)

This report was based on the premise that excess exposure to community or environmental noise causes damage to health. This report was a follow up to an earlier report produced in 1980 report.

Institute of Occupational Medicine (Butler et al, 1999)

This report reviews literature on the non-auditory effects of noise published since 1988. It was prepared on behalf of the UK Health and Safety Executive to update a previous review prepared by Smith and Broadbent in 1992, also on behalf of the HSE. In addition, two specific questions were addressed: what are the effects of habitual noise exposure on general health and well being? And what are the practical implications of existing knowledge of the effects of noise on performance?

The findings suggested that there is some evidence for a range of effects on performance and health due to continued exposures to noise at, or in some cases below, the levels specified by current UK legislation. However, due to limited and sometimes conflicting data, this evidence is not conclusive and it is not possible to estimate accurately the impact of these effects or to accurately estimate thresholds below which there would be no adverse effects.

4.3.2 Hearing Damage

In reviewing the available evidence the DETR (1999) report considered that the evidence for hearing damage was conclusive concerning noise exposure above currently agreed action levels. However, the risks at typical exposure levels associated with environmental noise were considered very low. The impact of environmental noise exposure levels and hearing damage was not considered in detail.

The WHO (1999) report refers to the ISO standard 1999 (1SO1990) which gives a method for calculating noise-induced hearing impairment in populations exposed to all types of noise. The relationships between LA_{eq} 8H and noise induced hearing impairment are given for frequencies of 500-6000Hz and for exposure times of up to 40 years. The available data imply that a lifetime exposure to environmental and leisure noise with an LA_{eq},24h of less than 70dBA would not cause hearing impairment in over 95% of people. However, the data do not adequately consider vulnerable groups such as children, or the effects of co-exposures. It is uncertain whether the relationships between hearing impairment and noise exposure in the

ISO 1999 Standard are applicable for environmental noise of short rise time, for example low-altitude flying areas. Unfortunately, dose-response data are currently lacking for the general population. It is considered that to avoid hearing impairment impulse noise should never exceed 140dB peak sound pressure in adults, and 120dB peak sound pressure in children. Even small values of hearing impairment (10 dB averaged over 2000 to 4000Hz and over both ears) may have an effect on the understanding of speech, and impair language development.

The IEH and IOM reports only considered non-auditory effects of noise and therefore this topic was not covered by these two reports.

4.3.3 Speech interference

The DETR (1999) report concluded that the extent to which any particular degree of speech interference can be overcome, or can contribute to stress in different situations is not well understood, and the underlying relationships vary from study to study. In addition the index used is often only a proxy for direct measurement, and being subjective, may lead to erroneous results. A guideline threshold noise exposure for speech communication of 45-55LA_{eq} was suggested for the elderly or impaired.

The WHO (1999) report highlighted that the groups particularly vulnerable to speech interference effects are those with hearing impairment, the elderly, children in the process of language acquisition and those who are not familiar with the spoken language. For complete intelligibility in listeners with normal hearing, it is considered that the signal to noise ratio (i.e. the difference between the speech level and the sound pressure level of the interfering noise) should be 15-18dBA (Lazarus 1990). Outdoor speech levels decrease by about 6dB for a doubling in the distance between talker and listener. With a speech level of 50dBA (at a 1m distance this level corresponds to casual speech), the sound pressure level of interfering noise should not exceed 35dBA.

Speech interference was not specifically considered within the IEH report or the IOM study.

4.3.4 Annoyance

The DETR (1999) report highlighted that noise annoyance is usually attributed to a specific source, although the underlying causal mechanisms which result in some subjects experiencing annoyance are not always clear. Again annoyance relies on subjective measures, and comparative studies were often flawed by comparison of dissimilar scales. There are also problems in interpreting the impact of both the direct and indirect routes from noise stimulus to the annoyance effect. However the DETR reported reviews which in the main considered that there was sufficient evidence for noise and annoyance.

The IEH (1997) also considered that it was beyond dispute that people are annoyed by a wide range of external noise. Annoyance is dependent on the context in which the noise is heard. Loudness or perceived intensity is one of the main characteristics determining the undesirability of noise. The report found mixed evidence on the importance of duration and the frequency components of sound in determining annoyance.

The report confirms that noise annoyance is the most widespread and well-documented subjective response to noise. In both traffic and aircraft studies noise levels have been found to be associated with annoyance in a dose-dependent fashion (e.g. Fields, 1984).

Reports have shown that even young children report disturbance by environmental noise. Their response is less subject to bias as they are less influenced by other factors such as environmental issues and political attitudes. Generally sex differences in annoyance have not been found.

The WHO report confirmed that there is no consensus on a model for assessing total annoyance, due to a combination of environmental noise sources. It was also considered that models to assess the total annoyance of combinations of environmental noises may not be applicable for those health effects for which the mechanisms of noise interaction are unknown. There is again insufficient knowledge to accurately assess combined effects on health when noise is combined with different types of environmental agents, such as vibration and odours.

Stronger annoyance reactions have been observed when noise is accompanied by vibrations and includes low frequency components, or when noise contains impulses.

Aircraft noise produces a stronger annoyance than road traffic for the same dose level. Interference with rest, recreation and watching television is important for aircraft noise. In contrast for traffic noise, sleep disturbance is more important.

In 1980, the WHO set a general environmental health goal for outdoor noise of 55LAeq. In 1995 this was changed to a threshold below which few people are seriously annoyed also set at 55LAeq.

The outcome of the IOM review was to qualify the main finding of Smith and Broadbent that 'noise may produce annoyance'. Although the degree of annoyance depends on the physical characteristics of the noise, it is also influenced by underlying psychological traits and by short-term variations in susceptibility. Noise level was not necessarily found to be a strong predictor of noise annoyance, with some negative effects being documented at levels as low as 51dB(A), well below current action levels.

4.3.5 Sleep disturbance

The DETR (1999) report confirms that the clinical or social significance of increments in sleep disturbance are still unclear. Studies also suggest that individuals tend to habituate with an increased number of sound exposures per night over time, and so the impact is reduced. However, on the basis of the studies reviewed it was concluded that the strength of evidence was sufficient for changes to sleep pattern such as those factors highlighted in the IOM review (below), but inconclusive in relation to residual daytime sleepiness and performance.

The findings of the IEH (1997) study concurred with the findings of the DETR study.

The WHO (1995) report suggest that for the negative effects on sleep to be avoided noise exposure should not exceed 30 LA_{eq}, which is 5dB lower than 1980. The report also emphasised that 80-90% of the reported cases of sleep disturbance in noisy environments are for reasons other than the noise originating outdoors. Most of the recent field research has been conducted for aircraft noise. Meta-analyses of field studies have tended to assume that the percentage of awakenings is linearly proportional to the number of night-time noise

events. Whereas laboratory experiments have shown that habituation occurs to night-time noise events, and awakenings decrease with increasing number of sound exposures per night.

The report also set a new limit of 45dBA to avoid disturbance caused by separate noise events. For sleep disturbance, sensitive groups include the elderly and those with pre-existing physical or mental disorders.

The IOM (1999) report highlighted that noise may influence sleep by prolonging the time needed to fall asleep, cause awakening once asleep, returning to sleep once awake, and may cause a shifting from deeper sleep to shallower sleep. The significance of these effects on long term health is stil unclear. Fatigue effects were apparent in the studies considered, however extrapolation of results based on noise exposure alone were unreliable due to limited consideration of the confounding effects of other exposures.

4.3.6 Performance

The DETR report (1999) concluded that there was sufficient evidence for detrimental effects on performance in school children as a result of noise exposure, but the evidence was not conclusive for adults.

The IEH (1997) report concluded that there was good evidence from laboratory studies that noise impairs performance. However, the level of control which an individual has over a noise source is an important variable. Noise may tend to reinforce the use of a dominant strategy for task performance, rather than a variety of methods, due to impact on cognitive processing. Even moderate amounts of noise can impact on verbal memory tasks.

The report also found evidence in children for effects on central information processing, language comprehension, problem solving, long term memory, concentration, motivation and auditory discrimination.

The WHO (1999) confirmed that exposure to occupational noise impairs cognitive task performance. In children, the available evidence suggests that environmental noise impairs a number of cognitive and motivational parameters. There are no published studies on whether environmental noise in the home also impairs cognitive performance in adults. Workplace studies also show that noise can act as a distracting stimulus. Experimental noise exposure consistently produces negative after-effects on performance. For aircraft noise it has been shown that chronic exposure during early childhood appears to impair reading acquisition and reduces motivational ability. Of recent concern are the concomitant changes in blood pressure and stress hormone levels in children exposed to such environmental noise on a regular basis.

The IOM report (1999) concluded that it can be stated with some certainty that noise <u>can</u> have an effect on performance. However it was by no means easy to predict when noise will have an effect or, indeed, what that effect will be. A number of studies have suggested that when performance effects do occur, they can be evoked by noise levels well below those identified in relation to noise-induced hearing loss (e.g. 85 and 90dB(A)). Smith and Broadbent (1992) suggested that effects on speech are due to memory effects rather than perception.

4.3.7 Cardiovascular and physiological effects

The DETR (1999) report concluded that there was weak but sufficient evidence for an association between noise exposure and the risk of ischaemic heart disease, but inconclusive evidence for an association between noise exposure and sustained high blood pressure.

The IEH (1997) report highlighted the fact that many existing studies are cross-sectional, thus limiting the opportunity of identifying causal relationships. It was considered that any effect in adults of traffic noise is small and obscured by imprecise measures of noise exposure. Studies of effects of noise on heart rate during sleep suggest that habituation does not occur. Recent community studies show no convincing evidence of a relationship between noise and risk factors for cardiovascular disease (there is little evidence for raised blood pressure and some evidence that noise may be a risk factor for ischaemic heart disease).

Seven out of nine studies of children suggest an increase in blood pressure associated with long term exposure to road traffic and aircraft noise. However, the range of blood pressure elevation in these studies were within normal limits and did not suggest hypertension. Regecova and Kellerova (1995) found similar increases in heart rate and blood pressure for children exposed to greater than 60dBA, although social class might have confounded these results.

Passchier-Vermeer (1993) suggests a model of secondary risk factors such that environmental noise influences primary risk factors such as blood pressure, although there is no conclusive evidence to support this.

The WHO report discusses the hypothesis that noise acts as an environmental stressor, acute noise activating the autonomic and hormonal systems, leading to temporary changes such as increased blood pressure and heart rate. Overall it was considered that the evidence suggested a weak association between long-term environmental noise exposure and hypertension, but no dose-response relationship could be established. The overall conclusion was that cardiovascular effects are associated with long-term exposure to LA_{eq} 24h values in the range of 65-70DB or more, for both air and road traffic noise. Only the average risk is considered in these estimates, and sensitive subgroups have not been characterised sufficiently. Even a 10% increase in risk factors (relative risk of 1.1) may imply an increase of up to 200 cases per 100,000 people at risk per year.

The IOM report (1999) considered that available studies continue to be limited by poor quantification of noise exposures and adequate consideration of confounders. Whilst an acute biochemical and physiological response to noise is acknowledged, it is not possible to extrapolate these findings to the longer term, due to the limitations of study design and of cardiovascular responses that are not sustained. The authors concluded there is still a lack of convincing evidence that chronic noise exposure at current levels causes cardiovascular disease.

4.3.8 Mental health effects

There are some acknowledged beneficial effects for noise exposure on arousal, however the authors reviewed possible detrimental effects on mental health. The DETR report (1999) considered evidence for a detrimental effect of noise exposure on mental well being was inconclusive.

The IEH (1997) report also concluded that the evidence that noise causes psychiatric disorder is equivocal. No interaction was found between noise exposure and noise sensitivity in increasing the vulnerability to psychiatric disorder. It was acknowledged that hospital admission is influenced by many psychosocial variables, which are more potent than exposure to noise.

The authors concluded that many of the industrial studies are difficult to interpret as workers were exposed to other physical hazards and there may be a selection effect of less healthy workers into these types of jobs. It was considered that similar factors may operate in relation to environmental noise, although the source may be less avoidable.

Noise sensitivity, based on attitudes to noise in general (Stansfield,1992) is an intervening variable that explains much of the variance between exposure and individual annoyance responses. Available evidence suggested that much of the association between noise sensitivity and psychiatric disorder may be accounted for by the confounding association with trait anxiety.

The WHO (1995) report stated that environmental noise is not believed to be a direct cause of mental illness but is assumed to accelerate and intensify the development of latent mental disorder. The findings on environmental noise and mental health effects are inconclusive, but show the importance of taking vulnerable groups into account.

The IOM report did not consider this health effect in isolation.

4.4 Quantification

4.4.1 How to estimate noise emissions, noise levels and population weighted noise levels from transport

This section details the approaches used for predicting noise levels. These form the basis for quantification frameworks, though as the above discussion shows, quantification through to actual health impact is currently not possible with confidence.

Predicting Noise

Noise data from road traffic sources can be collected from roadside measurements or calculated from knowledge of vehicles, speeds and traffic density. Noise levels are usually fairly constant for private vehicle road traffic (except possibly for peak hours or jam conditions). However, for intermediate (and other) modes of public transport, intermittent (but regular) occurrences of loud noise events occur. Due to the logarithmic scale used for decibels, calculation of additional noise sources is an involved matter, requiring some care. An integration method can be used to predict the change to L_{eq} levels, based on the speed of the vehicles passing and the frequency of operation. For areas adjacent to main noise source areas, empirical physical relationships can be used to predict approximate noise levels.

The DoT publication ‘Calculation of Road Traffic Noise’ (1988) provides well established relationships to predict basic noise levels and various corrections that can be applied as a result of physical conditions. The approach calculates the expected roadside noise level, depending on traffic and road conditions. Similar guidance also exists for railway noise.

The accuracy of the prediction depends on the inclusion of a number of factors involved in noise propagation. Generally prediction will be made for sites immediately outside receptors, and not for points within buildings. The factors that are included can be made on the basis of desired accuracy, the end-use of the prediction and the data resources that are available. These factors include:

- The distance between source and receptor;
- The topography between source and receptor and height of propagation of noise above ground;
- The nature of ground cover between source and receptor;
- The attenuation of noise by air, depending on atmospheric conditions;
- The screening effect of barriers and buildings.

These factors are covered in DoT (1988). Many of these factors will be site-specific and relatively data-intensive. The relationships and assumptions surrounding these correction factors are detailed elsewhere. It should be noted that the increasing complexity of prediction, especially when barriers are involved, increases the overall uncertainty. Prediction for road transport sources is usually limited to a distance of 300 metres from the noise source, beyond which the effects of other sources, wind and temperature differences make forecasting difficult, especially in urban areas. For more general noise levels over larger aggregated areas, a simple distance propagation relationship may be all that is required.

4.4.2 Mechanisms for relating noise levels to receptors

Once noise levels have been measured or calculated, their significance as potential impacts can be evaluated by a number of methods, which vary in complexity.

The first is to relate noise levels to the nearest dwelling. This provides a real estimate of impact. However, it only takes account of the 'most affected individual'. This information is useful in that it will provide a guideline to whether the national standard will be exceeded at any location. It can also be used to look at the level of impact at the nearest dwelling. For example, in assessment of UK new road developments, properties must be identified where noise levels will increase above 68 dB(A)$L_{10,\ 18hour}$ (DoT, 1993). A similar technique is used in Melbourne, Australia, where whole road sections within the city's network are identified where 63 dB and 68 dB thresholds are reached (VTES, 1994).

A more balanced approach is to look at the population-weighted noise. This approach looks at all the affected individuals and actually gives a measurable value for total impact, and allows good comparison of transport modes or other options.

The simplest way to calculate impacts is to use aggregated population data (for example at a street or electoral ward level) and use these to weight an 'averaged' noise level prediction for that area. Accuracy (and data requirements) will increase with a decrease in the size of the aggregation area. With larger areas the use of a single dB level will neglect the (possibly large) variations that would be expected across the area and lead to uncertainty in attributing any noise changes to the project or proposal under question.

Most approaches aim to predict L_{eq}[17]. Note however that the single dB level approach may be too simplistic and more complex approaches that attempt to define the noise environment based on human sensitivities are justified. The following factors may need consideration:

- Time of day effects (to allow for differences between day, evening and night);
- Intermittency;
- Noise sensitivity of receptors;
- Tonality, including degree of low frequency noise and vibration;
- Attitudes to the source of noise;
- Relation to background noise levels.

In the case of traffic noise in a 'noisy' urban setting, night-time, intermittency and vibration effects may be the most significant. Addition of quantitative penalty decibels to noise indices is a common way of accounting for such qualitative effects. Alternatively, annoyance probability indices can summarise the likely perception of a certain change in the noise environment. Generally, railway noise causes less annoyance than road traffic noise (Moehler, 1988), but average annoyance is higher where vibration is a factor up to 200m from the noise source.

Predicting impacts of noise

The above sections detailed the discussion in relation to noise and health. Some exposure-response functions do exist for noise. In general the curves produced from available data are generic and follow the same general shape irrespective of the particular noise being considered. At increasing noise levels the strength of the effect or the percentage of the population affected also increases. Although it is possible to derive guideline values, due to the scatter of data points around the curve, the shape is often determined by the statistical method applied rather than the actual data.

Most functions are presented in terms of L_{eq} levels. For example, relationships exist for predicting the number of people likely to be 'highly annoyed' by a particular noise level, as shown below.

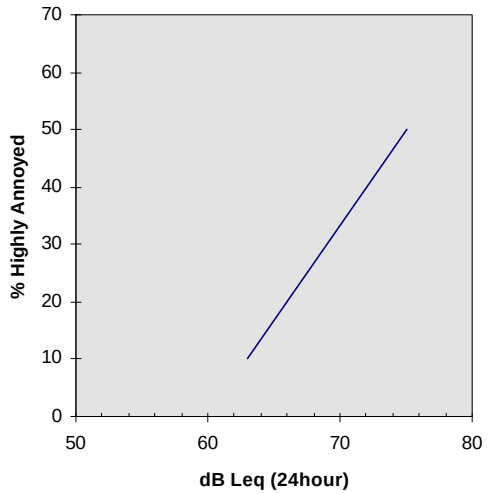

Figure 4.1. Relationship between % annoyed and L_{eq} level (London)

[17] Aircraft now also use L_{eq} (16 hour) dB(A). This is the equivalent continuous sound level that would have the same acoustic energy as the fluctuating (aircraft) noise. This is used by DETR as a daytime index for measuring people's exposure to aircraft noise and replaces the NNI (Noise and Nuisance Index).

Source Hede (1984).

This simplifies the analysis, but the drawback is the simplification of the noise environment to a single dB level does not consider other factors (such as intermittency and tonality etc.) which may be more important in actual effects.

4.4.3 Noise and perception

One of key issues with respect to noise is perception. Indeed some commentators dispute any attempt at quantification frameworks for impacts of noise on human amenity and health, arguing all effects are context specific (involving perception and the importance of voluntary vs involuntary exposure)

The Building Research Establishment (BRE) carried out a national noise attitude survey in 1991 to assess awareness of noise in the home and respondents' attitudes and emotional reactions to the various sources of noise. In the first part of the 1991 survey, respondents were asked if they heard noise from any of 49 different sources while at home, and if this noise affected them to some extent. In the survey, road traffic noise was shown to be the most commonly heard noise category and transport noise (from aircraft, trains and road traffic) was identified by the survey as affecting large numbers of respondents in some way. The higher percentages reporting disturbance to activity, and emotional reaction from neighbourhood noise and road traffic noise, are consistent with the relatively high proportions who objected in some way to noise from these sources. Different transport sources produce different responses. Some data on attitudes are available, also shown below.

Table 4.1 Number of respondents to the National Noise Attitude Survey who heard noise whilst at home and percentage who were affected: 1991

Noise source	N° who heard noise	Percentage affected (Great Britain)					
		Objected to noise	Irritated by noise	Disturbed by noise	Concerned about noise at times	Annoyed by noise at times	Consider noise a nuisance
Aircraft	969	48	67	76	41	47	49
Trains	306	43	71	78	31	50	49
Road traffic	1117	67	79	83	58	63	63
Neighbours	572	72	89	86	51	72	73
Other people nearby	302	74	86	88	53	69	68
Building construction	110	52	79	75	33	57	48
Factories or works	52	57	70	65	43	60	38
Sports events	125	26	67	59	30	41	41
Commercial premises	47	74	80	77	52	56	68
Entertainment/leisure	97	75	94	86	62	69	73
Farming/Agriculture	81	42	74	90	26	53	32

Source: BRE

Table 4.2 Number of respondents to the National Noise Attitude Survey who heard traffic noise whilst at home and percentage who were affected: 1991

Road type	N° who Heard	Percentage affected					
		Objected to noise	Irritated by noise	Disturbed by noise	Concerned about noise	Annoyed by noise	Consider noise a

	noise				at times	at times	nuisance
Motorways	27	67	67	75	75	67	75
Main roads	424	59	72	85	54	60	60
Minor roads	244	66	77	78	51	64	62
Residential/estate	217	65	75	84	52	65	62
Vehicle Type							
Heavy lorries	426	72	82	80	61	65	69
Smaller lorries/buses	350	68	81	81	53	60	61
Private cars/vans	511	64	78	81	55	63	62
Motor bikes	275	78	85	85	64	69	69
Special Noise							
Brake squeal	124	67	85	85	64	70	64
Police/other siren	203	48	62	83	46	41	44

Source: BRE

This highlights the problems in trying to derive quantification frameworks for noise impacts from transport. Many of the strongest responses are from intermittent sources (e.g. squealing of brakes). Most noise prediction frameworks only set out to assess the averaged noise levels and will therefore miss such effect. This reason, plus the subjective nature of individual responses to noise make the development of quantification frameworks especially difficult.

4.4.4 Uncertainty

It is difficult to reliably determine the evidence for health effects in relation to noise exposure, as currently no good models exist. Most adopted limits refer to levels associated with noise annoyance.

At present there are no agreed methods for combining noise exposure, health end points and likely impact into an overall response relationship. The context in which noise occurs is also very important due to differing socio-economic factors which may be influential.

There is also statistical uncertainty of multiple weak effects, multiple confounders and co-related variables, particularly when the impact of relevant noise exposures is only considered to affect a minority of the population. True effects if they exist will be swamped by this general statistical uncertainty. Traditional statistical methods are not helpful in this regard.

The DETR report concluded that ‘effect modifiers’ should not be considered as confounders but as intervening factors in the causal chain, where issues such as time relationships and context should also be considered.

The authors of the IEH report also concluded that biological mechanisms for noise exposure on health are still mainly unclear, and there is difficulty in establishing cause and effect relationships. The response is in part due to the characteristics of the sound, and the personal significance of the noise. Noise may act as a proxy for other environmental problems. Published studies often consider the indirect way in which the body responds to noise rather than effects directly caused by the noise itself.

The WHO makes it clear that protective standards are essentially derived from observations on the health effects of noise on ‘normal’ populations. It is therefore likely that vulnerable groups will be typically under-represented. Persons with impaired hearing are most adversely affected with respect to speech intelligibility. The authors considered that the evidence on

noise pollution and children's health is strong enough to warrant monitoring programmes to protect children from the effects of noise.

The IEH report considered that there are no good reasons to suppose that children or the elderly are more sensitive to noise than the general population, though they may be more vulnerable due to lifestyle factors. The authors felt that the goal should be to offer a reasonable level of protection to these groups which will also protect the normal population.

With the exception of a body of literature on the effects of heat on performance few studies have been conducted to reveal indirect effects of other physical agents comparable to the non-auditory effects of noise. Therefore it is difficult to reliably quantify the impacts of co-exposures such as noise and vibration. In conjunction with other agents, noise can produce additive, cancelling or interactive effects. However, the complexity of exposure variables make it difficult to identify consistent and stable response patterns.

It is evident that intermittent noise may be more important in some effects. Current data do not allow accurate quantification of these effects within a fluctuating background noise level. Night time noise is particularly associated with annoyance and sleep disturbance, however the cumulative effects of noise exposure over different time periods is not well understood. Also traffic and aircraft noise have been reported to be associated with different health outcomes, although there are currently limited data to support these findings for different types of noise sources. Hearing damage is particularly associated with noise in the 400Hz frequency band, although low frequencies have been reported to be associated with some non-auditory health effects of noise. The available data do not allow adequate clarification of this issue.

