

**Social & Environmental Benefits of Forestry
Phase 2 :**

**MORTALITY AND MORBIDITY BENEFITS OF AIR
POLLUTION ABSORPTION BY WOODLAND**

Report to

Forestry Commission

Edinburgh

from

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December 2002

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1. Introduction

Air pollution has a long history, perhaps reaching its peak during the industrial revolution. Although such extreme pollution is not observed today in Britain, poor air quality remains a problem for human health.

Physical damage functions relating health (mortality and morbidity) to air pollution levels have been estimated over a number of years in different countries. Although the net effect of pollutants on health is unclear, the Committee of the Medical Effects of Air Pollution (COMEAP), set up by the UK government has found the strongest link between health and pollution to be for particulates (PM₁₀), sulphur dioxide (SO₂) and ozone (O₃) (Department of Health, 1998). A subsequent study by the Department of Health (1999) investigated the link between deaths brought forward and hospital admission caused by air pollution and economic cost, and found this cost to be substantial.

Although the main consideration of policy must be the reduction in pollution at source, there has been an increasing recognition that the biosphere is an important sink for many pollutants, with plant canopies being considered more effective than other land uses. Thus, the biosphere provides benefits additional to those associated directly with their aesthetic and wildlife characteristics. Plants facilitate the uptake, transport and assimilation or decomposition of many gaseous and particulate pollutants. Indeed, the layered canopy structure of trees, which has evolved to maximise photosynthesis and the uptake of carbon dioxide, provides a surface area of between 2 and 12 times greater than the land areas they cover (Broadmeadow and Freer-Smith, 1996). A comprehensive study by Nowak *et al.* (1998), for example, found urban trees in Philadelphia, USA, to have removed over 1,000 tons of air pollutants from the atmosphere in the year of 1994. In terms of health effects, Hewitt (2002) found that doubling the number of tree in the West Midlands would reduce excess deaths due to particles in the air by up to 140 per year.

The report is structured as follows: section 2 outlines the relevant literature on pollution absorption by trees; section 3 reviews the literature on the epidemiological effects of air pollution; and section 4 the economic costs of air pollution in terms of health. Section 5 outlines the data sources, the approach adopted to estimate pollution absorption by trees, the health impact on the population of pollution absorption by trees, and the economic benefits. Finally, section 6 presents the non-market benefit estimates.

2. Pollution absorption

Pollutants occur in the atmosphere in the form of gases, particles or in aqueous solution in rain or mist. These pollutants are transferred to terrestrial and aquatic ecosystems as gases and particles by dry deposition, mist or aerosols by occult deposition or by rain or snow by wet deposition. Nowak *et al.* (2000) explained that trees in cities directly and indirectly affect pollution levels through:

- their impact on meteorology (air temperature, radiation absorption and heat storage, wind speed, relative humidity, turbulence, surface albedo, surface roughness, and consequently the evolution of the mixing layer and height);
- dry deposition of gases to the earth's surface;
- emission of volatile organic compounds; and
- anthropogenic emissions through reduced energy use due to lower air temperature and shading of buildings.

Although not all these effects will be applicable outside cities, it does illustrate the complexity of the situation being modelled.

Trees absorb pollutants through the same process they take up nutrients, i.e. stomata (pores on the surface of the leaf), roots and root hairs. Additional pollution is removed by capture on their leaf/needle and bark surfaces. Taylor and Constable (1994) estimated for SO₂ and O₃ that 70% and 80% of the pollution absorbed is internal to the leaf. For particulates, the main wet absorption route is through tree roots (Broadmeadow and Freer-Smith, 1996), whereas the main dry absorption route is through deposition on leaf and bark surfaces. Occult deposition is also difficult to measure and may only be important in uplands and coastal areas. Given the reliance on absorption coefficients from the literature, this study focuses on dry deposition.

The deposition velocity (v) is the rate at which the pollutants are absorbed/captured normalised for pollution concentration. In terms of dry deposition, an electrical resistance analogy is used, where v equals the inverse of the sum of resistances (Baldocchi *et al.*, 1987; Fowler *et al.*, 1989). Three resistances are usually calculated through the:

- aerodynamic resistance (R_a) of the turbulent layer;
- boundary laminar layer resistance (R_b) of the land surface; and
- canopy resistance (R_c) of the receptor itself.