At the present time, it is not possible to identify a single measure to quantify noise, and a metric(s) is required against which health effects can be assessed, and to allow standardisation of measures. However, even based on current evidence there are unlikely to be any direct community or environmental noise effects on body tissue, as the energy levels involved are far too small.

4.5 Current Impacts from Transport Noise in London

It is difficult to estimate the likely impacts from transport noise in London, partly because of the debate over which health effects are real and how they can be quantified, but also because of availability of data.

Data on the noise levels in London are likely to become available in the next few year. The proposed EC Noise Directive, will require London to prepare a noise map. Transport will be the dominant noise source for the map.

There is some data currently, collected by the Building Research Establishment (BRE) as part of the National Noise Incidence Study (NNIS). This study collected data on noise levels outside homes in England and Wales. Measurements were taken 1 metre from the front of a house and 1.2m above ground level for 24 hours. The sample was stratified by region and by population size and density.

The study concluded that over half the homes in England and Wales were exposed to noise levels exceeding the World Health Organisation recommended daytime level of 55dB(A)L_{eq}, above which noise levels cause annoyance. It also found that 7 per cent of homes were above

the qualifying level at which sound insulation improvements must be provided if there is an increase in noise resulting from the development of a new road. The data is shown below and shows population exposure to noise levels of at least 30, 40, 50, 60 or 70 dB, over different periods of time (T).

Table 4.3 Distribution of noise levels[1] outside dwellings: 1990. England and Wales.

	Percentage of population exposed to levels exceeding				
Noise index	**30 dB**	**40 dB**	**50 dB**	**60 dB**	**70 dB**
LA_{eq},16h	100.0	99.9	89.3	26.0	1.8
LA_{10}, 16h	100.0	99.5	81.2	28.2	4.8
LA_{90}, 16h	98.1	64.9	12.1	1.2	
LA_{eq},18h	100.0	99.8	87.7	24.7	1.6
LA_{10},18h	99.9	99.2	77.1	26.0	4.2
LA_{90},18h	98.0	61.2	10.9	0.6	
LA_{eq},24h	100.0	99.8	80.8	20.0	0.8
LA_{10},24h	99.8	98.4	55.8	14.9	0.7
LA_{90},24h	96.9	44.5	4.4	0.1	

Source: Building Research Establishment (1993)

Notes. Noise levels are measured in decibels on the A-scale, dB(A). The A-scale takes account of the response of the human ear to different frequencies of sound. An increase of 10 dB(A) corresponds approximately to doubling the loudness of a sound. Because the noise level varies from moment to moment, three measures of the noise level (L) are used - $LA_{90,T}$, $LA_{eq,T}$ and $LA_{10,T}$. These are normally in ascending orders of magnitude, although in some circumstances $LA_{eq,T}$ can exceed $LA_{10,T}$. $LA_{eq,T}$ measures the average noise energy level over the time period, T; $L_{A90,T}$ is the noise level exceeded for 90 per cent of the measuring period T and represents a measure of the background noise present when intermittent sounds are not heard; $L_{A10,T}$ is the level exceeded for 10 per cent of the measuring period T and gives a measure of the peaks in fluctuating noise levels, such as that from road traffic.

The study also showed specific values for London. Mean noise exposure for Greater London compared to the South East and All regions are shown below, showing levels of exposure in London are higher. A new survey of noise incidence is planned and the results are expected to be available in the year 2000.

Table 4.4 Mean noise exposure for Greater London.

Region	**Number of sites**	**Mean value of LA_{10}18 h dB**	**Mean values of LA_{90} 18h (dB)**
Greater London	140	56.7	45.4
South East	200	56.0	41.2
All England and Wales	1000	55.6	42.2

Source: Building Research Establishment (1993)

Data for London are also available in relation to aircraft. UK Transport Statistics (DETR, 1999) give data on the population affected by noise around London airports though data are not reported for City airport. Data for 1997 are summarised below.

Only Heathrow is within the M25, though the other two will give rise to flights that pass over areas of London. The noise footprints for London have been decreasing over time. The population exposed to the noise footprints for Heathrow are shown in the Figure below.

Table 4.5 Noise contour data (L_{eq} 16 hour dB(A)) from London Airports*.

	Heathrow	**Gatwick**	**Standsted**
Air transport movements (000)	431	230	84
Area (km^2) within			
57 L_{eq} contour	158.3	85.9	52.1
63 L_{eq} contour	53.8	30.4	17.7
69 L_{eq} contour	23.2	10.3	6.6
Population (thousands) in			
57 L_{eq} contour	300.0	12.6	6.0
63 L_{eq} contour	84.2	2.0	0.9
69 L_{eq} contour	13.8	0.4	0.2

Source DETR, 1999. All data refers to L_{eq} 16 hour dB(A)). *No data on City Airport are available.

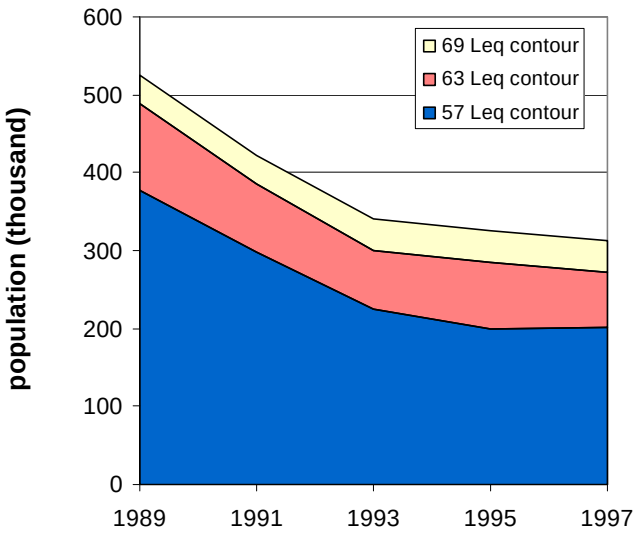

Figure 4.2 Population Exposed from Heathrow Noise Footprint (L_{eq} 16 hour dB(A)).

Despite these increases, a large population is exposed to significant noise levels from aircraft noise. There are additional noise emissions from helicopters, and some suggestions these are increasing in London, though no data are available to confirm this.

Relating this noise information to health impacts is difficult. The WHO general environmental health goal for outdoor noise is $55LA_{eq}$ (changed in 1995 this was changed to a threshold below which few people are seriously annoyed also set at $55LA_{eq}$). The data above indicates a very large number of people in London would be exposed to these levels (a minimum of several hundred thousand). Further work is needed to relate this to possible health effects.

4.6 Future Research Recommendations

There are a large number of further research recommendations with respect to noise. Those regarding the primary health impact data include longitudinal field studies and natural experiments (e.g. changes in the siting of an airport) in preference to laboratory and cross-sectional studies. Appropriate study design should also consider impacts on vulnerable groups, and confounding effects (such as age, sex, BMI, alcohol use, and family history of hypertension) and effect modifiers. Studies should also take account of relevant socio-economic and political factors across different communities exposed to noise sources.

Studies in children suggest that endocrine status, motivation and annoyance are affected by noise exposure. There is a need to establish whether these effects persist over time, or change in size, and a need to distinguish between the immediate and delayed effects of noise.

There is also a need to improve the measurement of noise sensitivity and annoyance. More work is needed on the appraisal of noise sources and the evaluation of coping mechanisms. A metric is required against which health effects can be assessed and measures standardised.

As with air pollution, any research recommendations here should be compared to other ongoing programmes. There are however, some other immediate areas that warrant research. These include a more detailed look at data sources in London (e.g. London transport data for above ground lines, Heathrow contours, T5 data, Tramlink and cross-rail, general road and rail links) to investigate noise levels across the capital. They also include the use of frameworks to look at specific policies or schemes. This would allow the evaluation of potential exposure response functions and would allow a first order calculation of the potential importance of noise (whilst accepting the confidence in estimates may be low) and so to evaluate how bigger issue noise and health might be for transport.

4.7 Conclusions

Noise is a major nuisance and is widely recognised as a dis-benefit affecting human amenity. It may also lead to a number of health impacts through a variety of direct and indirect effects, though there is considerable debate on the reliability of the evidence.

Transport is a major source of ambient noise levels and therefore may have important health impacts. This chapter provides a rapid review of the evidence relating to noise levels, especially from transport, and discusses how any evidence could by used in quantification frameworks.

The conclusive impact of the health effects of noise is mostly limited to cases of hearing loss and tinnitus caused by long periods of (occupational) exposure. These effects are generally not important at the typical levels of noise arising from transport. A number of studies also point to potential physiological and psychological impacts from the noise levels associated with transport (from road, rail and aircraft), including:

- Speech interference,
- Annoyance,
- Sleep disturbance,
- Performance,
- Cardiovascular and physiological effects,
- Mental health effects.

Speech disturbance from transport noise can occur, especially for groups vulnerable to speech interference effects (those with hearing impairment, the elderly, children in the process of language acquisition and those who are not familiar with the spoken language). However, the extent to which any particular degree of speech interference can be overcome, or can contribute to stress in different situations is not well understood and the underlying relationships vary from study to study.

There is greater evidence linking noise and annoyance, indeed, we conclude it is beyond dispute that some individuals are annoyed by a wide range of external noise. There are, however, problems in interpreting the potential health impacts of both direct and indirect routes from noise stimulus to the annoyance effect. These problems arise not least because annoyance relies on subjective measures and because of the issue of sensitivity of individuals, (susceptibility). There are also issues with respect to the context in which the noise is heard including the loudness or perceived intensity, the duration and the frequency components of sound, and because the effects also depend on underlying psychological traits of individuals. Nonetheless, studies indicate both traffic and aircraft studies noise levels have been found to be associated with annoyance in a dose-dependent fashion. We conclude that annoyance from transport is the most widespread and well-documented response to noise, but that at present it is difficult to quantify any health endpoints it may lead to.

The clinical or social significance of increments in sleep disturbance are still unclear. Studies suggest that individuals tend to habituate with an increased number of sound exposures per night over time, and so the impact is reduced. On the basis of the studies reviewed it was concluded that the strength of evidence was sufficient for a number of changes to sleep pattern. These potentially include prolonging the time needed to fall asleep, cause of awakening once asleep, returning to sleep once awake, and shifting from deeper sleep to shallower sleep. However, the significance of these effects on long term health is unclear and there may also be some issues with respect to confounding effects.

There is sufficient evidence for detrimental effects on performance in school children as a result of noise exposure, but the evidence is not conclusive for adults. The effect relates to central information processing, language comprehension, problem solving, long term memory, concentration, motivation and auditory discrimination. There are some studies that indicate there may be longer-term health related impacts (e.g. chronic exposure to aircraft noise during early childhood appears to impair reading acquisition and reduces motivational ability. There are also studies that indicate changes in blood pressure and stress hormone levels in children exposed to such environmental noise on a regular basis). However, it is concluded here that while there is evidence of performance effects, it is not easy to predict when noise will have an effect or, indeed, what that effect will be.

The evidence for cardiovascular and physiological effects is weaker. There is some evidence for an association between noise exposure and the risk of ischaemic heart disease, but inconclusive evidence for an association between noise exposure and sustained high blood pressure (i.e. a primary risk factor). Studies showing effects are limited by poor quantification of noise exposures and by adequate consideration of confounders.

Similarly, the evidence for a detrimental effect of noise exposure on mental well being is inconclusive. It is unlikely environmental noise is a direct cause of mental illness but it may accelerate and intensify the development of latent mental disorder. Such effects are

influenced by many psychosocial variables, most of which are more potent than exposure to noise.

Overall, while there is general agreement that noise is a source of annoyance, there is still debate about potential effects on health. The evidence of effects of environmental noise on health are strongest for annoyance, sleep disturbance, ischaemic heart disease and performance by school children. It is stressed that much of the evidence in support of actual health effects other than annoyance and some indicators of sleep disturbance is quite weak. The data on other possible health consequences, such as low birth-weight and psychiatric disorders, are inconclusive.

Existing exposure-response relationships are confounded by a number of variables that serve to scatter data points around the cause-effect curves. The scientific evidence suggests a threshold below which no effects are expected to occur, although this cannot be considered definitive. As well as this uncertainty in relation to exposure-response functions, there are other major problems in developing noise and health quantification frameworks. These relate to subjectivity and individual responses to noise, as well as how well average noise levels (commonly used to predict noise amenity effects) actually relate to a metric which is relevant for health endpoints.

In summary, although it is possible to assess quantitatively the noise levels from transport, it is very difficult to evaluate quantitatively what the health consequences of these levels are. A qualitative approach is possible, though there remains considerable debate on the reliability of evidence relating to health effects.

This makes evaluation of the health impacts from noise in London difficult. Moreover, data on noise levels in London is at present not available. What can be concluded is that a very large number of people in London (a minimum of several hundred thousand) are exposed to noise levels above the WHO environmental guidelines (a threshold below which few people are seriously annoyed).

A number of research areas have been identified. Those regarding the primary health impact data include longitudinal field studies and natural experiments (e.g. changes in the siting of an airport) in preference to laboratory and cross-sectional studies. Appropriate study design should also consider impacts on vulnerable groups, confounding effects and effect modifiers. Studies should also take account of relevant socio-economic and political factors across different communities exposed to noise sources. Further research is required for studies that suggest that endocrine status, motivation and annoyance are affected by noise exposure in children. There is a need to establish whether these effects persist over time, or change in size, and a need to distinguish between the immediate and delayed effects of noise.

There is also a need to improve the measurement of noise sensitivity and annoyance. More work is needed on the appraisal of noise sources and the evaluation of coping mechanisms. A metric is required against which health effects can be assessed and measures standardised.

As with air pollution, any research recommendations here should be compared to other ongoing programmes. There are however, some other immediate areas that warrant research. These include a more detailed look at data sources in London to investigate noise levels.

They also include the use of frameworks to look at specific policies or schemes. This would allow the evaluation of potential exposure response functions and would allow a first order calculation of the potential importance of noise (whilst accepting the confidence in estimates may be low) and so to evaluate how bigger issue noise and health might be for transport.

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5 Physical Activity

5.1 Introduction

The link between physically active transport and health has been recognised for more than two millennia. In 400 BCE, Hippocrates wrote: *"Eating alone will not keep a man well; he must also take exercise. For food and exercise, while possessing opposite qualities, yet work together to produce health."*(Boyer and Vaccaro, 1990)

William Buchan in 1792 regarded the increasing use of carriages and sedans to be due *"more by fashion than necessity"* and thought it strange that *"men should be such fools as to be laughed out of the use of their limbs, or to throw away their health, in order to gratify a piece of vanity."*(Royal College of Physicians, 1991)

More recently it has been shown that physical activity has significant health benefits in reducing coronary heart disease and in reducing other health impacts such as diabetes, obesity, hypertension and even depression. Indeed, epidemiological and clinical evidence suggest causal associations between regular physical activity and reduced rates of coronary heart disease, hypertension, non-insulin dependent diabetes mellitus, osteoporosis, colon cancer, anxiety and depression (BMJ, 1996).

Studies have led to commonly quoted values in the health promotion literature (e.g. HEA, 1995). These indicate that regular physical activity reduces the risk of developing certain diseases (relative to people living a sedentary lifestyle) in the order of:

- A 50 % reduction in the risk of developing coronary heart disease,
- A 50 % reduction in the onset of adult diabetes and obesity,
- A 30% reduction in risk of hypertension.

In addition, there is evidence that shows that physical activity may have other benefits. It can potentially help to improve mood, self-esteem and cognitive function. In addition, walking is a weight bearing activity, which helps to prevent reduction in bone mineral density and thereby reduce osteoporosis and the risk of fractures.

These benefits are realised through 30 minutes of activity of moderate activity on most, preferably all, days of the week. Physical activity is recommended (rather than specifying participation in organised sports or exercising) because most people can incorporate moderate physical activity into their daily routine – and it is in this respect that transport, through the encouragement of walking and cycling, can provide an opportunity for significant health benefits.

Walking and cycling can be easily incorporated into everyday life as a regular journey to work or school and this is important. Most people find it difficult to maintain an exercise programme (Rhodes et al. 1999) with limited recruitment and high drop out rates for structured and supervised exercise (Shephard, 1997). Given many of the benefits of physical activity decrease within a fortnight and disappear within two to eight months (U.S.Surgeon General, 1996) incorporating activity through commuting is an excellent way of maintaining regular activity.

However, current levels of these types of physical activity are low in the UK. Cycling accounts for less than 2% of trips in the UK (significantly lower than most other Western European countries) and walking for only 3% of distance travelled (though one-third of all journeys in Britain). There is therefore plenty of scope for improving the health of the UK through encouragement of such activities.

5.2 The Evidence for the Health Benefits of Physical Activity

There are widespread references in the literature showing links between physical activity and significant health benefits. The following sections discuss this literature, though given the time-scales of the project the review has had to be rapid, and has not been able to consider all the underlying studies.

5.2.1 Method of review

Medline (an electronic bibliography that includes all papers published in a selection of the world's medical journals since 1966) contains 25,376 records with the MESH (Medline subject heading) keyword "Exercise". Of these, 9,500 were published in the past five years. This review is therefore limited to published reviews (in reports or in peer-reviewed journals). Further restrictions included having exercise as the main focus of the paper and the review being available within the given time-frame.

5.2.2 Definitions

Physical activity is an optional behaviour. It is a global term referring to any bodily movement produced by the skeletal muscles that results in energy expenditure. (*Exercise* is sometimes used as a synonym for physical activity but often refers to participation in sports, gym-based activities or organised exercise classes). This report will therefore use the term physical activity, as defined above. *Physical fitness* is an achieved physiological condition. It is a set of attributes which people have or can acquire that relate to their ability to perform physical activity. An individual's level of fitness can be partly determined by heredity and partly be acquired through physical activity.

5.2.3 Effects and evidence

To assess the health benefits of physical activity, it is necessary to take into account the frequency, duration and intensity of the activity (light, moderate or vigorous). There are two complementary sets of recommendations regarding the amount of physical activity required for health gain (HEA, 1995):

- At least 30 minutes of moderate intensity activity on at least 5 days a week to achieve health benefits to minimise mortality.
- At least 20 minutes of vigorous intensity activity on 3 or more days a week, which maximises aerobic fitness as well as reducing mortality.

Higher levels of physical activity are associated with lower all-cause mortality in populations.

Intensity of physical activity can be measured as the percentage of the theoretical maximal heart rate; as oxygen consumption or energy expenditure; or as METs, multiples of resting metabolic rate (Table 5.1). Transport related activities are highlighted in bold.

Table 5.1 Measurements of Intensity of Physical Activity

Activity	Energy expenditure ($mlO_2/kh/min$)	Calorie consumption for 70kg person (kcal/h)	Multiples of resting metabolic rate (METs)
Driving a car, sitting at desk	**4**	**80**	**1.1**
Standing (relaxed)	5	100	1.4
Standing (eg cooking)	9	180	2.6
Scrubbing, polishing	10	200	2.9
Assembly line work	12	240	3.4
Walking at 5km/h	**13**	**260**	**3.7**
Walking, moderate work	15	300	4.3
Heavy arm work (lifting & carrying 10-20kg)	16	320	4.6
Walking at 7km/h	**21**	**420**	**6.0**
Walking uphill (1in 7) at 6km/h	24	480	6.9
Heavy arm work (lifting & carrying 38-45kg)	30	600	8.6
Running at 9km/h	**30**	**600**	**8.6**
Swimming (crawl 50m/min)	42	840	12.0

Source: Royal College of Physicians, 1991

Walking, jogging and cycling are all forms of aerobic activity that can be undertaken as a form of transport. The specific benefits of physical activity, by endpoint, are summarised below along with the key studies from the literature. For many studies, the type of activity is not specified or the activity level is a composite score from different activities during a week. Where possible, comments are restricted to the effects of those forms of activity that can be used for transport (walking, cycling and jogging). All known health benefits are described although some have not been quantified. Those that have are listed as bullet points at the end of each section.

All-cause mortality

A sedentary lifestyle is associated with raised all-cause mortality. Lee and Paffenbarger (1996) compiled a very detailed descriptive critical review of cohort studies that included all-cause mortality as an endpoint, although they gave no information on their method of selection of studies. The results of the main, methodologically-sound studies are given here.

Occupational activity

Low physical activity at work predicted increased mortality during follow-up in Finnish men and women (RR 1.9 (90% CI 1.5-2.5, $p<0.05$) for men and RR 2.2 (90% CI 1.5-3.3, $p<0.05$) for women with low cf. high occupational activity, corrected for age, cigarette use, diastolic blood pressure, serum cholesterol and BMI); (Lee and Paffenbarger, 1996).

Leisure activity

Age-adjusted mortality rates were inversely associated with leisure-time energy expenditure in US railroad men (1989 study, RR 1.21 (95% CI 1.03-1.42) for ≤250kcal/wk vs

≥2,000kcal/wk after 17-20 years follow-up); vigorous leisure exercise (≥7.5kcal/min) in British executive-grade civil servants (4.2% vs 8.4% in those not taking vigorous exercise, $p<0.001$); Finnish men and women (RR 1.5 (90% CI 1.2-2.0, $p<0.05$) for men and RR 1.6 (90%CI 1.0-2.3, $p<0.05$) for women with low cf. high leisure activity, corrected for age, cigarette use, diastolic blood pressure, serum cholesterol and BMI); and middle-aged Alameda County residents (RR 1.48 (1.08-2.02) aged 38-49, 1.38 (1.09-1.75) aged 60-69 for inactive cf. active). Unusually, men in the MRFIT study showed most reduction in all-cause mortality for those in the second tertile of leisure activity (27% reduction (9-41%) cf. lowest tertile) (Lee and Paffenbarger, 1996).

Other activity

Becoming more physically active or more fit in middle-age is associated with lower mortality than remaining inactive (Andersen, 1995). Andersen commented that misclassification bias when categorising people's levels of physical activity leads to underestimates of the benefits of activity (Andersen, 1995). Weller and Corey (1998) believe that this accounts for most studies that have included women failing to find an effect of measured activity. The age-adjusted odds of death was 0.73 for middle-aged women in the highest quartile of total activity compared with the lowest in women without known CHD, in Canada in the 1980s. On average, non-leisure activities (household chores) accounted for 82% of the women's total activity. Dividing the cohort according to time spent climbing stairs, time spent walking or time spent sitting showed that for each of these measures, activity reduced all-cause mortality and that moderate activity seemed as effective as vigorous.

Barry and Eathorne (1994) noted a lesser but probable reduction in all-cause mortality in the elderly who start being vigorously active.

- Increasing fitness through moderate physical activity may reduce an unfit man's risk of early death by 37% and an unfit woman's by nearly 50% (Boyer and Vaccaro, 1990).
- The 9½-year age-adjusted mortality for active men was 36.6% and that for women 40.4% of those for sedentary individuals (Boyer and Vaccaro, 1990).

Total activity

A 1982 Finnish cohort included women and assessed both occupational and leisure activity. Age-adjusted analyses comparing those with low activity levels at both work and in leisure time with those with high leisure and occupational activity gave relative risks (RR) for all-cause mortality of 3.9 (95% CI 2.7-55) for men and 3.5 (1.9-6.3) for women (Lee and Paffenbarger, 1996). A 1987 Finnish cohort of middle-aged men found low physical activity was associated with increased mortality in the first 10 years but disappeared after 16 years. The investigators suggested that physical activity can prevent premature death but cannot extend the lifespan (Lee and Paffenbarger, 1996). In the Harvard Alumni Health Study, walking (seven city blocks rated as 56kcal), stair climbing (28kcal for seven flights), and sports or recreational activities were summed to give weekly energy expenditure data. Mortality, adjusted for age, cigarette use, hypertension, diabetes mellitus, BMI and early parental death, was inversely related to total weekly energy expenditure and to vigorous energy expenditure but not to non-vigorous activity. Lee and Paffenbarger (1996) reported that the investigators hypothesised that imprecise assessments of non-vigorous activities may have been the explanation for this.

Lee and Paffenbarger (1996) concluded that the inverse relationship between physical activity and all-cause mortality is likely to be causal.

- The most active or fit have mortality rates that are 17 to 78% lower than the least so, comparable to the difference in mortality of non-smokers and smokers or between people of ideal weight and 20% heavier (Lee and Paffenbarger, 1996).
- 16% of all deaths would have been averted by physical activity expending ≥2,000kcal/wk (Lee and Paffenbarger, 1996). This activity level is equivalent to daily cycling for 30-40 minutes (Hillman, 1992).
- Two years of life can be gained by high physical activity (Lee and Paffenbarger, 1996).
- Expending ≥2,000kcal/wk compared with <500kcal/wk adds 2.15 years of life up to age 80, on average (Lee and Paffenbarger, 1996).

Intensity of activity required for effect on mortality

Vigorous activity but not light-to-moderate activity was required for a reduction in mortality in the 1989 US railroad and the Harvard Alumni studies (Lee and Paffenbarger, 1996).

The MRFIT and the Seventh-Day Adventist studies found greater benefit for those taking light and moderate activities than those with high intensity activity (Lee and Paffenbarger, 1996).

In the Aerobics Center Longitudinal Study (ACLS), the greatest difference in all-cause mortality was seen between the lowest and second quintiles of fitness, even after adjusting for cigarette smoking, systolic blood pressure, serum cholesterol, BMI, and parental history of CHD. The Norwegian study found no difference between the three lowest quartiles of physical fitness (Lee and Paffenbarger, 1996).

Fitness

Higher levels of physical fitness was predictive of lower all-cause mortality in US railroad men (RR 1.23 (1.17-1.30) for least fit quartile (exercise test HR >127) vs most fit (HR≤105) after adjusting for cigarette smoking, serum cholesterol and systolic blood pressure); the Lipid Clinics study (RR 1.8 (1.2-2.6) for healthy men and 2.9 (1.7-4.9) for men with pre-existing cardiovascular disease, adjusted for age, cigarette use, systolic blood pressure, HDL and LDL cholesterol); ACLS (RR 3.44 (2.05-5.77) for the least fit quintile of men cf. most fit and 4.65 (2.22-9.75) for women, adjusted for age); and a Norwegian occupational cohort (RR 0.54 (0.32-0.89) for most cf. least fit quartile, adjusted for age, cigarette smoking, systolic blood pressure, blood cholesterol, blood triglyceride, glucose tolerance, vital capacity, resting heart rate, BMI and physical activity) (Lee and Paffenbarger, 1996).

Multivariate analysis found that physical fitness but not physical activity was the significant predictor of lower all-cause mortality in the US railroad study; (Lee and Paffenbarger, 1996).

Coronary heart disease

Coronary heart disease is the commonest cause of death in the UK. Many studies refer to a reduction in risk in developing coronary heart disease, relative to those people who live a sedentary lifestyle. In general, this benefit is quoted as halving the risk of coronary heart disease. However, despite their other conclusions, Whaley and Blair stated in 1995: *"It is not at present possible to definitively describe the dose-response association between the intensity*

of physical activity and the risk of coronary heart disease. An answer to this important question awaits further research."

Risk factors for coronary heart disease

Regular aerobic physical activity increases levels of beneficial high density lipoprotein (HDL). This was seen in women who walked briskly for about 16-17km per week without significant weight loss. Physical activity to produce weight loss (jogging about 18km/wk) also increased HDL but did not reduce the atherogenic LDL (low density lipoprotein) (Royal College of Physicians, 1991). Men who walked >2.5h/wk also showed an increase in HDL cholesterol although trials of walking have had inconsistent results. Walking may also decrease fasting and postprandial triglyceride levels. HDL_2 levels are positively associated with distances walked by postmen (Morris and Hardman, 1997).

Aerobic activity can also increase fibrinolysis (Royal College of Physicians, 1991), decreases blood clotting (Health Education Authority, 1995), and improve glucose tolerance (Royal College of Physicians, 1991).

The Royal College of Physicians' (RCP) 1991 report acknowledges that the association between physical activity and lower blood pressure could be due to people who are less healthy avoiding physical activity. Morris and Hardman (1997) report that walking at 50% VO_{2max} for 1hr, 5d/wk for 4 weeks lowered systolic blood pressure by 2-3mmHg in normotensive individuals, compared with sedentary living. However, there was no association between walking at entry to the Harvard Alumni study and incidence of hypertension after 14 years.

In people with existing hypertension (raised blood pressure), 45minutes activity at 70% maximum working capacity reduced blood pressure by 11/9mmHg if performed three times per week and by 16/11mmHg if daily (Royal College of Physicians, 1991). Walking (1hr at 50% VO_{2max}, 3 times/wk) reduced blood pressure independent of weight change in a controlled trial of hypertensive people in their 60s and 70s (Morris and Hardman, 1997). Low intensity activity may reduce systolic pressure more than high intensity does, with diastolic pressure falling equally well with either. Blood pressure falls acutely and remains lower for 8-12hr after activity. Longer-term activity produces larger falls (Hardman, 1996).

- Regular endurance exercise lowers blood pressure by about 3-6mmHg in people with normal blood pressure (Health Education Authority, 1995).
- Regular endurance exercise has also been found to lower both systolic and diastolic blood pressure by about 10mmHg in men and women with mild hypertension (Health Education Authority, 1995).

Incidence of CHD

Boyer and Vaccaro quote a 1987 review by the US CDC, using sophisticated analyses to deal with issues of quality of studies, which concluded that "a causal inverse association exists between physical activity and CHD" (Boyer and Vaccaro, 1990). The RCP mentions problems with early studies, which ignored the effect of people with coronary heart disease limiting their activity but states that more recent, better quality studies suggest that physical inactivity increases the risk of developing CHD by a factor of two (Royal College of Physicians, 1991). Regular walking at entry to the Whitehall study was inversely related to the age-adjusted incidence of CHD, except for those who strolled. Walking at ≥6.4km/h for

>3.5hrs/wk halved the risk of first coronary event in 9½ years follow-up ($p<0.001$). Although they were healthier than average, adjusting for disease and lifestyle risk factors made no difference to the benefits of physical activity. Brisk walking accounted for half of the benefit due to physical activity in this cohort (Morris and Hardman, 1997).

Morris and Hardman (1997) also reported that regular walking by men in the British Regional Heart Study, was not associated with a reduced risk of heart attack in those without previous CHD except for those who walked 41-60min to and from work (more than 1mile at a fast pace, $p<0.06$ compared with other men).

Whaley and Blair (1995) reported the conclusions of two comprehensive reviews that sedentary individuals have approximately double the risk of CHD and that better studies have reported stronger inverse relationships between physical activity or fitness and CHD. They then review reports published since those reviews. All used leisure-time physical activity. They felt most of the studies were reasonably consistent with the twofold risk previously described for the least active compared with the most active men.

Two cohorts found benefits from moderate activity but two indicated that vigorous activity was required. They noted that very few men in the MRFIT cohort were vigorously active, so the study lacked power to find a graded dose-response relationship between intensity of activity and effect on CHD. Including non-fatal as well as fatal CHD, a third cohort found benefit only in the most active men. Finally, since the cut-off points between the activity intensity groups are arbitrary, the actual physical activity undertaken may have overlapped. They concluded that recent studies are consistent with an inverse and graded relationship between physical activity and CHD (Whaley and Blair, 1995).

- Physical inactivity increases the risk of developing CHD by a factor of two (Royal College of Physicians, 1991).
- Just under one-third of all CHD incidence and one-quarter of stroke incidence could be avoided by appropriate physical activity (Health Education Authority, 1995).
- Men at high risk of CHD who take part in mostly light- to moderate-intensity activity (mean 1,500kcal/wk) reduce their risk of CHD by 25-50% (Whaley and Blair, 1995).

Men who are regularly more vigorously active (>6 METS) may reduce their risk of CHD by 60-70% (Whaley and Blair, 1995).

CHD mortality

The Whitehall study found that middle-aged male civil servants who often participated in “vigorous” sports had less than half the fatal CHD of the other men. Hillman (1992) noted that “vigorous” cycling was defined for that study as at least one hour per week commuting or at least 25 miles of other cycling in the previous week. Hardman (1996) noted that brisk/fast walking by the average man could use sufficient energy to be classed as vigorous in this study. The age-standardised death rate from CHD was also inversely related to speed of usual walking pace. Those who strolled had a high rate of co-morbidity, were otherwise sedentary, and were more often smokers. Walking at ≥6.4km/h for >3.5hrs/wk halved the risk of fatal CHD in 9½ years follow-up (Morris and Hardman, 1997).

Dividing a cohort of women according to time spent walking, climbing stairs or sitting also showed an effect of moderate activity (including non-leisure activities, predominantly household chores) on CHD mortality (Weller and Corey, 1998).

The elderly who take up vigorous exercise may reduce their CHD mortality, though by less than those who have been that active lifelong (Barry and Eathorne, 1994). CHD deaths and hospital admissions were reduced in older people who walked >4h/wk compared with <1h/wk (RR 0.74), even when adjusted for other, more vigorous activities (Morris and Hardman, 1997).

Management of existing CHD

Regular aerobic activity has a training effect, leading to a reduced heart rate. This reduces the oxygen requirement of the myocardium (heart muscle) and increases exercise tolerance. This can improve symptoms as well as beta-blocker drugs can(Royal College of Physicians, 1991).

A meta-analysis reported by the RCP concluded that physical training after a myocardial infarction (MI, heart attack) improved survival (Royal College of Physicians, 1991). It improves well-being and increases return-to-work rates (Boyer and Vaccaro, 1990).

For those with mild to moderate cardiac failure , home-based physical activity can result in a reduced the heart rate with both symptomatic and objective benefits (Royal College of Physicians, 1991) Exercise tolerance and quality of life in people with congestive heart failure can be increased and fatigue and shortness of breath reduced by a progressive walking programme, increasing from 10km/wk at 13min/km to 21km/wk at 11.5min/km (Morris and Hardman, 1997).

- Physical activity programmes after a heart attack can reduce the risk of death by about 20% (Health Education Authority, 1995) to 25% (Haskell, 1994).

Stroke

Fentem (1994) stated that although fewer studies have been done, the results lead to similar conclusions for the benefits on preventing stroke as CHD. The Health Education Authority in 1995 quoted Wannamethee and Shaper’s work on the British Regional Heart Study, suggesting that physical inactivity gives middle-aged men a three-fold increased risk of stroke. It also mentions two other reviews that point towards a strong association between stroke and physical activity, but concludes that the relationship is not yet definite (Health Education Authority, 1995). Hardman (1996) also commented on the British Regional Heart Study, pointing out the steep and significant inverse gradient of age-adjusted stroke rate with physical activity category in men with or without heart disease or stroke at baseline. Moderate activity was associated with less than half the risk of stroke in sedentary individuals. In the Honolulu Heart Program, stroke risk was inversely associated with physical activity category in men aged 55-68 but not in younger middle-aged men. Haemorrhagic stroke was three- to four-fold higher in inactive or partially active men. Thromboembolic stroke was inversely related to activity only in non-smokers (double the risk in inactive compared with active men) (Hardman, 1996).

- One-quarter of stroke incidence could be avoided by appropriate physical activity (Health Education Authority, 1995).

Peripheral vascular disease

Regular physical activity improves the symptom of intermittent claudication (pain on walking that stops when the person stops walking). This can double the distance someone can walk without pain (Royal College of Physicians, 1991). The exercise may stimulate the proliferation of capillaries and mitochondria, so enhancing oxygen delivery to and use by local muscles. Relatively low intensity walking appears to suffice. Occlusive atherosclerosis does not regress (Morris and Hardman, 1997).

Physical activity is also of benefit because 50% of patients with intermittent claudication will die from a heart attack (Ernst, 1991).

Diabetes mellitus

Diabetes affects one in 30 people in England and Wales, with annual hospital costs of more than £2bn. A recent report predicts that the number of people in the UK with diabetes may double to 3m by the year 2010 because of the rising incidence of obesity and an ageing population (Audit Commission, 2000). There are two types of diabetes mellitus: type 1, with onset in childhood, and type 2, with onset in middle- or old-age. Type 1 always requires insulin treatment, being caused by a failure of the pancreas to produce insulin. It is not affected by physical activity (except for risk of adverse effects on glucose levels, see Hypo- or hyperglycaemia, below). In type 2 diabetes, patients' levels of insulin are above normal but the tissues are insensitive to insulin. Incidence increases with age, as does the even more common impaired glucose tolerance (IGT). The age-related increase in IGT is more due to increases in adiposity and decreases in physical activity and fitness than a direct effect of age (Laws and Reaven, 1990).

Insulin sensitivity

Women are more sensitive to insulin than men. Insulin stimulates the transport of glucose into, and the production of glycogen in, muscle. It also mediates these in adipose tissue but this is less important in determining an individual's sensitivity to insulin (Henriksson, 1995). In individuals with obesity or type 2 diabetes, there is also hepatic insulin resistance, with insulin unable to suppress hepatic glucose production normally (Henriksson, 1995).

Physical activity increases sensitivity to insulin in normal people. It increases the rate of glucose transport into muscle and of glycogen synthesis, both mediated by insulin (Royal College of Physicians, 1991). Walking also enhances insulin sensitivity and glucose uptake into muscle (Morris and Hardman, 1997). Henriksson (1995) reported that insulin and muscle contractile activity probably have different pathways, being additive *in vitro* and possibly synergistic *in vivo.* Both pathways are stimulated both during and for a period after physical activity. The direct effect of contractile activity stops within a few hours but the effect of physical activity on enhanced insulin sensitivity continues for one to two days afterwards. In the young, insulin sensitivity increases after a single bout of physical activity but in older individuals, a number of training episodes may be required. Both endurance and resistance training enhance whole-body insulin sensitivity.

Incidence

There is evidence to suggest that physical activity helps to defer or prevent the development of type 2 diabetes mellitus (Health Education Authority, 1995).

The RCP reported that although one study found that type 2 diabetes was less common in physically active men, this is not universally agreed (Royal College of Physicians, 1991). However, Kriska and Bennett (1992) reported prospective cohort studies in male college alumni, registered nurses and male physicians that showed a reduced incidence of self-reported physician-diagnosed diabetes in those participating in vigorous exercise, with a dose-response relationship in one of the three. In the first study, the reduction in incidence was greatest in those at highest risk. No studies of moderate activity were quoted (Kriska and Bennett, 1992).

Another review found a definite increased risk of developing type 2 diabetes in sedentary men and women compared with those exercising at least moderately, independent of other risk factors. For example, RR of developing type 2 diabetes in the subsequent 14 years in a cohort of University of Pennsylvania alumni was 0.90 with moderate, 0.69 with vigorous and 0.65 with a mixture of moderate and vigorous sport (relative to no sports) and 0.8 for climbing at least five flights of stairs daily, relative to fewer flights (with no additional benefit from ≥15 flights/d). The graded inverse relationship between weekly energy expenditure and incidence of diabetes was found whether total activity (including walking, climbing stairs and sports), all sources except vigorous sports, or vigorous sports alone were considered. The risks were independent of activity for normal weight individuals but in people with BMI ≥24kg/m^2, the inverse relationship held, as it did when normotensive and hypertensive individuals were analysed separately (Helmrich et al. 1994).

Moderately intense physical activity (≥5.5METS for ≥40 min/wk) was associated with a lower incidence of type 2 diabetes in Finnish men (OR 0.44). Effective activity included "very brisk walking", ≥8.0km/hr or "brisk walking on soft surfaces such as sand or grass" (Morris and Hardman, 1997).

- In those aged 45 and over, just under one-quarter of cases of type 2 diabetes could be avoided (Health Education Authority, 1995).
- Incidence of type 2 diabetes is reduced by 24% for every 2,000kcal/wk increment in leisure-time activity. This can be achieved by 1/2h jogging or 1h walking every day (Helmrich et al. 1994).

Treatment

Physical activity can also improve the management of type 2 diabetes.

Because of the effects on insulin sensitivity, physical activity improves control of blood glucose level in type 2 diabetics. The improvement lasts for >12 and <72 hours after the last bout of exercise; cessation of physical activity leads to reduced insulin sensitivity within three days (Anonymous1990).

Physical activity resulting in a 15% increase in maximum oxygen uptake caused a significant fall in glycosylated haemoglobin (a measure of longterm blood glucose levels), fasting glucose, and insulin levels (Royal College of Physicians, 1991). Henriksson reported that physical training in some people with type 2 diabetes improves glucose tolerance and whole-body insulin sensitivity, independently of reductions in body mass, while other studies have found little benefit. He suggested that physical activity is most effective in those with mild insulin resistance (Henriksson, 1995). Laws and Reaven (1990) reported that insulin response to oral glucose challenge can be normalised by one year of exercise training. High, not low, intensity activity seemed necessary for this effect (Laws and Reaven, 1990). However, there

are theoretical reasons for believing that brisk walking in the middle-aged or slower walking in older people would be of sufficient intensity (50-60% of VO_{2max}) to maintain stable blood glucose levels (Morris and Hardman, 1997).

People with type 2 diabetes have twice the risk of developing CHD (Laws and Reaven, 1990). Physical activity is protective against cardiovascular disease in people with type 1 diabetes (Health Education Authority, 1995)

Obesity

Obesity is increasingly common. In 1993, 13% of men and 16% of women were obese (body mass index, BMI, >30kg/m^2), and 57% of men and 48% of women overweight (BMI>25 and ≤30kg/m^2). The increase in prevalence of obesity have occurred as activity levels have decreased (Grilo, 1994). Obesity is associated with increased risk of CHD, hypertension, type 2 diabetes, arthritis, and bronchitis. Physical activity can reduce some of these risks even without weight loss (Health Education Authority, 1995). The metabolic rate of children in lower than it used to be (Hillman, 1993).

In overweight people, aerobic activity can cause modest weight loss even with out calorie restriction (Health Education Authority, 1995) although Grilo (1994) reports this as uncommon. Combined with dieting, regular physical activity is one of the most effective means of managing mild to moderate obesity. Regular aerobic activity increases energy consumption during the period of physical activity and for several hours afterwards (Royal College of Physicians, 1991). The RCP reports the beneficial effect on weight loss in moderately obese women (130 – 160% of ideal body weight) of adding to dietary restriction regular aerobic exercise, in the form of brisk walking five times per week sufficient to raise the heart rate to 60-70% of the predicted maximum for 20 minutes initially, increasing to 50 minutes daily. Moderately obese children and adolescents treated with a combination of physical activity (mostly riding a bicycle) and dietary restriction complied better with treatment and achieved greater weight loss than the group treated with diet alone (Royal College of Physicians, 1991).

Walking uses 4kJ/kg bodyweight, almost independent of pace, so walking more slowly than needed to increase fitness still aids weight control. For example, 1.6km (1mile) on the level requires 272kJ for a 75kg man and more in heavier individuals. However weight loss will not follow an increase in activity if food intake is also increased (Morris and Hardman, 1997).

Although physical activity does not consistently predict short-term weight loss, it does consistently predict successful maintenance of weight loss (Grilo, 1994).

Cancers

Most reviews have not distinguished between cancer incidence and mortality. Shephard and Futcher (1997) performed meta-analyses in their very thorough systematic review of physical activity and cancers. Their results are given at the end of each section.

All cancers

Kiningham (1998) summarised the studies that have looked at mortality from any cancer. Those measuring leisure-time physical activity have found a wide range of results although

two of three assessing occupational activity and both studies that included all physical activity found less cancer incidence or mortality in active than less active men. Most studies measuring physical fitness have found a marked inverse relationship with cancer mortality. Studies in women have been less likely to find a relationship, apart from those that combined leisure time and occupational activity or used physical fitness.

It is not known how active an individual needs to be, whether there is additional benefit of vigorous over moderate activity, or when during a lifetime for this protective effect to occur. The theories for mechanisms of action include enhanced immuno-surveillance by natural killer cells, improved defence against oxygen free radicals (although severe exercise enhances radical production more than antioxidant scavenging), confounding (with physical activity being a marker for healthy lifestyles, despite attempts at adjustment in design and analyses), or that genetic factors that increase susceptibility to cancers co-exist with genetic predisposition to be sedentary or to be less able to engage in prolonged physical activity (Kiningham, 1998).

- The variance-weighted geometric mean (VWGM) risk of all-cause cancers in sedentary workers in well-designed studies was 1.52 (95% CI 1.08-2.14) for men and 1.21 (1.04-1.40) for women relative to those in physically demanding occupations (Shephard and Futcher, 1997).
- The VWGM risk of all-cause cancers in well-designed studies of leisure activity was 1.84 (1.49-2.27) for men and 1.42 (0.97-2.08) for women with a sedentary lifestyle relative to those physically active in leisure time (Shephard and Futcher, 1997).
- The VWGM risk of all-cause cancers in well-designed studies of leisure or occupational activity was 1.43 (1.09-1.77) for men and 1.20 (0.79-1.74) for women with a sedentary lifestyle relative to those physically active in leisure time (Shephard and Futcher, 1997).
- Relative to high intensity activity, moderate intensity activity had a VWGM risk of 1.23 (1.00-1.51) and low intensity 1.66 (1.35-2.04) for men, with a similar trend for women (RR 1.31 and 1.72) (Shephard and Futcher, 1997).
- Physical activity could reduce all-cause cancer rates by as much as 46% (Shephard and Futcher, 1997).

Colon

Over 50 reports have been published on the association between physical activity and cancer of the colon (large bowel). Most have consistently found a protective effect of activity, despite different study designs (case-control or cohort studies), types of activity measured (recreational or occupational), methods of categorising activity (job title, questionnaire, athletic status at college), different sources of error, and in diverse populations. One found the inverse relationship with physical activity held for cancers of the left colon but not the right. A case-control study found that relative to people with a low fat diet and high physical activity, the RR of left-sided colon cancer was 1.2 for high fat intake and high physical activity but 5.1 for high fat and low physical activity (Gerhardsson de Verdier, 1997). Kiningham (1998) reported more studies that examined which side of the colon was affected and agreed that physical activity appeared to reduce cancer of the descending colon more than right-sided colon cancers. Studies have not found a relationship with rectal cancer; two studies reporting no effect of occupational activity on colon cancer had combined rectal and colon cancers (Kiningham, 1998).

A well-designed Swedish cohort considered both occupational and non-occupational activity and controlled for possible confounding by dietary factors, age, and population density.

People with either a sedentary occupation or a sedentary spare-time had a 1.6 higher risk of developing cancer of the colon. People who were sedentary both at work and in their spare time had a 3.6 higher risk of developing cancer of the colon than people who were physically active (Gerhardsson de Verdier, 1997).

A relationship between cancer of the colon and inactivity has been demonstrated consistently in men but not women, possibly due to imprecise measurements of women's activity and under-powered studies (Kramer and Wells, 1996).

It has been suggested that a sedentary lifestyle increases intestinal transit time, thus increasing contact between colonic mucosa and potential carcinogens in faeces. Another mechanism could be through stimulation of growth-inhibiting prostaglandins or of antioxidants by activity (Gerhardsson de Verdier, 1997). Kiningham (1998) also suggested raised insulin levels in sedentary individuals as the mechanism, since insulin is a growth factor for colonic mucosal cells. Adenomas of the colon are also inversely related with physical activity in men: VWGM 1.59 (0.62-4.06) for occupational, 1.80 (1.44-2.25) for leisure activity and 1.50 (1.12-1.87) when either was considered (Shephard and Futcher, 1997).

- The VWGM risk of cancer of the colon in sedentary workers in well-designed studies was 1.41 (95% CI 1.26-1.57) for men and 1.35 (1.06-1.71) for women relative to those in physically demanding occupations (Shephard and Futcher, 1997).
- The VWGM risk of cancer of the colon in well-designed studies of leisure activity was 1.28 (1.11-1.48) for men and 1.55 (1.15-2.10) for women with a sedentary lifestyle relative to those physically active in leisure time (Shephard and Futcher, 1997).
- The VWGM risk of cancer of the colon in well-designed studies of leisure or occupational activity was 1.39 (1.27-1.51) for men and 1.38 (1.15-1.66) for women with a sedentary lifestyle relative to those physically active in leisure time (Shephard and Futcher, 1997).
- Relative to high intensity activity, moderate intensity activity had a VWGM risk of 1.09 (0.97-1.22) and low intensity 1.37 (1.22-1.53) for men and 1.21 (0.97-1.50) and 1.21 (0.97-1.50) for women (Shephard and Futcher, 1997).