R_a and R_b are atmospheric resistances with R_a determined from wind profiles over the forest canopy and R_b through the combination of wind speed and surface properties. R_b for forests can take a wide range of values depending on the density of the foliage, leaf form, tree spacing and surface topography. R_c is calculated from the combination of the resistances caused by stomatal, mesophyll, cuticle and soil. Canopy resistance can then depend on air temperature, radiation from the sun, moisture on the surface and other factors.

Given that a health-pollution link has been established for particulates (PM₁₀), sulphur dioxide (SO₂) and ozone (O₃), the remainder of this section describes the extent to which trees alleviate this problem.

i) *Particulates (PM₁₀)*

Particulate pollution is a term that covers a broad spectrum of specific pollutant types that permeate the atmosphere, where sources can be both natural and anthropogenic. Within urban areas, exhaust fumes from road traffic have been the most significant source (Watkins, 1991). PM₁₀ is commonly classified into two further size groupings: coarse and fine. The coarse fraction includes all suspended particles in the PM₁₀ size range above 2.5µm in aerodynamic diameter, whilst the fine fraction contains the remaining. The coarse fraction has been judged to be made up mostly of natural and organic particles, whereas the fine fraction mostly particles of anthropogenic source (DoE, 1995). The PM_{2.5} particles are most likely to have a health damaging effect (Pekkanen, *et al.*, 1997) and remain in the atmosphere for longer distances from their source (Monn *et al.*, 1995; Janssen *et al.*, 1997), however, UK wide data on PM_{2.5} is not currently available.

Broadmeadow and Freer-Smith (1996) described three methods of particular deposition: sedimentation; precipitation and impaction. Sedimentation and precipitation occur due to gravity and collision with rain droplets respectively, and are unaffected by vegetation. Impaction occurs when a laminar air stream is disrupted as it passes the aerodynamically rough plant surfaces, while the particle continues in a straight line and strikes the obstacle, either through direct interception or electrostatic attraction. Retention can be helped by rough, pubescent, moist and/or sticky surfaces, where the literature review by Beckett *et al.* (1998) found increased stickiness of surface particularly facilitates greater coarser particle capture, while, roughness of the surface has the greater influence on the uptake of finer particles. Some particles may be absorbed into the tree but most are retained on the plant surface. Some particles will be re-suspended, but others will be washed off (particularly soluble particulates) or fall with leaves or twig fall. This may lead to pollution within the soil, however, Beckett *et al.* (2000a) argues that this will only be a major problem in countries using a high proportion of lead fuel. Re-suspension of fine particulates is less likely as they are easier embedded¹ within the leaf boundary layer (Beckett *et al.*, 2000b). Whatever the outcome of the particulates, vegetation is likely to be only a temporary resting place.

Recent studies have tested for particulate absorption through sampling leaves and examining the product of washing from broadleaved trees (plane, lime, elm, cherry and ash). Beckett *et al.* (2000b) sampled from five urban polluted sites within the South East of England², which were located in close proximity to road traffic. Using foliage density estimates, the total insoluble particle load per tree was found to vary between 41 and 2936 grams. Although this variation is partly due to the ambient pollution at the sites, significant same site species differences were observed providing evidence that pollution absorption varies between tree species. Due to the limitations of the research, however, it was not clear which species was the most efficient absorber of pollution. Although the proportion of PM_{2.5} particles was not measured, anthropogenic sourced particles were generally smaller than those produced from natural sources. Anthropogenic particles ranged from between 25 and 62% of those sampled.

¹ A particle is embedded in a leaf boundary layer either if it becomes attached to the leaf cuticle or is trapped in the semi-laminar boundary layer.

² See also Freer-Smith *et al.* (1997)

Beckett *et al.* (2000a) used a similar sampling approach for a further two sites, with one being rural and the other urban. Five species were considered: three broadleaved (whitebeam, field maple and hybrid poplar); and two coniferous (corsican pine and leyland cypress). Unlike Beckett *et al.* (2000b), trees of approximately two meters height were planted approximately two meters apart with one tree for each species at each site. At the urban site, the trees were planted along a roadside edge. Sampling was conducted following 10 dry days to enable particulate accumulation. Particle filtration was into three groupings: coarse (approximately less than 10µm but greater than 2.5µm); fine particles (within 2.5µm); and ultra fine (based on the ion analysis) soluble particles. Substantially more coarse particulates were found at the urban site than the rural. Considering fine particles, however, little site difference was observed, indicating the importance of pollutant capture in rural areas and the lack of a clear source-sink relationship. There were significant same site species differences in the rate of capture for coarse particulates, with pine trees capturing the most and poplar the least. Significant species difference was again observed for fine particles, with pine again capturing the largest amount. The ultra fine particles followed a similar species pattern and were statistically greater for the urban site, although there were complications with the statistical calculation. These were found to be of a similar weight to the fine particles. Despite the important contribution of the studies by Beckett *et al.* (2000a; 2000b), their results could not be used to estimate the total particulate uptake per year. Except for Corsican pine, the tree types were not characteristics of the main species in the FC estate and private commercial woodland. In addition the study sites were not characteristics of forests and woodland sites, and the study did not model the absorption rate of particulate matter over space.