Breast

Cancer of the breast, endometrium and ovary are often grouped together as being oestrogen-dependent and having similar, though not identical, risk factors. Kramer and Wells (1996) reviewed 13 studies examining links with physical activity. Ten showed that inactivity was associated with one or more of these cancers. Of the better quality studies, longterm athletic training reduced the risk of breast cancer (RR 1.85 in non-athletes) but activity in the past did not. The Framingham study was unusual in finding a (non-significant) increase in risk associated with increased leisure activity. There was little correlation between physical activity at entry to the study and 14-16 years later. Also, the earlier instrument was likely to give a poor measure of physical activity. Postmenopausal women with low non-recreational activity levels had a higher risk of breast cancer (RR 1.7), after adjusting for 12 covariates.

In a case-control study, premenopausal women who exercised showed a graded inverse relationship with risk of breast cancer (0.8-1.6h/wk OR 0.65, 1.7-3.7h/wk OR 0.80, ≥3.8h/wk OR 0.42). The effect was greatest in parous women who exercised for at least 3.8h/wk (OR 0.28). Seven covariates were adjusted for in this study (Kramer and Wells, 1996).

Studies looking at both occupational and non-occupational activity suggested that vigorous occupational and/or non-recreational activity was more protective than vigorous non-

occupational or recreational activity, possibly because the former is undertaken more frequently (Kramer and Wells, 1996). Kiningham (1998) suggested that there may be a threshold effect for physical activity and breast cancer, with vigorous but not moderate activity being required to decrease risk.

Four hypotheses for how physical activity protects against oestrogen-dependent cancers have been suggested. The most likely is through reduction in body fat and hence extraglandular oestrogen. Secondly, strenuous exercise may reduce the number of ovulatory cycles. This seems very unlikely for moderate intensity activity. Thirdly, moderate physical activity may enhance immune function. Fourthly, the association may be coincidental, since women who are more active are more likely to have other healthy behaviours (Kramer and Wells, 1996).

- The VWGM was 1.22 (1.00-1.50) (Shephard and Futcher, 1997).
- The VWGM in post-menopausal women was 1.25 (0.98-1.61) (Shephard and Futcher, 1997).
- Moderate intensity (VWGM 1.41, 95% CI 1.17-1.69) does not confer benefit compared with low intensity (1.26, 1.03-1.55) (Shephard and Futcher, 1997).

Endometrium

For current activity, high or moderate activity (including housework and walking) was associated with a reduced risk of endometrial cancer (RR 1.0) compared with moderately low (RR 1.3-2.3) or very low activity (RR >2.5), after adjustment for 9 covariates. Other studies found similar results (Kramer and Wells, 1996).

- The VWGM for sedentary individuals was 1.38 (0.75-2.55) (Shephard and Futcher, 1997).

Prostate

Kiningham (1998) concluded that there is a trend for studies to show an inverse relationship between physical activity and incidence of prostate cancer but that the evidence is less good than for cancer of the colon. He also pointed out that lesser knowledge of other risk factors has reduced the opportunities to adjust the findings for potential confounders.
Potential mechanisms include a reduction in testosterone by aerobic activity or altered fat metabolism because of exercise (Kiningham, 1998).

- The VWGM was 1.28 (1.01-1.61) for sedentary individuals in a pooled analysis of studies of leisure or occupational activity (Shephard and Futcher, 1997).
- Moderate intensity activity (VWGM 1.06, 95% CI 0.87-1.30) confers benefit compared with low intensity activity (1.26, 1.05-1.52) (Shephard and Futcher, 1997).

Testes

- The VWGM was 1.38 (1.11-1.72) for sedentary individuals in a pooled analysis of studies of leisure or occupational activity (Shephard and Futcher, 1997).
- Moderate intensity activity (VWGM 1.14, 95% CI 0.97-1.62) may confer benefit compared with low intensity activity (1.26, 0.97-1.42) (Shephard and Futcher, 1997).

Ovary

Studies quoted by Kramer (1996) were of too poor a quality to draw meaningful conclusions. Shephard and Futcher (1997) found a risk near unity in their meta-analysis of four studies.

Lung

Shephard and Futcher's 1997 meta-analysis found no effect of physical activity on lung cancer risk. Shephard and Shek (1998) postulated that although some authors have found an increased risk of lung cancer in sedentary individuals, this may be because pre-existing respiratory disease limits activity. However, they also reported a study in which excluding cases within four years of determining baseline activity did not alter the risk of lung cancer among sedentary individuals.

Respiratory disease

Asthma

Most asthmatics benefit from physical activity, despite the risk of provoking symptoms (page 90) (Royal College of Physicians, 1991). Some children with cystic fibrosis obtain as much benefit from physical activity as from physiotherapy (Royal College of Physicians, 1991). Although people with chronic obstructive airways disease have limited exercise tolerance, this can be increased by regular physical activity (Royal College of Physicians, 1991).

Chronic obstructive pulmonary disease

Chronic obstructive pulmonary disease (COPD) decreases exercise endurance because of diminished respiratory reserve. Physical activity can be used as a treatment to prevent further deterioration (Barry and Eathorne, 1994).

Osteoporosis

Osteoporosis is a very common problem in the UK. After early adulthood, bone mass decreases with age, particularly in post-menopausal women. The risk of fracture is directly related to the bone mineral density. Weight-bearing physical activity stimulates bone metabolism and can increase bone mass. Some studies have shown that one hour walking plus aerobic activity twice weekly increases lumbar spine bone mineral density (Royal College of Physicians, 1991). Swezey (1996) concluded that walking and jogging can have a modest effect in preventing and treating osteoporosis but resistive exercise is more important for site-specific bone mineralization and muscle strength.

Participation in strength and weight-bearing activities is positively associated with bone mineral density and may be related to a reduced long-term risk of osteoporosis. Morris and Hardman (1997) noted that bone density was higher in the legs and trunk of postmenopausal women who habitually walked >12km/wk compared with those who walked <1.6km/wk. They also reported that the most recent study had shown that 20 min/d of brisk walking at 5.8km/h had positive effects on bone density and quality at the heel and possibly on bone density at the lumbar spine.

The most important time for physical activity to start is in preadolescence and adolescence, for larger, denser bones to grow (Swezey, 1996).

Hip fracture

Falls account for the majority of accidental deaths among the elderly. Each year, 2-6% of the elderly sustain a fracture because of a fall (Poulstrup and Jeune, 2000).

Physical activity in elderly men and women protects against hip fracture (Royal College of Physicians, 1991). In addition to the effects on osteoporosis, reduced risk of fractures may also be due to improved muscle strength, balance and co-ordination reducing the risk of falls (Health Education Authority, 1995). Walking up hill at least once per day was associated with a 40% reduction in the risk of hip fracture in Asian women (Morris and Hardman, 1997).

Joakimsen and colleagues found that the association between physical activity and hip fractures was strong and consistent for leisure-time activity but weaker for occupational activity. Activity both in teenage and young adult years and current activity are important but activity aged 50 years may not protect against hip fractures later. Studies in the frail elderly show, not surprisingly, that the more active are at greater risk of falls than others, who presumably spend more time in bed or sitting in chairs and less time at risk of falling. Climbing stairs and walking also seem to protect against hip fracture (Joakimsen et al. 1997).

- In those aged 45 and over, just over half the hip fractures could be avoided (Health Education Authority, 1995).

Low back pain

Low back pain is a common cause of sickness absence (Liemohn, 1990) and costs the NHS 156m in 1988 (Mindell, 1994). Acute lower back injuries are more common in those who are the least physically fit (Royal College of Physicians, 1991). Liemohn (1990) commented that opinion was divided whether aerobic activity, including jogging, was more likely to protect against or cause low back pain.

Mental health

Ten percent of patients visiting their GP have depressive symptoms (Glenister, 1996). Those who are regularly physically active feel less depression, tension, fatigue and aggression and sleep better. Feelings of well-being may also be associated with the sense of achievement, improved physical appearance, and distraction from worries (Royal College of Physicians, 1991). With prolonged activity, runners and cyclists become more self-sufficient, serious and more relaxed (Casper, 1993).

Glenister (1996) reviewed randomised controlled trials of physical activity. Aerobic activity improved perceived health without changing measures of mental health. Improvement in aerobic conditioning correlated with reduced psychological distress, depression and perceived stress. It improved generalised feelings of well-being, freedom from worries and it enhanced energy levels. In older people, physical activity was associated with faster psychomotor speed, less anxiety, and self-reported enhanced mental alertness and energy levels.

One study of 10 weeks' aerobic activity found significant psychological benefits in the moderate intensity group but not the high intensity or attention placebo groups, while another found significant improvements in depressive or anxiety symptoms, particularly among smokers, with no difference between moderate and high intensity activity. Social aspects of exercising at a leisure centre rather than at home had no effect (Glenister, 1996).

Regular participation in physical activity is also associated with raised self-esteem in children and adults (Rejeski et al. 1996).

Peripheral levels of endorphins rise in response to physical activity but endorphins do not cross the blood-brain barrier (Royal College of Physicians, 1991). Habitual dynamic aerobic activity reduces physiological, cardiovascular and catecholamine reactivity to stress, in particular to psychosocial stress (Morris and Hardman, 1997).

Other benefits to well-being, from companionship and pleasant physical surroundings (Royal College of Physicians, 1991), are not necessarily relevant to active transport in London.

North and colleagues (1990) conducted a series of meta-analyses on physical activity and depression. Physical activity reduced depression most in patients receiving medical and/or psychological care. Published studies had larger effect sizes (-0.69) than unpublished studies (-0.37). Physical activity was more effective at reducing depression as medical rehabilitation (-0.97) than in academic experiments (-0.67), psychological rehabilitation schemes (-0.55) or when done to improve general health (-0.29). Home exercise (-1.34) reduced depression more than exercise at other locations. Largest effects were seen in people on haemodialysis (-2.31), with schizophrenia (-1.43) or after a heart attack (-0.95).

- Exercise affects depression, within an effect size in non-clinically depressed people of –0.53 ($p<0.001$) (North et al. 1990).
- Even a single exercise session reduces depression, with effect size –0.31 ($p<0.001$) (North et al. 1990).
- The effect of walking and/or jogging on depression is –0.55 ($p<0.001$) (North et al. 1990).
- There is a graded effect of length of exercise program, with effect sizes of –0.3 at 5-12 weeks, -1.0 at 17-20 weeks, -2.9 at 21-24 weeks and –2.0 beyond 24 weeks (all $p<0.001$) (North et al. 1990).

Mental illness

One in seven British adults aged 16-64 have some form of neurotic health problem. A tenth of all people consulting their GP are depressed, half of whom have major depression (Glenister, 1996). Regular aerobic activity is of benefit in treating mild depression and mild anxiety (Health Education Authority, 1995; Royal College of Physicians, 1991).

Compton and colleagues believed that poor study design prevents claims that physical activity is proven to reduce anxiety and depression in those with emotional or behavioural problems (Compton et al. 1989). Casper (1993) concluded regular activity has a powerful therapeutic effect in depressive disorders. Glenister (1996) felt that despite methodological flaws, physical activity has a positive effect on depressed patients. Physical activity does not affect severe depression (Royal College of Physicians, 1991). Physical activity was as effective an anti-depressant as psychotherapy and was more effective than relaxation and enjoyable activities. Anaerobic activity was as beneficial as aerobic (North et al. 1990).

- 7% of mental disorders could be prevented by physical activity (Health Education Authority, 1995).
- Exercise affects depression, within an effect size in clinically depressed people of –0.59 ($p<0.001$) (North et al. 1990).
- Physical activity in reactive depression has an effect size of –0.67 ($p<0.05$) (North et al. 1990).

- Physical activity in situational depression has an effect size of –0.71 ($p<0.001$) (North et al. 1990).
- Physical activity in unipolar depression has an effect size of –0.60 ($p<0.01$) (North et al. 1990).

Dementia

Dementia results in isolation, leading to inactivity, physical deterioration, weakness and loss of range of movements. Barry and Eathorne (1994) report that some but not all studies have found that physical activity can be of benefit but requires supervision.

Effects in the elderly

Many older people have grown up expecting not to be physically active as they become older. The evidence shows that those who are physically active in middle age and continue to be active have less deterioration with age than control groups. Muscle bulk and strength can be increased by 10-20% by appropriate exercise in men in their early seventies. Aerobic activity, including walking, jogging or cycling, can improve stamina (Royal College of Physicians, 1991). Self-selected walking speeds tend to remain constant in adult life until the 6th decade then decrease sharply by 12-16% after that. Most pedestrian crossings assume a walking speed of around 4km/h but those in their 8th decade are often more comfortable walking at 3.2km/h. Gradually increasing physical activity to increase fitness and muscle strength may increase possible walking speed and therefore safety and independence (Morris and Hardman, 1997). Physical activity does not extend lifespan but reduces premature morbidity and mortality (Shephard, 1995).

Physical activity has been shown to have many beneficial effects in later life, including improvements in balance, co-ordination, mobility, strength and endurance and in the control of chronic disease. Benefits in the very elderly can also occur from starting a new programme of physical activity, for example walking (Health Education Authority, 1995). Activity leading to even slight increases in physical fitness can improved self-esteem and confidence in performing daily tasks (Rejeski et al. 1996).

Long-term physical activity can improve performance of some cognitive tasks but the potential for physical activity to improve cognition and storage or recall of information is not known (Rejeski et al. 1996). However, reduced memory loss in the elderly who are physically active has been reported (Health Education Authority, 1995).

Disability

Physical activity appears to contribute to compression of morbidity as well as to reduced age-specific mortality. Among a cohort of Americans over 50, vigorous activity (running for 280 minutes weekly on average) delayed the onset of disability by 10 years, with the increase in disability occurring at a rate one-third of that in non-exercising controls. With increasing age (58 to 70), the difference in physical function increased between the two groups. Lifetime disability in exercisers is only one-third to one-half that of sedentary individuals (Fries, 1998). Physical activity can preserve function to perform the activities of daily living and can avoid institutionalisation (Shephard, 1995).

Other

Senior managers participating in exercise programmes reported reduced stress, improved productivity, enhanced problem-solving ability and increased concentration. Organisations have also found benefits of physically active employees to include improved work performance, decreased absenteeism and reduced turnover, although these effects can be small (Boyer and Vaccaro, 1990).

Immunity

A lower incidence of upper respiratory tract infection has been noted in moderately active people and a higher incidence in athletes, so a J-shaped relationship has been proposed between activity and immune function. Walking for 45min/d at 60% VO_{2max}, 5d/wk for 15 weeks led to a 57% increase in killer lymphocytes (Morris and Hardman, 1997).

Health-related quality of life

Rejeski and colleagues (1996) reviewed health-related quality of life (HRQL), representing "the functional effects of an illness and its consequent therapy upon a patient, as perceived by the patient." It represents patient-defined outcomes and is multidimensional. In general, physical activity improved HRQL, regardless of age, activity status or health. In healthy sedentary adults, walking led to reductions in mood disturbance in women and increased positive affect in men. Different intensities of walking all improved quality of well-being in people with chronic pulmonary disease in proportion to improvement in exercise tolerance in comparison with controls. Walking improved pain in people with osteoarthritis (OA) and reduced anxiety and depression.

Sleep

A meta-analysis of studies showed that acute and chronic activity each increased slow wave sleep and total sleep time and decreased sleep onset latency and REM sleep (Kubitz et al. 1996).

5.2.4 Would active transport produce these effects?

The ACTIVE for LIFE campaign (Heath Education Authority) was commissioned in 1996 by the Department of Health (HEA, 2000). They recommend that walking two 1 mile journeys per day would be enough to satisfy the 'Half an Hour a Day' physical activity recommendation for adults (the accumulation of 30 minutes of more of moderate-intensity activity on most, or preferably, all days of the week (Lumsdon and Mitchell, 1999)). Similarly, cycling at about 10mph would be moderate intensity activity for most people - the level recommended for health benefits. Cycling two 3 mile journeys per day would be enough to satisfy the 'Half an Hour a Day' physical activity recommendation for adults.

Incorporating activity into routine daily living can facilitate better compliance with the recommendations for physical activity by the general population than requiring attendance at sports facilities (Hillsdon et al. 1995). It is also cheaper, less demanding of extra time, and is more readily accessible, important considerations since lack of time, lack of facilities and costs are the main reasons people give for being inactive (Shephard, 1997). It is important that children are encouraged to be more physically active, not only for the short-term effects

on their well-being and body weight (Dietz, 1996) but because adult habits are formed predominantly in childhood (Hillman, 1993; Trippe, 1996).

Shephard (1997) calculated that cycling at 16km/h uses 29kJ/min on average, adequate for health benefits. According to Hillman (1993b), regular cyclists have a level of fitness equivalent to that of people 10 years younger although a Finnish study found that while physically active men and women (including cyclists) had the explosive muscle power of someone 10 years younger, 55-year-olds had the aerobic fitness (VO_{2max}) of people of the same sex aged 25 years (Kujala, 1994).

Fentem (1994) stated that brisk walking will fulfil the recommendations for physical activity although Shephard (1997) believes this is only the case for the elderly. Shephard reviewed whether active daily living could provide the cardiovascular and other health benefits obtained from vigorous aerobic activity. He noted that the most recent reduction of recommended exercise intensity, 50% of an individual's maximal oxygen intake, sustained for one hour three to five times per week, would be achievable through walking for older people but that young adults were unlikely to reach that intensity of activity through walking. However, the greatest health benefits are gained by increasing activity levels in elderly, sedentary or obese individuals. For them, (fast) walking is likely to be sufficiently effective as well as the most acceptable form of activity. Shephard calculated that young and middle-aged individuals would need to walk at the unrealistically fast rate of 8.4km/h, unless they walked on soft or uneven terrain: walking up a 5% gradient increases energy expenditure by 50% while walking downhill uses only slightly less energy than walking on the level.

Walking is the most important physical activity for the elderly (Barry and Eathorne, 1994). Only the very frail or those with serious disabilities cannot participate in this form of sustained dynamic aerobic activity (Morris and Hardman, 1997). Walking a mile in 20 minutes (slower than the 3.5 – 4.5 mph most often recommended for health benefits to accrue to the middle-aged (Lumsdon and Mitchell, 1999)) expends the same amount of energy as cycling at 9.4mph for 16 minutes, running a mile in 10 minutes, swimming breast stroke for 10 minutes, playing squash for 8 minutes, medium-intensity aerobic dancing for 16 minutes, playing football for 12 minutes or golf for 19 minutes, or using weight-training machines for 17 minutes (Greater Glasgow Health Board, 1999). Regular "brisk" or "fast" walking can improve cardiovascular fitness (Morris and Hardman, 1997).

Hillsdon and colleagues' review of randomised trials of physical activity promotion found that whether individuals were prescribed high or low intensity activity, they tended to move towards moderate intensity activity (Hillsdon et al. 1995).

A series of Finnish studies have explored the feasibility and effectiveness of physically active commuting. Oja and colleagues found that half their sample of working-age people in Tampere, a city of 200,000, worked outside the home. One-quarter of these commuted by foot or bicycle in the autumn and winter and one-third in the summer. More women than men walked or cycled. Most physically active commuters reported their journey caused slight sweating and shortness of breath. A 10-week randomised trial of volunteers who had previously commuted by car or bus found mean walking speed increased from 5.8km/h at the start of the study to 6.2km/h, with V_{O2max} 53% initially, reducing to 51% (mean distance 3.4km). For cyclists, the equivalent figures were 17.6km/h and 65% of V_{O2max} at the start, changing to 20km/h and 62% (mean distance cycled 9.7km). The volunteers walked or cycled for more than three-quarters of their commuter journeys in the 10 weeks. Measured

effects of this activity were a net 10.3% increase in maximum treadmill time in the active group and a 5% increase in HDL cholesterol. This physically active commuting also increased fitness. Cycling was more vigorous than walking and produced greater effects but walking at participants' favoured speed also produced measurable changes (Oja et al. 1998).

5.2.5 Risks

Whilst physically active transport does give rise to many benefits, there are also risks.

Reported risks of exercise

Cardiovascular effects

Ventricular arrhythmias are not uncommonly provoked by activity, particularly in those with pre-existing myocardial damage due to CHD. Care must be taken when advising patients with cardiac failure to be more active (Royal College of Physicians, 1991). Supraventricular tachycardia is less common but some people have a tendency for such attacks to be precipitated by vigorous activity. Beta-blocking drugs can be of benefit, allowing normal physical activity in such young people (Royal College of Physicians, 1991).

Sudden cardiac death occurs occasionally during vigorous activity, even in those with no previous symptoms of myocardial ischaemia and no CHD risk factors (Royal College of Physicians, 1991). It is even more uncommon in people under 30, in whom the underlying cause is usually hypertrophic cardiomyopathy or a congenital abnormality of the heart or conducting system. In those over 30, it is almost always due to CHD (Firor and Faulkner, 1988). Firor and Faulkner quote estimates of the incidence of sudden death during vigorous activity, ranging from 3.5 per 100,000 man-years in British Armed Forces personnel to one per 16,000 person-hours of exercise in cardiac rehabilitation programmes. The highest rate found was one in 3,000 hours for rugby referees. Studies have found one sudden death while jogging per 396,000 man-hours, equivalent to one death per year per 7,620 joggers. Others have found higher rates or none. Firor and Faulkner quote the studies by Siskovick et al., who found the overall incidence of primary cardiac arrest to be 55 to 60% lower in people with high leisure time physical activity compared to those with low leisure time physical activity. They also found that although the risk of sudden death was higher during exercise than while inactive, the overall risk of primary cardiac arrest in those who are habitually active and physically fit was only 40% that of sedentary men. Even after allowing for conventional risk factors, the overall risk of cardiac arrest is lower among habitually vigorously active men than sedentary men. This is because a relatively small proportion of the time is spent in vigorous activity while the protective effects last all day(Firor and Faulkner, 1988).

Two cohort studies have found that the most active men had an increased risk of CHD. Further analysis of one showed tht this applied only to men with hypertension (Whaley and Blair, 1995).

Older, previously sedentary individuals can safely be advised to walk more (Royal College of Physicians, 1991) and to increase gradually the distance travelled and/or the speed at which they travel. Individuals should be warned to heed symptoms such as chest pain, an irregular heart beat, faintness or shortness of breath. Vigorous activity should also be avoided during viral infections such as influenza (Royal College of Physicians, 1991).

Ischaemia can be prevented by warming up before and warming down after a period of activity (Royal College of Physicians, 1991). Post-exercise hypotension (low blood pressure) can be a problem in elderly people. It can be reduced by warming down after a period of physical activity (Royal College of Physicians, 1991).

Hypo- or hyperglycaemia

Exercise can precipitate hypoglycaemia in insulin-dependent diabetics unless additional carbohydrate is taken (Royal College of Physicians, 1991). In the presence of insufficient insulin, exercise can cause hyperglycaemia and ketosis (Royal College of Physicians, 1991).

Respiratory symptoms

Activity can induce symptoms in 70 – 90% of children with asthma, 30-40% of non-asthmatic individuals with atopy, and 15% of high performance athletes. Standard therapy before activity usually abolishes or at least alleviates the problem (Royal College of Physicians, 1991). Walking is unlikely to cause asthmatic symptoms except in very cold weather.

People with other respiratory diseases may have their exercise tolerance limited by respiratory symptoms but physical activity within their own limits should not produce problems (Royal College of Physicians, 1991).

Musculoskeletal injuries

One series of elderly people taking up jogging found that nearly half had sustained injuries in a two month period (Royal College of Physicians, 1991).

Other injuries can occur: death of runners due to collision with motor vehicles is almost as likely as sudden cardiac death (Firor and Faulkner, 1988).

Incapacity - risks of inactivity

The above risks need to be weighed against the risks of inactivity, which leads to a progressive reduction in the capacity for physical exertion. Greater effort becomes necessary for shorter and slower activity, with fatigue developing faster. Both muscle strength and cardiovascular fitness are affected by prolonged or habitual inactivity (Royal College of Physicians, 1991). Much of the deterioration of function attributed to increasing age may actually be due to decreasing activity, leading to a worsening in capacity to exercise. A vicious spiral develops (Barry and Eathorne, 1994).

Risks of physically active transport

Lower intensity activities, such as walking and cycling, are less likely to result in any of the above (Kriska and Bennett, 1992). Low impact exercise (including running and jogging) does not increase OA except in those with a history of knee injuries or anatomical abnormalities (Lane, 1995). While cycling can be injurious to those with OA of the knee, walking is the exercise of choice for people with arthritis (Restivo, 1991). Indoor walking can improve walking pace in elderly people with OA, improving their daily activity score, their perception of ability and disability and their use of drugs. Walking at any speed is low impact and is also

self-regulated in intensity and duration, so the risk of injury is low (Morris and Hardman, 1997).

Road traffic accidents

This risk is important, though there are studies that have indicated the benefits of cycling far outweigh the risks from accidents (British Medical Association, 1997). Hillman (1993) reported that more than 80,000 cyclists are injured each year in the UK, with a fatality rate of one in every 20 million km cycled. He also pointed out (1992) that CHD accounts for 33% and motor-vehicle traffic accidents for 1.4% of all deaths among commuters. The latter figure is non-significantly lower for cycle commuters, indicating that they are not at undue risk.

Using road accident and actuarial data, Hillman calculated that the 294 cyclists fatally injured in 1989 lost 11,324 years of life (Hillman, 1992) p112). Reduction in premature mortality by regular cycling can also be calculated from data similar to those in the earlier sections of this chapter, for example that cycling 60 miles per week from the age of 35 could add two-an-a-half years to life expectancy. Relating these data to the number of people shown in the National Travel Survey to be regular cyclists, Hillman (1993) calculated that the benefits of regular cycling outweigh the years of life lost in fatal injuries by around 20 to one. He also pointed out that providing safer cycle routes would reduce the risk of serious injury and would encourage more people to cycle who are currently dissuaded by it apparent danger.

Non-fatal injuries requiring in-patient treatment were mostly of moderate severity, with an average stay in hospital of only two days in 1983 for those requiring admission to hospital . Less than 20% were severe injuries. No longitudinal studies on such individuals have been conducted, to determine the effects on life expectancy, quality of life or residual disability. Hillman therefore did not add any life-years lost for non-fatal injury to his calculation. Although this is a criticism of the calculation, it is probably balanced by not including beneficial effects of regular cycling on health and well-being not affecting premature mortality.

In the Tampere study of physically active commuting, 7% of women and 5% of men reported having had an accident in light traffic in the previous year. No details were given of severity (Oja et al. 1998).

Data on London specific accident rates were given in Chapter 2. Injuries are also caused by tripping on uneven pavements. Morris and Hardman (1997) reported that nationally 6.3-8.4m of British adults had tripped or fallen on damaged pavements or on snow, ice, etc.

Air pollution

Air pollution is hazardous to health (chapter 3). Many people believe walking or cycling along a road increases their exposure to air pollution (Hazucha et al. 1994). However, exposure is likely to be as great, if not greater inside vehicles. In congested roads, cars take in air from the exhaust of the vehicle in front and concentrations in-car can be higher than the surrounding air (AEA Technology Environment, 1999) where dispersion of pollutants reduces levels. where dispersion of pollutants occurs. For exposure to a given concentration, cyclists have a higher dose because of breathing faster or more deeply. As they are usually riding at the side of the road, the concentration to which they are exposed is lower so overall their dose is similar to or lower than motorists (Taylor and Fergusson, 1997). However, there

is an additional factor: time of exposure. The effect of this parameter is difficult to assess. It may be that cyclist and pedestrian have a heavier exposure to pollutants from their slower speeds (apart from very congested areas). It may also be that the travel time (e.g. from home to work is on average longer than for private or public transport).

5.2.6 Exposure-Response Relationships

The above studies show the positive link between physical activity and a number of health outcomes. In order to be used in a health evaluation framework, a number of additional issues are important with respect to the nature of the exposure response functions.

Is physical activity or fitness more important?

Andersen summarised five studies in which both activity and fitness were assessed. Physical fitness appears to have a graded inverse relationship with CHD in healthy middle-aged men, after adjustment for level of physical activity. However, in another study, after dividing people into quintiles of fitness, active men had lower death rates than inactive men within each quintile. Fitness predicted future CHD only in active men but not in inactive men (Andersen, 1995). The conclusion seemed to be that fitness is the more important factor but that physical activity is also required for fitness to be protective against CHD.

Shephard (1997) postulated that although the results of most studies could mean that aerobic fitness must be increased to enhance health, another interpretation is that patterns of physical activity are indicated better by measurement of fitness than by activity questionnaires.

Whaley and Blair (1995) also found a significant inverse graded relationship between physical fitness and CHD but did not discuss whether this or physical activity was more important. They gave age- and sex-specific thresholds for activity that marked low fitness associated with a significant increase in risk. These were below 8-9 METS for middle-aged men and 6-7 METS for women. This contrasts with the level of activity Shephard (1997) reported as fulfilling the US recommendations (50% of aerobic power for one hour, three to five times per week). He calculated that an average 75kg middle-aged man would need to expend 5 METS (27kJ/min, 6.5kcal/min) but an elderly or unfit man would need to use only 3 METS (19kJ/min, 4.5kcal/min).

Is lifetime, previous or current activity more important?

Lee and Paffenbarger (1996) commented on the results of the Harvard Alumni studies, with assessments of activity in the 1960s and 1977 and follow-up to 1985. Mortality was the same in men who were active (≥2,000kcal/wk) at both assessments or just at the more recent one, around 15% lower than in those who were less active at both assessments, after adjusting for age, cigarette smoking, hypertension and BMI. In the ACLS, however, fitness at both assessments predicted the lowest age-adjusted mortality (RR 0.33 (0.23-0.47) cf. unfit at both examinations) with those who were fit at one and unfit at the other assessment having a similar mortality (RR 0.5 (0.4-0.7) (Lee et al. 1995).

The Health Education Authority has concluded that there is strong evidence that current physical activity, not previous activity in early adult life, is beneficial for cardiovascular health (Health Education Authority, 1995).

- Improvements in physical fitness could reduce risks of death by 60% (more than the 50% risk reduction from quitting smoking (Lee and Paffenbarger, 1996).

Is there a threshold?

There is some evidence that no threshold exists and even moderate increases in amount of physical activity will have benefits (Health Education Authority, 1995). Shephard (1997) is one of many reviewers to note that most recent reports suggest that the greatest gain in health benefits occurs when sedentary or unfit people start being active enough to move to the next lowest category of activity or fitness, although a few reports still maintain that vigorous activity is required.

The RCP 1991 report cited one study that found a continuous graduated benefit of increasing levels of activity but the report suggests that there is probably a threshold of a minimum amount of activity to reduce the risks of developing CHD. This might be 2,000kcal/week (the equivalent of five hours' brisk walking or three and a half hours' running). Another study quoted found benefits only in those taking vigorous aerobic activity, expending 420kcal/hr twice weekly. Walking, cycling and stair climbing were not of benefits unless pursued vigorously (Royal College of Physicians, 1991).

The Whitehall study is frequently quoted as demonstrating that vigorous activity is required to produce health benefits. It found a halving of the risk of CHD but only in those participating in vigorous exercise, with no benefit from moderate activity (Andersen, 1995). However, Morris has stated that "a brisk walk of 30-60 min each day" would have been graded as "vigorous" in that study (Morris, 1994).

The mortality of male Harvard alumni who took part in moderately vigorous activity in the 1960s and in 1977 or just in 1977 was 29% (95% CI 4-45%) and 23% (4-42%) lower respectively than those who were less active at both assessments, after adjusting for age, cigarette use, hypertension and BMI (Lee and Paffenbarger, 1996).

Is the effect linear?

It is generally thought that further activity increments further reduce risk. Among people who are already active, vigorous activity probably adds benefit, though there is a potential risk of injury (see above). The MRFIT study found higher mortality in people in the lowest tertile of activity but no difference between people in the top and middle tertiles (Andersen, 1995). Other studies have found a linear relationship between fitness and CHD or mortality, independent of other CHD risk factors, age, blood pressure, glycemic status, anxiety or depression (Andersen, 1995).

Henriksson (1995) reported that when total energy expenditure was held constant, low- and high-intensity activity were equally effective in increasing insulin sensitivity.

What are the correct endpoints?

Health benefits will include mortality and morbidity effects. The analysis of the benefits from reductions in coronary disease raise similar issues to air pollution related mortality. The risk of coronary heart conditions will be higher in the susceptible population (which will include sedentary people) - activity will reduce those risks, but does not extend life expectancy

indefinitely. Therefore, as with air pollution, the correct endpoint is the gain in life years, rather than a reduction in mortality per se. This has particularly important implications should later stages of the work want to consider monetary valuation. To properly quantify these factors data are needed on the extension in life expectancy with regular physical activity, taking into account age-dependence and other factors (smoking, diet, etc). There is some information on these issues with respect to air pollution (see Chapter 3) and some specifically related to activity, discussed below.

A meta-analysis of Finnish studies found the lowest and highest adjusted relative risks for CHD deaths among Finnish men aged 30-63 years who were sedentary (physically active less than four times per week) to be 1.4 and 1.9. They calculated the population attributable risk of a sedentary lifestyle for CHD deaths and found that between 22 and 39% of CHD deaths in that population would be prevented if the individuals were sufficiently active (Haapanen-Niemi et al. 1999).

Powell and Blair (1994) calculated the effects on deaths from CHD, colon cancer and diabetes assuming various changes in levels of physical activity, using relative risks derived from the published literature. Relative risks and population attributable fraction (for the USA) are given in Table 5.2 and the percentage change in deaths of increased activity in Table 5.3. As the Allied Dunbar Fitness Survey (1992) found higher, though still inadequate, levels of physical activity (page 95), the population attributable fraction and the deaths prevented will be a little lower in the UK.

Table 5.2 Estimated Relative Risks and Population Attributable Risk of Inactivity (in USA)

Exposure group	**Prevalence of activity level in USA, 1990**	**CHD**		**Colon cancer**		**Diabetes**	
		RR	**PAR**	**RR**	**PAR**	**RR**	**PAR**
Sedentary	24%	2.0	16%	1.8	13%	1.8	12%
Irregular	54%	1.5	18%	1.5	18%	1.6	21%
Regular	10%	1.1	1%	1.1	1%	1.3	2%
Vigorous	12%	1.0	-	1.0	-	1.0	-
Total population risk attributable to inadequate physical activity			35%		32%		35%

Source: (Powell and Blair, 1994)

Table 5.3 Estimated percentage reduction in deaths from CHD, colon cancer and diabetes for different increases in physical activity (based on activity levels of USA1990).

Disease	**Reduction in deaths if:**			
	50% of Sedentary become Irregular	**50% of Irregular become Regular**	**50% of Regular become Vigorous**	**50% of each category move to next category**
Coronary heart disease	3.9%	7.1%	0.3%	11.4%
Colon cancer	2.5%	7.4%	0.4%	10.1%
Diabetes	1.5%	5.2%	1.0%	7.8%

CHD, colon cancer and diabetes combined	3.6%	7.0%	0.4%	11.0%
All-cause mortality	1.0%	1.9%	0.1%	3.0%

Source: Powell, 1994

This information provides a useful starting point for examining these issues, though it is not possible to follow this through to quantification within the time-scales of this study.

5.3 Quantification

5.3.1 How might estimate physical activity benefits of transport

The above review shows that there are beneficial effects from cycling and walking. The key question for development of a framework is whether it is possible to use the evidence to derive dose-response functions, and then how we can apply these to look at the issue of exercise. The latter is particularly difficult within a generic framework. It might however be easier when looking at the effects of specific schemes, for example, examining incremental changes in transport by mode.

A number of issues need to be assessed in any quantification framework, discussed below.

5.3.2 Background activity patterns

In order to provide quantification using the types of relationships described above, information is needed on the existing activity patterns of the relevant population. These activity factors are in turn determined by societal and individual factors. Information on background rates can be taken from *Our Healthier Nation* (DoH). A few key factors that are relevant are presented below.

The Allied Dunbar National Fitness Survey (1992) identified adult population groups who were particularly sedentary, these were:

- Young women aged 16-24 years.
- Middle aged men.
- Older men and woman aged over 50 years.

Population groups with special access and/or communication needs were also identified, these were:

- People from black and minority ethnic groups
- Disabled People

In England in 1990-91, 29% of men and 28% of women aged 16-74 (equivalent to 5,086,800 men and 4,955,600 women in 1993) were leading a sedentary lifestyle, i.e., engaging in less than one session of 30 minutes of continuous moderate intensity physical activity a week. Only thirty-six percent of men and 24% of women (equivalent to 6,314,700 men and 4,247,600 women in 1993) were regularly active at a moderate level, the level recommended for health benefit.

Older people are more likely to lead a sedentary lifestyle than younger people – among 16-24-year-olds, 16% of men and 25% of women are sedentary, while among 65-74-year-olds, 55% of men and 54% of women are sedentary.

There are also concerns as levels of physical activity in children are declining. The prevalence of being overweight or obese is increasing in children in the UK. Many young people possess at least one modifiable CHD risk factor; and many young people have symptoms of psychological distress.

Cross-national studies of school-aged children indicate that between the ages of 11-15 there was a marked decrease in the proportion of girls who were active daily, while among the boys there was less change.

Fifty-six per cent of men and 52% of women believe they do enough exercise to keep fit, while only 36% of men and 24% of women are active at a regular moderate level.

The main motivating factors for taking part in physical activity are: to feel in good shape physically, to improve and maintain health, to feel a sense of achievement and to get out-of-doors.

Over 40% of women and women cite `lack of time' as the main barrier to them taking more exercise. Thirty-seven per cent of women and 24% of men say that not being `the sporty type' stops them getting more exercise.

With this study we have not undertaken a detailed review of this information and stress other information on background prevalence is available In some cases, e.g. in London, there is also local specific data, which may provide more representative information.

5.3.3 Uncertainty

There are a number of problems in taking effect estimates from the published literature to use in a health impact assessment.

Bias in published reviews

It must be emphasised that this chapter is based on published reviews of primary studies. Most did not mention their search strategy or indications for including or excluding studies. Many were written before there were established principles for systematic searching for studies, evaluation of the quality of studies for exclusion, or agreement on how to conduct meta-analysis and the developments of graphical tests to check for publication bias (Eggar, 1997; Taylor, 1998a). Their conclusions may not be those of a rigorous systematic review.

Where possible, studies of the effects of walking, jogging or cycling but not of other forms of physical activity have been used in this review as these are the only forms relevant to physically active transport. In some cases, authors have not specified the type of activity in which participants engaged. Particularly where studies were of more vigorous activity, the reviews will have overestimated the benefits to health of physically active transport.

A final problem with using published reviews is that by definition, they are more out-of-date than the original studies on which they are based.

Bias in individual studies

Although the issues below must be considered, Shephard (1997) noted that in general, the stronger the study design, the larger the gradient in risks for all-cause and cardiac deaths.

Generalisability

Volunteers in studies tend to be younger, fitter and leading healthier lives than the general population. Even if the results are qualitatively generalisable to the rest of the population, quantification may be misleading.

Measurement bias

Many different measurements of frequency, duration and intensity of physical activity have been used in different studies, some of which have been well-validated (Paffenbarger et al. 1993).

Kriska and Bennett (1992) discussed some of the problems of measuring physical activity levels in epidemiological studies, not only because of different methods of making assessments but also because many studies consider only either leisure or occupational activity, and the latter often excludes unpaid occupations. They also pointed out that shorter time-scales reduce recall bias but may be less accurate in recording "usual" activity levels, particularly when there is seasonal variation, thus increasing the risk of misclassification bias. However, they also noted that questionnaire responses usually correlate well with monitored data.

Total activity undertaken

Studies that include only occupational or leisure activity may exclude substantial episodes of physical activity, particularly in women (Weller and Corey, 1998).

Intensity of activity

Depending on an individual's level of fitness, the same activity will require different energy expenditure. For example, walking deliberately through a shopping mall could require 70-80% of maximal oxygen intake (Shephard, 1997). Similarly, the energy expended in any activity depends partly on body weight. For example, walking a mile in 20 minutes uses 90kcal for someone weighing 56kg, 108kcal if 68kg and 132kcal if 83kg (Greater Glasgow Health Board, 1999).

Confounding

Co-existence of healthy behaviours

Many studies have shown the association between healthy behaviours. Those who choose to be physically active are also more likely to eat a healthy diet (Matthews et al. 1997) and are less likely to smoke (Shephard and Shek, 1998). They are therefore at lower risk of most diseases and would be expected to have lower morbidity and mortality than other groups.

Similarly, those who are unable to be physically active, due to financial, mental or physical barriers, are likely to have worse health (Compton et al. 1989).

Selection bias

Many studies have been cross-sectional, demonstrating an association between those who are more active and their current health status. Others have been case-control studies of those with disease. These study designs do not allow the direction of the association to be examined: they cannot exclude the theory that those with poor health are less able to be physically active. Several cohort studies have examined different time-periods within the study. Some have found the relationship persists, such as in three consecutive three-year periods in the Whitehall study (Lee and Paffenbarger, 1996). Some have found a reduced relationship in later periods, such as the Finnish study reported by Lee and Paffenbarger (1996). This may be due to those who are already ill being less physically active. However, other explanations are possible. The increasing length of follow-up reduces the likelihood of individuals remaining within their initial category of activity level, thus underestimating the relationship if current activity is more important than former activity. Another possibility is that those who are at highest risk from a sedentary lifestyle may die sooner so the relationship persists but attenuates.

Loss to follow-up

Most studies have had good or excellent follow-up, particularly those discussed by Lee and Paffenbarger (1996).

Differential effects for different groups

The effect of a given activity may differ depending on:

- Previous level of activity;
- Previous fitness;
- Current fitness;
- Age;
- Sex;
- Health status.

Background rates of physical activity

Although these are addressed above, studies would be required to assess whether those who changed their mode of transport to walking or cycling (or jogging) were those who were previously sedentary, insufficiently active, sufficiently moderately active, or took regular vigorous activity. It would also be important to know whether physical activity through transport replaced or was additional to other physical activity.

5.3.4 Recommended Framework for Quantification

The evidence presented above, along with the data on background health status do mean it would be possible to develop some degree of quantification for the health benefits of physical activity. Nonetheless, important questions remain on issues of transferability, on thresholds and on linearity with respect to developing these into exposure-response functions. There is one other issue here that is relevant for transport. An additional step is needed to translate

functions to levels of transport activity. This is likely to be easiest for studies which look at specific changes in transport levels (e.g. from the introduction of new schemes or policies, where the modal shift can be estimated). Even so the nature of the evaluation will be very greatly affected by the nature of the proposed change. A evaluation that is appropriate for, say, a local road improvement will be different from the requirement for the evaluation of, say, a proposal to introduce a congestion charging scheme or a flat, London-wide bus fare.

5.4 Current Benefits from Transport Related Physical Activity in London

Data is available on the number of people who commute to work in London (DETR, 1999).

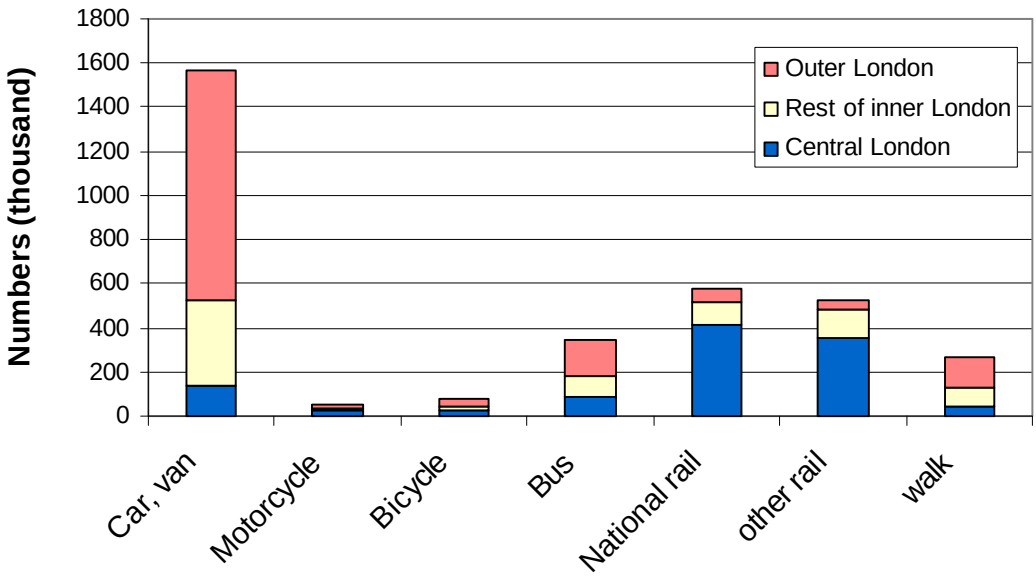
Table 5.4 Main Mode of Transport to Work 1998.

	Percentage						
Area of workplace	**Car, van**	**Motor-cycle**	**Bicycle**	**Bus**	**National rail**	**other rail**	**walk**
Central London	13	2	2	8	38	32	4
Rest inner London	46	2	3	11	12	16	10
Outer London	69	1	2	11	4	3	9
London	46	2	2	10	17	16	8

	Numbers						
Area of workplace	**Car, van**	**Motor-cycle**	**Bicycle**	**Bus**	**National rail**	**Other rail**	**walk**
Central London	141,960	21,840	21,840	87,360	414,960	349,440	43,680
Rest of inner London	381,340	16,580	24,870	91,190	99,480	132,640	82,900
Outer London	1,046,040	15,160	30,320	166,760	60,640	45,480	136,440
London	1,581,020	68,740	68,740	343,700	584,290	549,920	274,960

Source: DETR, 1999.

The values (presented graphically below) show the dominance of private vehicle use, especially in outer London.



Nevertheless, a total of 343,700 people walk or cycle to work every day. We can assume all of these receive some health benefit, though translating this number into quantitative numbers is difficult. To do this, information is needed on the length of trips (e.g. do they offer exercise benefits such as the 30 minutes of activity of moderate activity) and information on the existing health state and exercise regimes of these people. Some information is available on the former (DETR, 1999), giving data on travel times, though the data is not split by mode.

We can however demonstrate that the benefits are likely to be large.

The annual numbers of deaths in London from all cause mortality, cardio- vascular morality and cancer are shown below:

	Cases/year
All cause mortality	61,685
Cardio- vascular mortality	26,645
Cancer (all cause)	~18,000

The data above shows that currently around 343,700 people commute by bicycle or walking per year (5% of London's population).

For this sub-set of the population, physical activity from travelling to work is likely to lead to a reduction in life-time risk by up to one half for the endpoints above. Further work is needed to equate risks in equivalent terms to air pollution related health effects and transport accidents[18], though we conclude the benefits maybe of a similar magnitude (i.e. several hundred deaths avoided per year). Expressed using one of the alternative quantification indices, these people who cycle or walk to work might be expected to extend their life by around 2 years. Again this equates to a broadly equivalent life extension as lost from current levels of air pollution in London (i.e. several tens of thousands of life years). We highlight the derivation of specific numbers for these endpoints as one of the immediate research priorities following on from the study.

5.5 Future Research Recommendations

Research priorities are separated into underlying research and policy relevant research. For the former we highlight:

1. The need for a series of systematic reviews and meta-analyses of primary studies to answer the question "to what extent can physically active transport influence health and disease outcomes?" For cancers, this would entail updating the existing meta-analyses.
2. Specific work to try and get sensible functions and relate to frameworks. This includes derivation of baseline factors for future analytical frameworks.

More immediate and policy relevant aspects are highlighted as:

1. The derivation of specific numbers for London for the endpoints listed;
2. The use of the numbers to look at specific policies, i.e. what benefits might you see with modal shift to cycling and walking. This could be extended to investigating policies to say how benefits can be maximised, whilst reducing the risk of detrimental effects.

[18] Further work is needed to get risk factors and incidence in equivalent terms and to annualise for comparison. Work is also needed to look at risk by sub-groups and confounders in more detail before numbers are presented.

3. Considering the relative risks of exercise benefits against accident risk and air pollution exposure specifically for London, as well as comparing against other areas (e.g. quitting smoking).
4. The interaction of promotion of exercise in the context of other dimensions of policy: safety; public transport capacity, e.g. how much public transport capacity is taken up by trips that could equally be walking trips especially in London? Is there a risk that lower fares and improved services would actually encourage less physical activity? What additional benefits are there in targeting the elderly to keep them mobile (for whom benefits may be greater)?

Points 1 and 2 are highlighted as research priorities for follow-on from the current study.

5.6 Conclusions

Physical activity has significant health benefits in reducing coronary heart disease and in reducing other health impacts such as diabetes, obesity, hypertension, osteoporosis and even depression. There is also increasing evidence linking increased physical activity with decreased incidence and mortality from cancer, especially cancer of the colon. Transport (through the encouragement of cycling and walking) can therefore provide significant health benefits.

These benefits are currently being encouraged through national and regional programmes, such as the Health Education Authority's 'Active for Life' campaign. The benefits are realised through 30 minutes of activity of moderate activity on most, preferably all, days of the week. Physical activity is recommended (rather than participation in organised sports or exercising) because most people can incorporate moderate physical activity into their daily routine – and it is in this respect that there is a link between transport and health.

This chapter has reviewed the evidence for the health benefits of physical exercise. All health benefits are described although some have not been quantified. For many studies, the type of activity is not specified or the activity level is a composite score from different activities during a week and this has meant it has been difficult to define specific exposure-response relationships that are relevant for assessing the health benefits of cycling and walking.

The quantification evidence is summarised below:

- There is an inverse causal relationship between physical activity and all-cause mortality. For example, the most active or fit have mortality rates that are 17 to 78% lower than the least so, comparable to the difference in mortality of non-smokers and smokers or between people of ideal weight and 20% heavier (Lee and Paffenbarger, 1996). These equates to a benefit gained by high physical activity of around two years of life. There is also evidence of a lesser but probable reduction in all-cause mortality in the elderly who start being vigorously active (Boyer and Vaccaro, 1990). Physical activity programmes after a heart attack can also reduce the risk of death by about 20% (Health Education Authority, 1995)
- Physical activity reduces the risk of developing coronary heart disease by 25-50% in men (Whaley and Blair, 1995). This could result in major benefits - just under one-third of all CHD incidence and one-quarter of stroke incidence could potentially be avoided by appropriate physical activity (Health Education Authority, 1995).

- Physical activity helps to defer or prevent the development of type 2 diabetes mellitus (Health Education Authority, 1995) and it estimated that just under one-quarter of cases of type 2 diabetes could be avoided in those aged 45 and over (Helmrich et al. 1994).
- Physical fitness has a marked inverse relationship with cancer mortality. For example, physical activity could reduce all-cause cancer rates by as much as 46% (Shephard and Futcher, 1997). The greatest benefits are seen in the association between physical activity and reduction in cancer of the colon (large bowel). There is also positive, but weaker benefits for breast cancer, endometrial cancer, prostrate cancer and cancer of the testes.
- Physical activity in elderly men and women protects against hip fracture. In those aged 45 and over, just over half the hip fractures could be avoided (HEA, 1995).

There are also a number of other benefits from physical activity, for which quantified estimates of benefits are not available including:

- Participation in strength and weight-bearing activities is positively associated with bone mineral density and may be related to a reduced long-term risk of osteoporosis.
- Those who are regularly physically active feel less depression, tension, fatigue and aggression and sleep better. Regular participation in physical activity is also associated with raised self-esteem in children and adults. Regular aerobic activity is of benefit in treating mild depression and mild anxiety.
- Physical activity has been shown to have many beneficial effects in later life, including improvements in balance, co-ordination, mobility, strength and endurance and in the control of chronic disease.
- Aerobic activity also has a role in reducing weight and especially in causing weight loss for the obese.

These benefits would be realised through cycling to work. The picture for walking is not as clear cut. Some studies state that brisk walking will fulfil the recommendations for physical activity although others state this is only the case for the elderly. As the greatest health benefits are gained by increasing activity levels in elderly, sedentary or obese individuals, walking will have major benefits, though it is possible that young adults may not reach the necessary level of intensity of activity through walking alone.

There are however, some risks from cycling and walking. These include risks from exercise such as precipitating cardiovascular or respiratory symptoms (e.g. angina or wheezing), though for both of these the potential benefits are much greater. They also include musculoskeletal injuries, though cycling and walking are low impact activities and so less likely to lead to these problems relative to other activities (e.g. jogging). Of potentially greater concern is the risk from traffic accidents. Previous chapters showed that pedestrians and cyclist are particularly vulnerable to traffic accidents. Nevertheless, studies show the benefits of regular cycling outweigh the years of life lost in fatal injuries by a factor of around 20 to 1. There is a potential risk from air pollution, though the evidence indicates that in-vehicle pollution concentrations are as great or greater than roadside pollution.

Although some quantitative estimates of benefits are available, there are still some issues that have been identified for their application as exposure-response functions. The most apparent of these is the lack of data on the linearity of functions and the presence or absence of thresholds of effects. Moreover, the effects on modal shift and travel patterns in response to specific projects or policies are not well characterised. Finally, there are also important questions over how to assess background health status and activity levels, though we note that

by looking at specific policies, options or schemes, most of these background issues could be addressed and quantitative estimates could be made.

The study has also looked at how important these potential benefits might be in London. Further work is needed to equate risks in equivalent terms to air pollution related health effects and accidents. Nonetheless, initial calculations indicate the ***benefits*** of physical activity through transport may be of a similar order to the ***dis-benefits*** of accidents or to air (i.e. physical activity from transport may results in several hundred deaths per year avoided and several thousand years of life added to the population). We highlight the derivation of more accurate versions of these numbers as one of the immediate research priorities following on from the study.

This chapter presents one of the most interesting aspects of the current study and one that has received relatively little attention to date. It is highlighted as warranting a focus for follow-on studies in general. We have however highlighted a number of specific areas. These are:

1. The need for a series of systematic reviews and meta-analyses of primary studies to answer the question "to what extent can physically active transport influence health and disease outcomes?" For cancers, this would entail updating the existing meta-analyses.
2. Specific work to try and get sensible functions and relate to frameworks. This includes derivation of baseline factors for future analytical frameworks.
3. The derivation of specific numbers for London for the endpoints listed;
4. The use of the numbers to look at specific policies, i.e. what benefits might you see with modal shift to cycling and walking. This could be extended to investigating policies to say how benefits can be maximised, whilst reducing the risk of detrimental effects.
5. Considering the relative risks of exercise benefits against accident risk and air pollution exposure specifically for London, as well as comparing against other areas (e.g. smoking).
6. The interaction of promotion of exercise in the context of other dimensions of policy: safety; public transport capacity, e.g. how much public transport capacity is taken up by trips that could equally be walking trips in London? Is there a risk that lower fares and improved services would actually encourage less physical activity? What additional benefits are there in targeting the elderly to keep them mobile (for whom benefits may be greater)?

Points 3 and 4 are highlighted as research priorities for follow-on from the current study.

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6 Community Severance, Inequality and Other Effects

6.1 Introduction

As stated earlier in this report, this study aims only to look at the direct effects of transport on health. It does not set out to assess the significant benefits transport has on health (indirectly) through access to goods and services, and through economic and social development. These benefits are however extremely important and must be included when looking at transport more generally.

The direct effects of transport this chapter is considering are often grouped under the generic heading 'inequality'. Care must be taken with this term, as it has different implications for economists, health specialist, etc. Given the multidisciplinary nature of this project, we have tried to be more specific in our definitions about which issues we are considering.

The main aspect we have included in this review is community severance. We have also tried to relate the inequality effects of all the other categories of effects considered in the study, i.e. with respect to accidents, air pollution, noise and physical activity. In this context inequality sometimes is with respect to income, sometimes with respect to societal class or age group. Finally, we have also tried to assess other possible direct inequality effects from transport, such as social or economic exclusion and also tried to discuss some of the other psychological effects of transport.

It should be noted that the effects that are discussed in this chapter are very different to the effects described in previous chapters. Although transport may have a direct effect (e.g. a new road leads to community severance), the actual health effects are often secondary and arise as an indirect consequence (e.g. severance may impact on well-being and quality of life, which in turn has an influence on mental health). Because of the chain of effects between transport activity and actual health endpoint, the issues here are more difficult to assess and even harder to quantify than with other chapters.

Because of these difficulties, there is a tendency for researchers to assume transferability from specific studies, when in practice, effects are extremely site specific. This chapter therefore reviews the general evidence, but also tries to assess potential issues within a London specific context.

6.1.1 Method of Review

This is a rapid review. It is based on two recently published reviews (Shaw *et al*, 1999: Marmot & Wilkinson 1999), together with papers from scientific journals found during an earlier search of Medline and HMIC using the text words "community severance", "social network", "social support" and "social relationship".

6.2 Community Severance

6.2.1 Definition and Introduction

Community severance arises when roads bearing high levels of traffic cut through housing areas. The physical presence of the traffic, particularly heavy goods vehicles, as well as the risk of accidents presents a barrier to the community, limiting or disrupting interpersonal networks and reducing social contact. The Department of Transport's Manual of Environmental Appraisal defines community severance for trunk roads as: *"the separation of residents from facilities and services they use within their community, from friends and relations and, perhaps, from place of work as a result of changes in road patterns and traffic levels."*

Reviews of community severance indicate that geographical areas are not inherently socially cohesive and that severance may already exist and does not require new changes to occur. Clark *et al* (1991) stated that: *"severance is perceived by the public as a number of effects including pedestrian delay, trip diversion and suppression, noise, pollution, perceived danger and overall unpleasantness."* and proposed the wider definition of community severance as: *"the sum of the divisive effects a road has on those in the locality"*.

6.2.2 The Health effects of Community Severance

The issue of community severence was first identified by Appleyard and Lintell (1972) in their classic study in San Francisco. They studied three parallel streets in an area with a homogenous population in terms of social class, income, education and mix of ethnic origin. Traffic volumes had increased considerably in two of the streets over the previous 10 years, so by the time of the study, the three streets carried light, moderate or heavy traffic.

Their study revealed the number of friends and acquaintances of residents were inversely proportional to the volume of traffic. Use of the street for social activities, predominantly meeting places and conversation by older residents and play by children, was common in the street with light traffic street and almost absent in the street with heavy traffic, with the street with moderate traffic being in-between. The street with little traffic promoted a rich social climate and a strong sense of community. This was also borne out by the area considered as "home territory" by the residents. Those living on the street with light traffic included at least their whole building and often the whole street. Those on the street with heavy traffic often felt even part of their own flat was not home territory because of the intrusion of traffic noise, making them live just in the back part of their home. Families with young children had mostly moved out of the street with heavy traffic, because of the adverse effects on quality of life, and the elderly and those too poor to move became isolated within their own homes.

Cassel (1976) proposed two mechanisms these social factors might influence disease causation. Firstly, deleterious factors cause stress which increases susceptibility to disease. A number of studies have demonstrated greater health effects in those exposed to greater psychosocial stress, particularly from new circumstances. Disruption of social relationships can have health consequences: widowers have three to five times the age-specific death rate of married men. Widows have increased morbidity and mortality, particularly in the first year after bereavement that is only partly explained by other known risk factors (Berkman and Syme, 1979).

Cassel's second mechanism was of protective factors that reduce the impact of noxious stimuli. Animal studies have demonstrated the moderating effect of social support: noxious stimuli produce greater adverse effects when the animal is isolated or with strangers than with family members. A study of pregnant women showed twice the rate of complications in women with high life change scores and low social support but no effect of life changes in those with high social support (Cassel, 1976).

These findings are backed up by other studies. A nine year cohort study of Alameda County residents found lower age- and sex specific mortality rates in:

- Married people (especially men);
- In those with higher numbers of contacts with close friends or relatives (especially for women);
- In those belonging to a place of worship; and
- In members of a larger number of formal or informal groups.

On multivariate analysis, each of these factors was independently predictive of mortality but the first two were more important. From this, a Social Network Index was created, incorporating the number and importance of social ties. This correlated even better with age- and sex-specific mortality (Berkman and Syme, 1979).

This is taken to indicate that the extent of social contacts and the existence of a larger social support network have a beneficial effect on health. It is possible that ill-health reduces social contact but the relationships held when analysed within categories of baseline health status. Mortality was also higher for those with fewer social connections than those with more within each of five socio-economic groups and also regardless of smoking history, alcohol intake or physical activity, for both men and women. The differences were less but persisted when all health practices were included, combined into a single index (Berkman and Syme, 1979). A 28-year follow-up of these individuals found that frequent religious attendance still predicted all-cause mortality, after adjustment for age, gender, ethnicity, education and religious group. For women and the whole cohort, the relative risk of dying remained significantly lower when baseline health conditions and social connections were included (relative hazard 0.57 (0.44-0.74) for women, 0.69 (0.57-0.83) overall). Allowing for health practices (smoking, exercise and alcohol consumption) and body mass index reduced the effect of frequent religious attendance but it remained significantly reduced (relative hazard 0.66 (0.51-0.86) for women, 0.77 (0.64-0.93) overall (Strawbridge *et al*, 1997).

A nine-year follow-up of a cohort of Swedish middle-aged men showed a continuous inverse relationship between mortality and social activities or activities outside the home. Such activities were significantly inversely related with self-rated health at baseline, with those who rated their health worse having fewer activities. Multivariate analysis including age, coronary heart disease (CHD) risk factors and perceived health found that outside-home activities, social activities and number of people per household were still significantly related to mortality. Mortality was three to four times higher in those with few compared with many social activities and two-and-a-half times higher for those with few activities outside the home (Welin *et al*, 1985).

After 12 years follow-up of this cohort, multivariate analysis showed that a low level of social activities was related to cardiovascular but not cancer mortality (Welin *et al*, 1992). A review published in 1996 found that social support had a greater effect on CHD than did stress, with both influencing mortality more than incidence. Emotional aspects of support were more

important than the size of the network (Greenwood *et al*, 1996). In a 1999 review of CHD, five out of eight prospective cohort studies of healthy people had found a possible aetiological role for social support. In patients with CHD, nine out of ten studies found a prognostic role for social support (Hemingway and Marmot, 1999).

A cohort study of Norwegian women found that loneliness and dissatisfaction with life were significantly associated with an increased risk of hip fracture, after adjusting for age, body mass index, medication, smoking, physical inactivity and physical illness (Forsen *et al*, 1999).

The longer Alameda County study shows that much of the health benefits of religious attendance are not merely through concentration of healthy behaviours or favourable health status of those attending regularly. The authors found a greater likelihood of an increase in healthy behaviours among this group and among those with more social connections. They suggested that psychological feelings of coherence and stronger resistance to mental and physical stressors may be the mechanism (Strawbridge et al. 1997). A number of studies support the notion that social support acts as a buffer to moderately stressful life events and symptoms of distress (Dahlem *et al*. 1991; Greenwood *et al*. 1996). These add suggestive evidence to the importance of community severance and its consequent reduction in social contacts as contributing to poorer health.

There is a distinction between *"social networks"*, comprising the number and frequency of contacts and their inter-relatedness, or density, and *"social support"*, which is functional, for example emotional, information-providing, or practical and which can be negative (Stansfeld, 1999). Although the studies examining social networks have mostly found significant inverse relationships with morbidity or mortality, studies considering the functional aspect of support, particularly from close family have found deleterious as well as beneficial effects on health (Stansfeld, 1999). For example, one study, looking at depressive symptoms and healthy cardiovascular behaviours, found that family functioning was more important than social support (Franks *et al*. 1992).

In the context of the effects of transport, social networks may be more relevant. However, studies have not been done linking health with community severance or lack of social contacts or social support that are due to transport problems. Community severance thus remains a plausible but unproven cause of inequalities in health.

6.3 Inequality and Accidents, Air Pollution, Noise and Physical Activity

This section discusses the possible inequality issues with respect to the previous chapters in the report.

6.3.1 Accidents

The obvious inequality issues regarding accidents are with respect to different types of road users. Pedestrians and cyclists have higher levels of fatal and serious casualties from collisions with other road users. There are also links between modal choice and both social class and vulnerable groups, particularly children.

Although the number of walkers involved in collisions is smaller than the number of car occupants, the risk of serious or fatal outcome is high – shown by the statistic in Chapter 2 for London.

Road traffic injuries account for 49% of all accidental deaths in children (Roberts et al. 1998). Data for London shows a similar pattern. Children under 15 comprised 30% of pedestrian casualties in London in 1997. Almost two-thirds of the deaths plus serious injuries in children occur in pedestrians (MacGibbon, 1999). There is also a very sharp social gradient for both incidence of and mortality from injury from road traffic injuries in children.

This can also be seen in the death rates over time. Although these have fallen over the past decade, the decline shows big differences by social class. The decline in social classes I and II (32% and 37% respectively) was much larger than in social classes IV (21%) and V (2%). As a result the social class gradient is now fourfold from social class I to V for all road traffic deaths (McCarthy, 1999) and fivefold for pedestrian deaths (Roberts et al. 1998; MacGibbon, 1999).

This reflects a number of factors that are unequally distributed. Children in low-income families will spend more time walking or cycling as they have less access to other modes of transport. Risks are also higher per kilometre walked. There are a number of reasons for this. Firstly because of the exponential rise in risk to pedestrians with increasing traffic speed and because speeding is more common in less affluent areas (McCarthy, 1999: MacGibbon, 1999). Secondly because children from low income families cross more roads (Davis, 1999), are more likely to be unaccompanied and may have less understanding of road safety (MacGibbon, 1999).

One reason for the falls that have occurred in childhood injuries has been less exposure. Parental fear of accidents is one of the reasons children are driven to school and their independent mobility is restricted (Hillman et al. 1990). Roads with a very high risk of injury to pedestrians are deemed “safe” because there have been no such injuries, without acknowledging the paucity of pedestrians using such roads (Adams, 1995).

In areas with lots of traffic, children may not be allowed to play on the streets, reducing their opportunities for social interaction, (or may play there and have a high risk of injury). It is difficult to separate out what is the dominant source of concern from parents - the fear of injury or the fear of attack by strangers. However, the statistics show it is the former that actually has greater impact - fifty times more children have been killed by traffic injuries than by strangers in the past 15 years (Sustrans, 1996).

The social class gradient for road traffic deaths is less marked in adults but persists. If all men had the same death rate as those in social classes I and II, 600 deaths per year and 18,000 years of life lost before the age of 65 would be saved (using 1991-3 data) (MacGibbon, 1999).

The elderly are also at particular risk as pedestrians. Their increased death rate (McCarthy, 1999) is due both to reduced ability to avoid collisions, due to physical impairments, and to a higher case-fatality rate for those injured (Hazucha *et al*, 1994). Many elderly people also restrict their journeys because of fear of traffic .

6.3.2 Air pollution and Noise

A number of studies in the UK, Europe and globally have shown that less affluent districts tend to be concentrated in areas with a higher density of roads and traffic. This in turn leads to increased air and noise pollution, as well as the higher accident rates (discussed above).

For air pollution, these are often further exacerbated by other factors such as poor diet and health care access (with vulnerable groups most at risk).

The question is whether this is true for London. Certainly the effect will exist at a localised level. For example, within any area of similar housing stock, property prices will be lowest for those houses direct alongside busy roads, because of the dis-amenity effects of road traffic noise, the physical presence of vehicles, etc. However, it is not clear whether the correlation exists at a London wide level ,i.e. what needs to be examined is whether the main transport corridors and ring roads in London show the association more broadly.

The results of current studies by the project team indicate that the relationships does exist for London and air pollution, but that the association may be less pronounced than for other cities in the UK. This might be expected due to the very high background levels of pollution in much of London. The figure below shows the correlation between air pollution and a deprivation index (taken as a surrogate for poverty) for London areas. This index includes metrics for:

- Unemployment.
- Over-crowding.
- Lack of amenities.
- Low earning.
- No car ownership.
- Numbers 17yr not in education

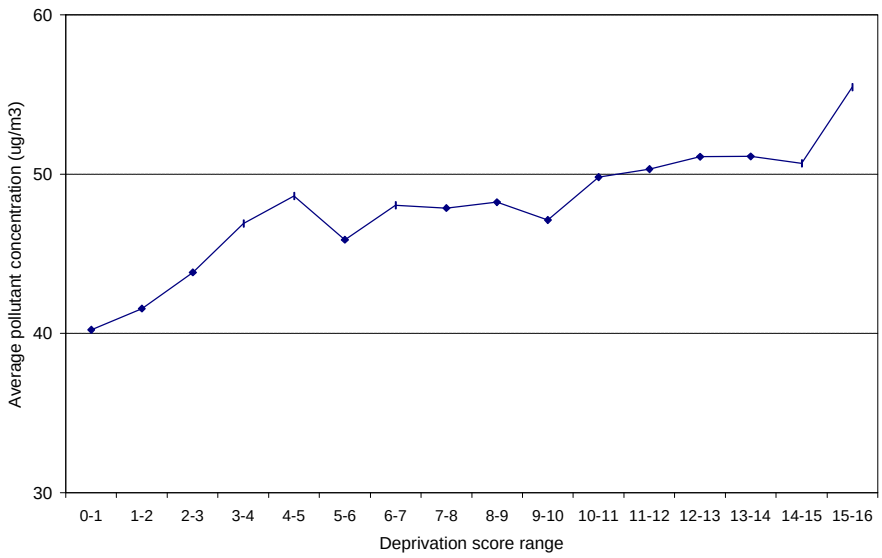

Figure 6.2 Average pollution (NO_2) vs Average Deprivation Score in London.

Source: AEA Technology Environment (2000)

Interestingly the same data shows that in London, there is a clear relationship between air pollution exposure and car ownership – such that the people who own the least cars get exposed to the most air pollution. Correspondingly, the health effects of air pollution will be higher in this group, even though they make less contribution, on average, to air pollution concentrations.

As discussed in Chapter 3, the elderly and those with pre-existing respiratory and cardiovascular diseases are particularly vulnerable to the effects of air pollutants. These diseases also show a marked socio-economic gradient (Acheson, 1998). This would be expected to exacerbate the inequalities in the impact of air pollution.

6.3.3 Physically Active Transport

Physical activity reduces with increasing age (chapter 5). Access to exercise and leisure facilities has a marked social class gradient but walking is a much more common form of transport for those with lower incomes. Many studies (e.g. Cauley *et al*, 1991) show that participation in sport was more frequent for higher socio-economic status individuals.

In London in 1995/97, residents of inner London walked more often (349 journey/year) and further (250 miles per person per year) than anywhere else in the country but this was still a 21% decline from 10 years previously (DETR, 1998). However, they also made fewer bicycle journeys (only 9 per person) and travelled less distance (which fell by 15% to 38 miles per person) than anywhere else.

Not surprisingly, increasing volumes of traffic restrict people's willingness to walk or cycle. Lower income households, with less access to a car, are thus more affected by this (MacGibbon, 1999).

People with greater educational or professional qualifications take more regular moderate or vigorous physical activity, while those from black and minority ethnic groups are less physically active than average. Twenty-eight per cent of men and 23% of women living in rented council accommodation have a sedentary lifestyle, compared with 15% of male and 14% of female owner-occupiers. People who live in areas they perceive to be unsafe are less likely to be physically active.

A sedentary lifestyle is also more common people who are older, economically inactive, have no qualifications, or who belong to a manual social class. However, the same surveys show that this last group are twice as likely to be active enough to benefit health as those from non-manual social classes (Coggins *et al*, 1999). Those with physical or mental disabilities, who are ill or frail are most often excluded from using physically active transport.

Walking or cycling has greater health benefits for those who were previously inactive than as additional activity in those already taking some form of exercise (Andersen, 1995: Shephard, 1997). It is also particularly beneficial for the obese and those at higher risk of CHD and therefore could contribute to reducing inequalities in health, since incidence, prevalence, case-fatality rates and risk factors for CHD show a strong social class gradient (Marmot, 1999).

On average, the ages at which children are allowed to walk, cross roads, cycle or use buses alone have increased, while child journeys by car have increased (Hillman et al. 1990). In 1971, 14% of junior school children were escorted to school while in 1990 this was 64% (Davis, 1992). Children's development may be impaired by reduced opportunities to explore new environments and learn new skills (Davis, 1992).

Of increasing concern is the influence of children being driven to school and having less opportunity to play outside. Both encourage sedentary lifestyles with their accompanying

risks (from lack of physical exercise), and limit independence, with its impact on self-esteem and mental well-being.

6.4 Other Inequality Effects from Transport

6.4.1 Social Exclusion (Inequalities in Access to Transport)

"Social exclusion" is a term used to emphasise not just the lack of economic resources implied by *"poverty"* and the living conditions implied by *"relative deprivation"* but also the process of marginalisation from aspects of social and communal life (Shaw *et al*, 1999). Social exclusion is "*a short-hand term for what can happen when people or areas suffer from a combination of linked problems such as unemployment, poor skills, poor housing, high crime environments, bad health, poverty and family breakdown."* (Coggins *et al*, 1999). It is different from *"inequalities"*, which influence health right across the social spectrum, for example among civil servants of different grades (Marmot & Wilkinson 1999). Both inequalities and social exclusion may have health implications.

For some aspects of transport's effects on health, there is a gradient across all socio-economic groups. For others, the lack of benefits is concentrated in a small minority. Using White's typology of social exclusion, all four elements of social exclusion occur. The main barrier is seen as economic exclusion, exclusion from normal social consumption. The young, those with certain chronic diseases and the visually impaired may not drive a car (legal exclusion). For some, these barriers lead to de-legitimisation, exclusion from social production and others have problems of social exclusion, a failure to provide social goods to a group with particular needs. Such groups include the unemployed, ethnic minorities, particularly refugees and immigrants, the homeless, pensioners, single parents and the disabled (Shaw *et al*, 1999). When considering transport, children should also be added to this list.

There are inequality issues with respect to transport and access, especially for lower income groups. Car ownership is closely linked with reduced morbidity and mortality rates. A continuous gradient has been shown for the number of cars per household and health outcomes (Hazucha *et al*, 1994). It is not known to what extent this is a confounder for income and other factors or whether the benefits of increased access to people, goods and facilities contributes (MacGibbon, 1999). In 1993-95, car ownership in Great Britain demonstrated a sharp gradient by household income (MacGibbon, 1999), shown in the Figure below.

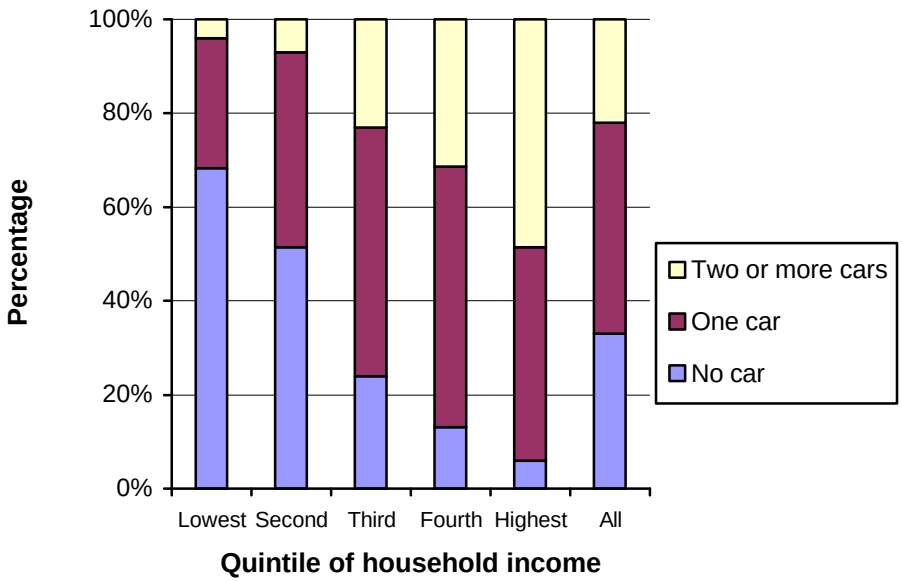

Figure 6.1 % Ownership of cars (1993/95) by quintile of household income in GB.

However, London is atypical with respect to car ownership. The equivalent statistics for inner London show that 51% of households owned no car, 39% one car and only 10% two or more. For outer London these proportions were 33%, 45% and 22% (DoT, 1996).

At the UK level, those owning cars make more trips, as do those household with higher incomes. Statistics for the UK are shown below.

	Number of journeys per week	
Ownership per household	by foot	by car or van
Bicycle but no car	9.6	2.4
1 car	5.8	12.9

Household	by foot	By bus	by car or van
Low incomes	7.3	2.1	5.2
High incomes	4.3	1.0	16.9

Source: McCarthy, 1999, based on UK statistics for 91/93.

Again, the situation is, however, different in London, where the lower travel levels occur (for both distance and numbers of journeys made) which are not explained only by lower car ownership (DETR, 1998). The main users of cars in London households drove 10% fewer miles than those in other conurbations and 29% less than those in the rest of southern England (DoT, 1996). It is not known to what extent the need for travel leads to car purchase or the absence of access to a car prevents beneficial journeys and therefore to what extent low car ownership in London reflects inequalities in access opportunities.

A lack of access to car use increases social isolation for those with difficulties using public transport or walking or cycling, impacting on the elderly, the disabled, and those responsible for young children. At the same time, increasing traffic volume inhibits the use of streets for social purposes (Appleyard and Lintell, 1972).

There is also an issue with respect to access to public transport. Some groups may be excluded from use of public transport by financial barriers. The costs, particularly the marginal costs, of using public transport are greater and have increased faster than the costs of using a car. In the ten years to 1997/98, London bus fares increased by 34% in real terms and London Underground fares by 36% (DETR, 1998), while motoring costs fell by 5% from 1980 to 1995 (MacGibbon, 1999). The wealthier, who are more likely to have access to a car, pay lower marginal costs for their travel than the less well-off, who have to pay more transport.

Over recent decades, development aimed at a car-dependant society have led to social exclusion, in that economic and recreational benefits may only be accessible by private transport. Therefore those groups that depend on public, rather than private, modes of transport will be excluded (economic or social exclusion). For example, access to good quality, cheaper, nutritious food is often more readily available, or even only available, to those with private transport, for example to out-of-town supermarkets. Access is difficult for those without cars, so poorer families may have to spend more on basic food, which impacts on diet. Because of the financial and physical barriers to public transport use and the lower availability of cars, those who are frail or disabled or accompanied by young children are more reliant on shopping locally, spending more on a less nutritious and varied diet (MacGibbon, 1999). In some areas, referred to as food deserts, wholesome food, particularly fruit and vegetables, is not available at all. This is a particularly important type of social exclusion, since an unhealthy diet is a major factor in cardiovascular disease (Wilkinson & Marmot 1998) and contributes a third of cancer deaths (Doll and Peto, 1981).

Those who are more dependent on public transport are also less likely to be able physically to use it. The elderly, the disabled, those with young children (predominantly young women) have physical barriers to use of public transport, leading to social exclusion. Steps, the need for sticks or other walking aids, and use of push-chairs all increase difficulties in using public transport and also in carrying shopping if a car is unavailable. Some of these issues may be particularly relevant for the NHS, particularly with the siting of hospitals and accessibility to patients and visitors.

6.5 Psychological Effects from Transport

There are a number of psychological effects of transport which are additional to the effects described above. These include stress and anxiety from congestion and from other drivers (including road rage), possible anxiety effects on public transport (e.g. from breakdowns especially underground), as well as effects such as the fear from the physical presence of heavy goods vehicles on roads.

6.6 Quantification

6.6.1 Community Severance

Quantification of Effects

A number of studies have attempted to quantify community severance. Indicators of community severance that have been proposed include traffic volume, noise levels and pedestrian delay in crossing roads (Davies, 1992).

Current DETR guidelines (in the new approach to appraisal) includes guidance on accessibility[19]. Three subsidiary objectives have been identified as supporting the accessibility objective:

- To improve conditions for pedestrians and others (including cyclists and equestrians);
- To improve access to public transport; and
- To reduce community severance by roads.

The community severance objective is concerned with the role of roads as a 'barrier' between parts of a community, and the resulting distortion of journey patterns, especially for the aged, disabled and children. This criterion provides a summary assessment of the impact of the trunk road on community severance, as it affects non-motorised modes of transport, particularly pedestrians (as cyclists are less susceptible to severance because they can travel more quickly than people on foot). It takes account both of the extent of severance and the numbers of people affected and is therefore quantitative.

The guidance (based on DMRB 11) provides guidance on the assessment of severance, leading to a three point assessment scale, the points being slight, moderate and severe. The approach is based on an estimate of the total numbers of pedestrians experiencing a change in severance, allowing for the severity of the change experienced. For quantification, the following information needs to be provided:

- Numbers of pedestrians experiencing new severance, subdivided by the severity of the effect,
- Numbers of pedestrians experiencing relief from existing severance, subdivided by the scale of relief.

Where severance varies along a route, it may be appropriate to assess severance at a number of locations. Using this information, the Assessment score may be obtained using the following guidelines (in each case, the assessment is beneficial if relief is greater than new severance, adverse otherwise):

- The overall assessment is likely to be Neutral (if new severance is broadly balanced by relief of severance) or Slight where:
  - new severance and/or relief from existing severance is slight
  - or the total numbers of pedestrians affected across all levels of severance is low (less than 200 per day, say)
- The overall assessment is likely to be Large where:
  - new severance and/or relief from existing severance is severe/ substantial, and affects a moderate or high number of pedestrians
  - or the total numbers of pedestrians affected across all levels of severance is high (greater than 1000, say)
- The overall assessment is likely to be Moderate in all other cases.

For all three sub-criteria, this assessment is based on changes in accessibility in the scheme opening year.

[19] Accessibility is concerned with the 'ease of reaching' opportunities (jobs, shops, leisure activities) or the 'ease of being reached' by contacts (such as clients, customers, workers). As such, it should include accessibility by all modes of transport. The new approach to appraisal broadly distinguishes between motorised travel, covered by the economy objective, and non-motorised travel, covered by accessibility. Defined in this way, the accessibility objective provides a focus for impacts on non-motorised journeys (or parts of journeys), including the impact on those without access to a car.

Quantifying the Health Effects of Community Severance

The problem for this study is translating community severance into a health effect.

Berkman's Social Network Index predicted an increase in all-cause age-adjusted mortality of middle-aged men (relative risk 2.3 from those with most connections to those with least) and women (relative risk 2.8) with relative risks 1.8 to 4.6 for different age and sex groups (Berkman and Syme, 1979). However, this index can be applied only by individual questionnaire and is therefore of use only in a research setting. Interventions on a housing estate that included traffic management improved mental health but these effects are also not yet quantifiable (Stansfeld, 1999). At present we conclude that quantification of the health effects of community severance is not possible. However, this does not mean that inequalities should be omitted from frameworks that aim to quantify the health effects of transport. Where quantification is not possible, the potential effects should be described qualitatively, with answers to four key questions:

1. Are the proposals likely to have positive or negative effects on health?
2. Are these effects likely to increase or decrease inequalities?
3. How large are the effects likely to be?
4. How likely are these effects to occur in response to the proposals?

6.6.2 Other Effects

Reductions in injury and deaths from road traffic accidents can be assessed with reasonable accuracy for given changes in traffic behaviour. The impact on inequalities relating to type of road user should also be quantifiable but those relating to age or social class will be much harder to quantify.

Note the new approach to appraisal (DETR) includes improved conditions for pedestrians and cyclists as an objective. This would touch upon a number of the issues raised above for these groups, though the guidance is constrained to journey time analysis with some assessment of amenity. The qualitative assessment is based on numbers of pedestrians and others affected, and on changes in the duration and quality of their journeys.

The new approach to appraisal (DETR) also includes access to public transport as an objective. The approach quantifies the impact of proposals on numbers of public transport users (bus, rail and other modes) in terms of access times (by non-motorised modes) or service quality (journey time, frequency, reliability and so on). The changes in access times and quality of service are used to determine whether the summary assessment is neutral, positive or negative.

However, given the lack of information on the possible health effects of access, it is not possible to use this information within the health assessment context. Nevertheless, access is an important issue and should be included, even in quantitative terms with respect to access alone (rather than potential health benefits).

6.7 Uncertainty

Of all the categories of effects considered in this study, the uncertainties are greatest for inequality. This uncertainty concerns the underlying studies (i.e. how they pick out effects from complex socio-economic studies, with high numbers of confounders), problems relating

levels of transport to health endpoints (and even concerns over the actual mechanism linking effects to endpoints).

Some commentators dispute whether impacts are as great as some studies initially indicate, because with many social-economic effects (particularly over long time scales), people and communities adapt.

6.8 Current Effects from Transport in London

As the above discussion shows, it is not currently possible to estimate how transport impacts through community severance and inequality in London.

The assessment of these effects requires more detailed studies and can only really be achieved through site specific studies. This means it should be possible to look at the some potential effects of a specific scheme, but it is not possible to provide a generic framework as for other chapters. However, even for such examples, it is difficult (or impossible) to proceed to quantification through to health analysis because of difficulties in linking changes in transport activity to health endpoints.

6.9 Future Research Recommendations

The issues discussed here are less certain than other parts of the study. This area is therefore one for which more primary research might be needed. We highlight the following areas:

- An agreed measuring tool to assess community severance;
- Sufficient use of this tool to evaluate background levels of community severance and to be able to model the effects of transport proposals on community severance;
- Research to quantify the effects of community severance on a range of health outcomes, including both morbidity and well-being, especially with respect to transport severance.

There are also areas that warrant further investigation for inequality effects related to other main effects (accidents, air pollution, noise and physical activity). Some of these, such as the associations between poverty and levels of air pollution and accident risk to vulnerable groups have been shown to be relevant in London, and further studies to look at these in more detail, and how they can be reduced, would be extremely interesting. We believe quantification of these effects may be possible, but this was beyond the time available for this study.

Finally, the area of accessibility, especially for public transport is one that warrants further attention, both for the underlying health associations, and as part of the wider benefits and impacts of transport more generally.

6.10 Conclusions

As stated earlier in this report, this study aims only to look at the direct effects of transport on health. It does not set out to assess the significant benefits transport has on health (indirectly) through access to goods and services, and through economic and social development. These benefits are however extremely important and must be included when looking at transport more generally.

There are, however, a number of other effects that arise directly from transport, which have not been covered in previous sections.

The first of these is community severance, which arises when roads bearing high levels of traffic cut through housing areas. The physical presence of the traffic, particularly heavy goods vehicles, as well as the risk of accidents presents a barrier to the community, limiting or disrupting interpersonal networks and reducing social contact.

There is evidence regarding likely health effects of community severance, though the effect is indirect. Studies indicate social contact may be inversely proportional to the volume of traffic. These social factors in turn may influence disease causation. Firstly, deleterious factors cause stress which increases susceptibility to disease. Secondly, there may be reduction in social support, which is believed to have a moderating effect in dealing with noxious stimuli.

There is evidence which indicates that social contacts does result in lower all cause mortality. However, translating these studies into quantitative frameworks is extremely difficult because of the lack of a relevant link between transport activity and the degree of community severance that occurs. Some qualitative estimates are possible based on traffic volume, though it is stressed there is a high degree of site specificity with effects.

The possible inequality effects of other impacts (accidents, air pollution, noise and physical activity) have also been assessed. For all of these categories, disproportional effects have been identified on vulnerable groups. Of these the relationships between income/poverty and air pollution, and the fear/risk of vulnerable groups (cyclists, children) of traffic accidents are highlighted as most important. For both of these quantification of effects may be possible.

Finally, a number of other categories of effects have been reviewed. These include transport access and social or economic exclusion and the potential stress and psychological effects caused by transport. For both of these we conclude that effects on health are likely to be low relative to other impact categories, and that at present, quantification is not possible.

A number of research gaps have been identified in the study. These include:

- An agreed measuring tool to assess community severance and use of this tool to evaluate background levels of community severance and to be able to model the effects of transport proposals on community severance;
- Research to quantify the effects of community severance on a range of health outcomes, including both morbidity and well-being, especially with respect to transport severance.
- Further research into the health effects of access and comparison with wider benefits offered by transport.
- Further studies into the mental health effects from transport, with respect to annoyance, frustration and anxiety from delays and congestion, and from fear of accidents.
- Quantification of the size of inequality effects on other impact categories, especially associations between poverty and levels of air pollution and accident risk to vulnerable groups.

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7 Other Impacts and Issues

7.1 Other Impacts

The previous chapters discuss the impacts of transport on health. However, in assessing the wider impacts and benefits of transport, other environmental and socio-economic aspects are important and should be considered in balanced decision making.

For environmental effects, these include:

- Effects on the man-made environment (for example the soiling of buildings from particles, building erosion from acidic emissions),
- Effects on amenity (for example from visibility, from noise pollution, etc),
- Effects on natural and semi-natural ecosystems (including deposition of pollutants, water and waste discharge emission to rivers, etc.).
- Effects of climate change[20].

There are also other economic and socio-economic effects that need to be considered. Any approach which aims to help make overall decision for transport policy or schemes needs to take these effects into account. Guidance on a number of these issues is given in the DETR guidance to road appraisal and multi-modal appraisal (recently revised under the new approach for appraisal (DETR, 2000)). London Transport also uses similar assessment criteria, based around multi-criteria analysis. The DETR appraisal system attempts to capture different effects under the following generic headings:

- **Environment** (including noise, local air quality, landscape, biodiversity, heritage, and water)
- **Safety**,
- **Economy** (including , journey times and vehicle operating costs, journey time reliability, scheme costs, regeneration)
- **Accessibility** (including access to public transport, community severance, and pedestrians and others),
- **Integration**.

With the exception of accidents, these criteria do not attempt to specifically quantify health impacts.

7.2 Use of the Framework

The discussion in previous chapters show that it is currently not possible to quantify with confidence all the different health impacts from transport. Nonetheless, the information here could provide important additional information on how large potential effects might be.

This information could be useful for input to other frameworks, for example:

- Strategic environmental assessment (the assessment of environmental impacts associated with a policy, programme or plan),
- Green transport plans,

[20] Climate change also has a very large potential effect on health.

- Urban and transport planning.

The information also has some relevance for current or proposed policy objectives, these include:

- The National Air Quality Strategy (NAQS). This requires local authorities to assess their air quality levels and above certain thresholds (set to protect health) declare Air Quality Management Areas and implement Action Plans. The proposed framework would have a valuable use in helping local authorities look at the likely health benefits of their plans.
- The EC Noise Directive, which is likely to be implemented in the next couple of years and will require London to prepare a noise map. Transport will be the dominant noise source for the map and the transport and health study will again provide important information on assessing the impacts of the noise levels to aid interpretation of the noise maps.
- London specific strategies, for example, the air quality, noise, transport, health strategies to be implemented by the new mayor for London and the roles of transport for London, the GLA, etc.
- The National Cycling Strategy and the London Cycling Campaign, the DETR programmes on 'Developing a Strategy for Walking' and 'Green Transport Plans', the London Walking Forum, and the initiatives from the Health Education Authority. The framework would provide valuable information for these initiatives, showing the importance of these initiatives.

8 Conclusions

This study was commissioned by the NHS executive to investigate frameworks for Health Impact Assessment (HIA) for transport. The objectives of the study were to evaluate the **direct** relationships between transport and health in London and consider whether it is possible to use these for quantification. In the context of this study, health is not restricted to biomedical effects, but also includes a broad view of health as a state of physical, mental and social well-being.

The direct health effects of transport included in the study are:

- Traffic accidents;
- Air pollution related health effects from transport emissions;
- Noise related health effects from transport activity;
- Health benefits from physical activity from cycling and walking;
- Community severance, mental health and inequality effects.

The study aimed to cover only direct transport-specific effects. It therefore does not assess the very large indirect benefits of transport, including the quality of life and health benefits that transport provides through access to goods and services and from economic and social development. We stress however that consideration of these indirect effects is essential when applying HIA to transport, or indeed in transport policy more generally.

The study has assessed the literature to examine positive and negative relationships between transport and health for each of the five categories listed above. Within this study, the aim has not been to assess the underlying studies, but instead to undertake rapid reviews, and from this (where possible) to propose relationships for quantification of effects. For each category, possible ways for incorporating and applying these relationships have been assessed. Finally, in undertaking these studies we have tried to take a London specific perspective, in recognition of the important differences in transport in London relative to other areas of the UK.

The output from the study is intended for use in Health Impact Assessment (HIA). It provides evidence for the direct health effects of transport, which is important during *screening*. The information in this study shows how important direct health effects from transport are (in total and by category), and so can help in judgements when using HIA as to whether potential health effects from transport are negligible or need more investigation.

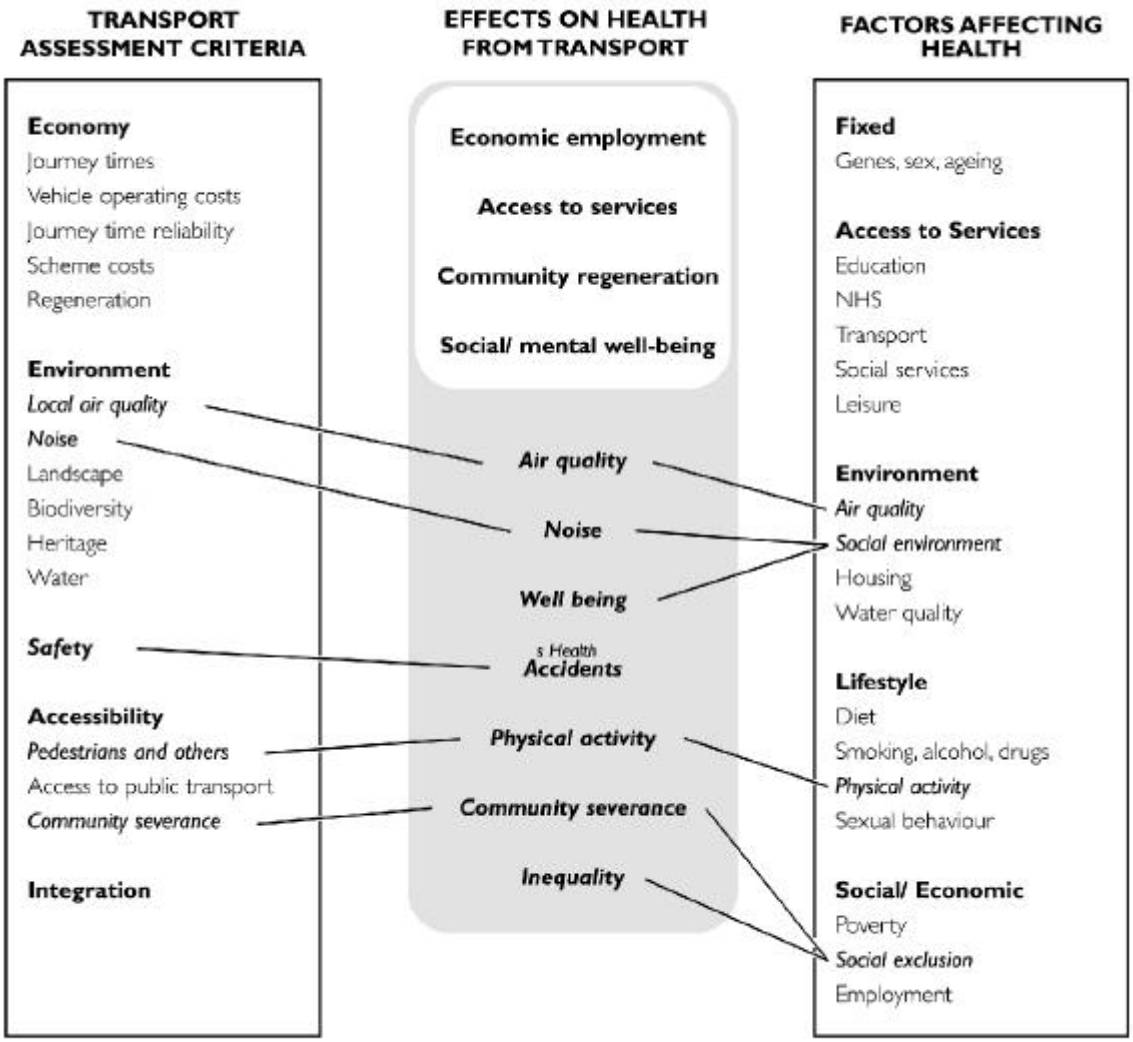
It also provides important information for *appraisal*, which assesses a proposal's effect on the health of the population. The study provides information that can be used in the identification and characterisation of health effects and potential changes in health risk. The information here should allow the evaluation of the nature of effects, their size and severity, and the time and likelihood of occurrence. We have also set out to assess when effects occur dis-proportionately in vulnerable, disadvantaged or marginalised groups, consistent with HIA.

However, in undertaking this study, it is important to consider transport in a wider context than for Health Impact Assessment alone. There are many inter-connections between transport and health, as shown in the figure below. Transport policy, programmes and

projects have traditionally been assessed using specific appraisal techniques. These techniques aim to cover the direct and indirect effects of transport across all criteria, and as such assess health alongside other impact categories such as economic costs, congestion, etc.

We stress that Health Impact Assessment should not be used as an alternative to existing appraisal for setting transport policy, as it only considers a sub-sector of the wider criteria involved. However, it can be used alongside traditional transport appraisal to provide a better understanding of the health consequences of transport. By doing so it may be able to help improve the health aspects of transport policies, programmes or plans, minimising negative health effects and maximising positive health effects, as long as these are balanced against effects on other categories (environment, economy, etc.).

The context of the current study (the shaded area below) with respect to HIA and transport appraisal is shown in the figure below.



Finally, it is stressed that this research project is the first of a two part study. This stage provides details of the evidence and evaluation on whether quantification is possible. To fit with the scoping goal of HIA, it has assessed the relative size of the health effects for different categories from transport in London, to provide some guidance on how important these effects are relative to each other and in the context of other factors affecting health. One of the key aims of this phase of the work has been to make explicit where impacts can be quantified and where this is problematic: by doing so we hope to identify gaps in the knowledge and highlight research priorities. This is particularly important given the uncertainty for some of the impacts.

Within this first phase it is impossible to recommend one generic approach and framework that is universally applicable for researchers and decision-makers in health assessment and in transport appraisal, and one that can be applied to detailed schemes and at the same time to London wide policies. What this study hopes to achieve is to provide evidence and relationships which can be applied to these specific applications, once combined with the relevant level of detail and incorporated in the frameworks representative of the specific type of assessment.

The second phase of the work, to follow on from this study, is to use this information for looking at the marginal effects on health from new transport policies, schemes, or measures.

Finally, as part of the study, consultation was held with a number of organisation (e.g. DoH, GLA, TfL, DETR) on evidence and potential frameworks, though we stress that wider consultation is needed. This needs to involve both potential users of the information from this study, as well as a wider group of stakeholders to assess how to relate the relative effects from different categories of impact and against other criteria (both for health and for transport).

The evaluation by effect category is presented below.

8.1 Accidents

The most obvious and one of the largest effects of transport on health are traffic accidents. Quantification of accidents from transport is already included in current transport policy and scheme assessments and is usually based on historical relationships between levels/types of transport activity and accident rates. These approaches are well established and have agreed methodologies (unlike other categories of impact).

The impacts of transport on health from accidents are obvious; the cause and effect chain is simple and immediate. The assessment of accidents is therefore easier than with other categories considered in this study. There are already established quantification methodologies for predicting transport accidents and casualties. These methods use historical accident data as a means of predicting future accident rates from new schemes or policies, adjusting for road type and speed, as these influence the numbers and severity of accidents.

The current levels of health impacts from road transport accidents in London are recorded and reported. It is worth noting that there are differences in the trends in transport statistics in London compared to the UK generally: accident rates in London remain relatively constant year on year, whilst rates in the rest of the country decline.

Finally, the distribution of accidents does impact on certain road-users and age groups disproportionately. The relative risk of serious injury or death by distance and by journey for pedestrians, cyclists and motor-cyclists is much higher (indeed of the accident fatalities in London 1998, more than half were pedestrians). There are also links between modal choice (and accident rates) and both social class and vulnerable groups, particularly children.

8.2 Air pollution

Studies of pollution episodes (such as the London smog episodes of the 1950s) have shown that very high levels of ambient air pollution are associated with strong increases in adverse health effects. Recent studies also reveal smaller increases in adverse health effects at the current levels of ambient air pollution typically present in urban areas. These health effects include a range of endpoints, such as premature mortality (deaths brought forward), respiratory and cardio-vascular hospital admissions, and possibly exacerbation of asthma, respiratory symptoms and loss of lung function. The evidence for these effects is strongest for the pollutants PM_{10}, SO_2 and ozone and the relationships are widely accepted as causal. Recent studies also suggest that long-term exposure to these pollutants, especially particles, may also damage health and that these effects may be substantially greater than the acute effects described above.

Transport is a major source of these atmospheric pollutants in urban areas and therefore can be assumed to have adverse health effects. Frameworks for quantifying the health impacts of transport related air pollution do exist. The frameworks require a series of steps and involve additional analysis to that typically found in transport model outputs. Such approaches first quantify emissions from transport vehicles (taking into account that emissions vary with vehicle type, fuel type, technology and speed). They then proceed to assess the effects of these emissions on local air pollution concentrations, usually with the use of dispersion models. The pollution data is then combined with data on population to estimate the population weighted air pollution increase. The final step is to quantify health impacts with the use of exposure-response functions from epidemiological studies, which link ambient air quality to health endpoints. There is however debate on the exact exposure-response functions and health endpoints that should be included in any assessment framework.

For this study we have compared the results from three sets of exposure-response functions: from the Department of Health's COMEAP sub-group, from the EC's ExternE Project and from functions from specific London epidemiological research studies. We have combined the first two of these to provide a framework for quantification for health damages from transport in London. This uses COMEAP recommendations to quantify those effects for which there is the greatest confidence (least uncertainty) and ExternE recommendations to estimate additional health impacts where the evidence is strong but where quantification is more uncertain. Impacts have been classified into uncertainty bands, to reflect the different confidence levels attached to different health endpoints and quantification approaches. The approach is applicable for looking at marginal changes from transport policies or schemes. We have however demonstrated it by estimating the current levels of air pollution related effects from all transport in London, in order to compare the potential importance of air pollution relative to other categories of impact. This analysis leads us to conclude that air pollution related health impacts from transport may be equivalent to, if not greater than transport accidents in London.

8.3 Noise

Noise is a major nuisance and is widely recognised as a dis-benefit affecting human amenity. It may also lead to a number of health impacts through a variety of direct and indirect effects, though there is considerable debate on the reliability of the evidence. Transport is a major source of ambient noise levels and therefore may have important health impacts.

The evidence for noise impacts, as discussed in recent major reviews has been assessed. The conclusive impact of the health effects of noise is mostly limited to cases of hearing loss and tinnitus caused by long periods of (occupational) exposure. These effects are generally not important at the typical levels of noise arising from transport. A number of studies also point to potential physiological and psychological impacts from the noise levels associated with transport (from road, rail and aircraft), including speech interference, annoyance, sleep disturbance, performance, cardiovascular and physiological effects, and mental health effects.

There is general agreement that noise is a source of annoyance, indeed, it is beyond dispute that certain individuals are annoyed by a wide range of external noise. There are, however, problems in interpreting the potential health impacts of both direct and indirect routes from noise stimulus to the annoyance effect. These problems arise because annoyance is related to the duration and the frequency components of sound and because annoyance relies on subjective measures and the sensitivity of individuals (susceptibility).

The remaining evidence of effects of environmental noise on health are strongest for sleep disturbance, ischaemic heart disease and performance by school children. It is stressed that much of the evidence in support of actual health effects other than annoyance and some indicators of sleep disturbance is quite weak. The data on other possible health consequences, such as low birth-weight and psychiatric disorders, are inconclusive.

Existing exposure-response relationships are confounded by a number of variables that serve to scatter data points around the cause-effect curves. The scientific evidence suggests a threshold below which no effects are expected to occur, although this cannot be considered definitive. As well as this uncertainty in relation to exposure-response functions, there are other major problems in developing noise and health quantification frameworks. These relate to subjectivity and individual responses to noise, as well as how well average noise levels (commonly used to predict noise amenity effects) actually relate to a metric which is relevant for health endpoints.

In summary, although it is possible to assess quantitatively the noise levels from transport, it is very difficult to evaluate quantitatively what the health consequences of these levels are. A qualitative approach is possible, though there remains considerable debate on the reliability of evidence relating to health effects.

8.4 Physical Activity

Physical activity has significant health benefits in reducing coronary heart disease and in reducing other health impacts such as diabetes, obesity, hypertension, cancer, osteoporosis and even depression. Transport (through the encouragement of cycling and walking) can therefore provide significant health benefits.

The benefits are realised through 30 minutes of activity of moderate activity on most, preferably all, days of the week. Physical activity is recommended (rather than participation in organised sports or exercising) because most people can incorporate moderate physical activity into their daily routine – and it is in this respect that there is a link between transport and health.

There is quantification evidence relating physical activity to health endpoints. In theory this should allow quantification of possible benefits for transport related travel (cycling and walking). Inverse causal relationships exist between physical activity and all-cause mortality. These relationships may be significant (equivalent to the difference in mortality of non-smokers and smokers). There are also similar relationships for the risk of developing coronary heart disease. Relationships also exist for the development of type 2 diabetes mellitus, cancer mortality (and specifically cancer of the colon) and hip fractures in elderly men and women.

There are also a number of other benefits from physical activity, for which good evidence exists but for which quantified estimates of benefits are not available. These include reduced long-term risk of osteoporosis, greater well-being and self-esteem, as well as benefits in reducing mild depression and mild anxiety. They also include benefits in later life (for the elderly), including improvements in balance, co-ordination, mobility, strength and endurance and in the control of chronic disease, and in reducing weight especially for the obese, as well as general benefits in keeping people mobile.

It is likely these benefits would be realised through cycling to work. They may also be realised by walking to work, though this activity level may not reach the necessary level of intensity of activity for some groups (e.g. young adults). However, the greatest health benefits are gained by increasing activity levels in elderly, sedentary or obese individuals, and walking will have major benefits for such groups. The risks from cycling and walking (musculoskeletal injuries and accidents) are less than the benefits obtained (by a significant factor).

There do however remain some problems in providing reliable quantitative estimates. The most obvious of these is the lack of data on the linearity of functions and the presence or absence of thresholds. Moreover, the effects on modal shift and travel patterns in response to specific projects or policies are not well characterised. Finally, there are also important questions over how to assess background health status and activity levels, though we note that by looking at specific policies, options or schemes, most of these background issues could be addressed and quantitative estimates could be made.

8.5 Community Severance and Other Effects

As stated earlier in this report, this study aims only to look at the direct effects of transport on health. It does not set out to assess the significant benefits transport has on health (indirectly) through access to goods and services, and through economic and social development. These benefits are however extremely important and must be included when looking at transport more generally.

There are, however, a number of other effects that arise directly from transport, which have not been covered in previous sections.

The first of these is community severance, which arises when roads bearing high levels of traffic cut through housing areas. The physical presence of the traffic, particularly heavy goods vehicles, as well as the risk of accidents presents a barrier to the community, limiting or disrupting interpersonal networks and reducing social contact. There is evidence regarding likely health effects of community severance, though the effect is indirect. Studies indicate social contact may be inversely proportional to the volume of traffic. These social factors in turn may influence disease causation. Firstly, deleterious factors cause stress which increases susceptibility to disease. Secondly, there may be reduction in social support, which is believed to have a moderating effect in dealing with noxious stimuli.

There is evidence which indicates that social contacts does result in lower all cause mortality. However, translating these studies into quantitative frameworks is extremely difficult because of the lack of a relevant link between transport activity and the degree of community severance that occurs. Some qualitative estimates are possible based on traffic volume, though it is stressed there is a high degree of site specificity with effects.

The possible inequality effects of other impacts (accidents, air pollution, noise and physical activity) have also been assessed. For all of these categories, disproportional effects have been identified on vulnerable groups. Of these the relationships between income/poverty and air pollution, and the fear/risk of vulnerable groups (cyclists, children) to traffic accidents are highlighted as most important. For both of these quantification of effects may be possible.

Finally, a number of other categories of effects have been reviewed. These include transport access and social or economic exclusion and the potential stress and psychological effects caused by transport. For both of these we conclude that effects on health are likely to be low relative to other impact categories, and that at present, quantification is not possible.

8.6 The Relative Effects of Transport on Health

One of aims of this initial phase was to assess the relative importance of different health effects of transport in London, to illustrate where evidence existed and where quantification could be undertaken. By doing so we have aimed to provide some feel for how important transport is with respect to health (overall and by category).

For accidents, statistics are available on the current health effects from transport in Greater London. The latest figures report that there were 45,547 road accident casualties in London in 1998. Of these, 226 were fatalities, 6,632 were serious and 38,689 were minor (or slight).

Comparing these against the effects of air pollution is extremely interesting. Using the framework outlined here, we have estimated the current levels of air pollution related effects from all transport in London. We estimate 380 fatalities (deaths brought forward) and 350 respiratory hospital admissions per year occur in London from transport related pollution (excluding ozone). Interestingly the number of fatalities is of a similar order of magnitude to the numbers of deaths in London from traffic accidents, though it is stressed there are important differences in the age and health-state of people affected by the two impacts. Many of the deaths associated with pollution are probably in the elderly and the sick and the period of life lost may be small. The attribution of causality is also far more certain for accidents than for air pollution. In addition, there are also estimated to be an additional 815 cardio-vascular/cerebro-vascular hospital admissions and half a million minor respiratory symptoms from transport related air pollution in London each year, though a slightly higher uncertainty

rating is attached to these numbers. Air pollution in London is also thought to lead to changes in life expectancy (chronic mortality). We estimate that transport related air pollution leads to the loss of around 34 thousand years of life per year, though stress there is a higher uncertainty in this value than for the endpoints above. Finally, there are a number of other possible impacts that have been reported in US studies. The use of these estimates leads to additional health impacts from transport emissions in London including a small number of deaths (around 30 per year) from carcinogenic emissions, and a very large number (half a million) of respiratory symptoms. These effects are given the highest uncertainty rating.

Overall, these values indicate transport related air pollution is as important (perhaps more so) than accidents with respect to the health of Londoners. Moreover, the combined health impacts from accidents and transport related air pollution are estimated here to be responsible for at least 1% of annual deaths in London. They are also responsible for thousands of serious health effects / injuries and tens of thousand (possibly hundreds of thousands) of minor health effects / injuries each year. However, these impacts should be evaluated against the significant benefit transport produces (though this is beyond the scope of this study).

The study has also looked at the potential benefits from transport related physical activity in London. Further work is needed to equate risks in equivalent terms to air pollution related health effects and accidents. Nonetheless, initial calculations indicate the benefits of physical activity from the current numbers of people cycling or walking to work in London could be of a similar order to the dis-benefits from accidents or air pollution (i.e. in terms of the life expectancy gained per year for London). We highlight the derivation of more accurate versions of these numbers as one of the immediate research priorities following on from the study.

The relevant numbers of people affected by health impacts from noise and severance are harder to evaluate. Data on noise levels across London are not currently available. Nonetheless, it can be concluded that a very large number of people in London (> several hundred thousand) are exposed to noise levels above the WHO environmental guidelines, a threshold below which few people are seriously annoyed.

For both noise and community severance, potential health impacts occur as a secondary consequence of transport activity. Initial estimates might be possible, especially for noise, when applied for the evaluation of specific schemes or policies (rather than generically for London as for other categories).

8.7 Research Recommendations

There are a number of different categories of further research recommendations that arise from a study such as this. The first centre on the primary research required to improve our understanding on the links between transport and health. Many of these are longer-term research priorities. The second concern the more immediate or short-term priorities. Even though an area can be investigated in detail with a detailed research programme, in many cases some additional input is needed to the debate now, to help inform decision makers in the short term. Areas identified, by impact category are summarised below:

Accidents. The research recommendations from accidents are lower than other parts of the study, as the methods of appraisal are widely accepted, and much research effort has been input into this area. Nonetheless there do remain questions about the relationships of certain

key assumptions, for example the link between marginal changes in traffic volume and accident rate and severity. There are also health specific areas that warrant more research. It would be interesting to look at the effects of accidents on the NHS, both in terms of numbers (with a break down by impact type) and health costs. It would also be interesting to investigate how potentially important mental health effects (trauma) from injuries might be, as well as further studies into the fear of accidents affecting people's decision to walk or cycle more generally.

Air pollution. There are a large numbers of possible research areas for air pollution that warrant further research. Any research recommendations here should be compared to other ongoing programmes though we highlight the issue of uncertainty analysis as a priority, as well as further work to improve the estimates of chronic effects. In addition we recommend the use of the approach to look at specific scheme and policy measures, particularly in the context of current legislation with respect to health based local air quality standards (e.g. as part of the National Air Quality Strategy).

Noise A number of research areas have been identified. Those regarding the primary health impact data include longitudinal field studies and natural experiments (e.g. changes in the siting of an airport) in preference to laboratory and cross-sectional studies. Appropriate study design should also consider impacts on vulnerable groups, confounding effects and effect modifiers. Studies should also take account of relevant socio-economic and political factors across different communities exposed to noise sources. Further research is required for studies that suggest that endocrine status, motivation and annoyance are affected by noise exposure in children. There is a need to establish whether these effects persist over time, or change in size, and a need to distinguish between the immediate and delayed effects of noise.

More work is needed on the appraisal of noise sources and the evaluation of mechanisms to relate to health impacts. There is also a need to improve the measurement of noise sensitivity and annoyance. A metric is required against which health effects can be assessed and measures standardised. As with air pollution, any research recommendations here should be compared to other ongoing programmes. There are however, some other immediate areas that warrant research. These include a more detailed look at data sources in London to investigate noise levels. They also include the use of frameworks to look at specific policies or schemes. This would allow the evaluation of potential exposure response functions and would allow a first order calculation of the potential importance of noise (whilst accepting the confidence in estimates may be low) and so to evaluate how important an issue noise and health might be for transport.

Physical activity. The assessment of physical activity benefits comprises one of the most interesting aspects of the current study. It is highlighted as warranting a focus for follow-on studies in general. We have however highlighted the following specific areas:

- The need for a series of systematic reviews and meta-analyses of primary studies to answer the question "to what extent can physically active transport influence health and disease outcomes?" For cancers, this would entail updating the existing meta-analyses.
- Specific work to try and get sensible activity-effect functions and relate these to frameworks. This includes derivation of baseline factors for future analytical frameworks.
- The derivation of specific numbers for London for the endpoints listed.
- The use of the numbers to look at specific policies, i.e. what benefits might you see with modal shift to cycling and walking. This could be extended to investigating policies to say how benefits can be maximised, whilst reducing the risk of detrimental effects.

- Considering the relative risks of exercise benefits against accident risk and air pollution exposure specifically for London, as well as comparing against other policies (e.g. stopping smoking). This could also be extended to look at the health benefits from displacing private car journeys – for example calculating the direct health benefits and the avoidance of impacts from air pollution, accidents, etc.
- The interaction of promotion of exercise in the context of other dimensions of policy: safety; public transport capacity, e.g. how much public transport capacity is taken up by trips that could equally be walking trips especially in London? Is there a risk that lower fares and improved services would actually encourage less physical activity? What additional benefits are there in targeting the elderly to keep them mobile (for whom benefits may be greater)?

Community severance, other effects and inequality. A number of research gaps have been identified:

- An agreed measuring tool to assess community severance and use of this tool to evaluate background levels of community severance and to be able to model the effects of transport proposals on community severance;
- Research to quantify the effects of community severance on a range of health outcomes, including both morbidity and well-being, especially with respect to transport severance.
- Further research into the health effects of access and comparison with wider benefits offered by transport.
- Further studies into the mental health effects from transport, with respect to annoyance, frustration and anxiety from delays and congestion, and from fear of accidents.
- Quantification of the size of inequality effects on other impact categories, especially associations between poverty and levels of air pollution / accident risk to vulnerable groups.

From our discussions with numerous people during the course of the project, the one dominant aspect raised has been the need to focus the project by looking at the quantification frameworks in relation to actual schemes or policies, i.e. looking at marginal changes in transport. This would provide useful information and was the original plan of the research study of the NHS (to be implemented via a second phase of work).

It is essential that the techniques be developed with the help of a few, well chosen, specific policy proposals. Until that is done the study here will only be a review of the existing knowledge of generic relationships – a valuable thing to have, but a long way from an operational evaluation framework or a useful tool for decision making.

This is particularly important for some effects, such as physical activity, which do not lend themselves to generic quantification. These effects tend to be highly site specific, and so can only be put properly into context by looking at specific examples.

We believe there are three areas of marginal changes that are a priority for investigation:

- Assessment of health effects at the transport scheme level,
- Assessment of health effects at the transport from London wide policies,
- Assessment of health effects from NHS decisions that have an impact on transport. This is important given the NHS is one of the largest employers in London and has a major impact on transport provision and activity levels.

During the course of our discussions, a number of possible areas of investigation have been identified. These include

- Congestion charging;
- Speed reduction policies;
- Home zones;
- Pricing and fare structures, including road pricing;
- New public transport links;
- New access roads for re-generation (stronger links to network, east-Thames river crossing);
- NHS related effects, from say hospital closure or for green transport plans.

Finally, we have not considered the very large health benefits that transport has within this study, i.e. the indirect effects of transport. We highlight this as a major omission, but one that lay outside of the original remit.

8.8 Quantification Frameworks

The evidence in the study shows that transport may have important health effects through all of the categories assessed. These are summarised in the table below.

	+ = Beneficial effect		**– = Detrimental effect.**		
Endpoint	Accidents	Air pollution	Noise	Physical activity	Community severance
Physical injury	---			-	
Respiratory & cardio-vascular disease		---		+++	
Cancer		-		++	
Mental health/well being	-	-	--	+	--
Diabetes, obesity, osteoporosis				++	

Key, -/+ low, --/++ medium, ---/+++ high effect.

At present we conclude that it is not possible to quantify all the health impacts of transport with similar confidence. There is considerable uncertainty associated with many of the impacts we have addressed. This uncertainty is relevant to the effect itself (i.e. is it real?), as well as with respect to the reliability of quantification.

It is worth noting however that impacts from different transport modes do vary. These are summarised below. This has important consequences for choices over modes and policies.

	+ = Beneficial effect		**– = Detrimental effect.**		
	Accidents	Air pollution	Noise	Physical activity	Severance
Air transport	-	--	---		
Road – motorised	---	---	---		---
Road – non-motorised	-			+++	
Rail	-	--	--		-
Underground	-	-	-		

River	-	-	-

Key, -/+ low, --/++ medium, ---/+++ high effect.

A summary of the quantification framework for each impact category is presented below.

For those areas which have been more intensively studied, and for which more summary information exists, we have been able to summarise information from key review studies and provide quantification methods. For the more uncertain aspects, more work has been undertaken looking at the underlying literature.

The table shows that there is still uncertainty over many areas. Moreover, people hold very different views about the causality for different effects and the area of transport and health remains a controversial one.

Category	Effect	Certainty of impact	Certainty and approach for quantification
Accidents	Injury	High	High Direct cause and effect based on historic rates.
Air Pollution	Respiratory and cardio vascular mortality and morbidity	Medium	Medium. Quantification possible through 1 Assessment of effects of traffic on air quality 2 Assessment of health impacts with exposure-response functions Debate on which health endpoints and which functions should be used.
Noise	Indirect through annoyance and sleep disturbance to well-being, mental health and mortality	Low	Low. Quantification **potentially** possible through 1 Assessment of effects of traffic on noise levels 2 Assessment of health impacts with exposure-response functions Many endpoints are secondary and are difficult to quantify. Questions over functions and how they relate to noise specifically. Issues of perception, sensitivity of individuals, thresholds, non-linearity.
Physical activity	Cardio-vascular, diabetes, cancer, (**beneficial**)	Medium – High	Low. Quantification **potentially** possible through 1 Assessment of benefits of physical activity 2 Assessment of background levels/confounders 3 Estimate changes in likely physical activity from transport policy or scheme Questions over linearity and threshold with functions. Some issues relating traffic activity to levels of effects.
Community Severance		Low	Low. Many endpoints are secondary and as such difficult to quantify, especially in relation to transport activity. Overall difficulty in linking traffic activity and levels of impact.

Overall, we conclude that it is possible to evaluate the health effects of accidents and air pollution, though stress the uncertainty associated with the latter is higher and the consensus on effects lower. Frameworks exist for both categories to assess the marginal effects of transport, though the analysis for air pollution is complex. It is also likely that the health benefits of cycling and walking can be quantified, though further work is needed to provide quantification methods that fit conventional frameworks.

It is possible to assess quantitatively the noise levels from transport, though it is very difficult to evaluate quantitatively what the health consequences of these levels are. A qualitative approach could be undertaken, though there remains considerable debate on the reliability of evidence relating to health effects. Finally, the evidence and assessment methods for other direct effects from transport are less well characterised, though it may be possible to qualitatively assess the potential health effects of community severance.

READER NOTES