Three previous studies considered the impact of PM₁₀ absorption; McPherson *et al.* (1994); McPherson *et al.* (1998) and Nowak *et al.* (1998). Nowak *et al.* (1994) estimated that on average approximately 9.8 tons per day of PM₁₀ had been removed by trees in the Chicago area, improving the average hourly air quality by 0.4% (2.1% in heavily wooded areas). McPherson *et al.* (1998) considered a new plantation of trees in Sacramento, USA, and estimated that 30 year average annual deposition of PM₁₀ per 100 trees was 13.5 kg, however, given the small area considered, it was not appropriate to estimate the overall improvement in air quality. Nowak *et al.* (1998) calculated the trees in Philadelphia had removed approximately 418 tonnes of PM₁₀ in 1994, improving air quality by 0.72%.

ii) Ozone (O₃)

Ozone mainly occurs in the stratosphere, between heights of 15 and 50km. It is formed from the action of ultraviolet light. Ozone is also present in the troposphere, formed mainly by the action of ultraviolet light on volatile organic compounds (VOCs) and natural nitrogen oxide (NO_x), although a small amount comes from diffusion from the stratosphere. This naturally occurring pollutant provides low and stable concentrations and provides little risk to health. Additional ozone can be formed in the troposphere as a secondary pollutant and is produced by photochemical reactions with other pollutants, primarily volatile organic compounds and nitrogen dioxide. For example, although a reduction in NO_x would normally improve local air quality, under certain conditions it may increase the ozone concentration due to a reduced NO_x scavenging of O₃. Indeed, the formation mechanisms are very complex

involving a large number of gases and, as ozone is formed through ultraviolet light and affected by temperature, its concentration varies throughout the day and night.

McPherson *et al.* (1994), Broadmeadow and Freer-Smith (1996) and McPherson *et al.* (1998) have demonstrated that trees can remove large quantities of ozone from the atmosphere³. Nowak *et al.* (1998, 2000) strongly criticised the work of McPherson *et al.* (1998) for simplifying the issue and not calculating the net effect on urban trees. Taha (1996) and Nowak *et al.* (2000) have provided net effect estimates. Taha (1996) considered the following, where tree loss:

- changes chemical reaction rates;
- increases biogenic hydrocarbon emissions from vegetation through increasing temperatures;
- decreases biogenic hydrocarbon emissions from having less trees;
- changes the depth of the mixing layer; and
- decreases pollutant deposition in the vegetative canopy.

The findings suggested that a 20% loss in the wooded area due to urbanization in Los Angeles would lead to a 14% increase in ozone concentrations. Nowak *et al.* (2000) provided a more detailed consideration of the net effect on ozone levels for urban areas in the North Eastern United States, but the findings were less clear. The model produced found an increase in tree cover to both increase and decrease ozone levels throughout the day. Between the hours of 5am and 19:00 a net decrease in ozone levels of 1.9% was recorded due to urban trees, but during the evening there may be a local increase in Ozone. Furthermore, although there was a localised net decrease in ozone, due to decreased wind speeds and dispersion, increased tree cover could lead to a slight overall increase in ozone concentrations in surrounding areas. Although the net impacts in rural areas are not known, it is necessary to be aware that there is a need to consider other effects beyond an increase in the pollutant deposition by the tree canopy.

iii) Sulphur dioxide (SO₂)

Sulphur dioxide is a primary pollutant that is formed when sulphur is burnt with oxygen during the burning of fossil fuels. Currently the main source of sulphur dioxide is from coal-fired power stations, with other fossil fuels being less contaminated with sulphur. The formation of sulphur dioxide is less complex than ozone, and as such is simpler to model. McPherson *et al.* (1994), Broadmeadow and Freer-Smith (1996), McPherson *et al.* (1998) and Nowak *et al.* (1998) have demonstrated that trees can remove large quantities of sulphur dioxide from the atmosphere. McPherson *et al.* (1994) estimated that on average approximately 3.9 tons per day of SO₂ had been removed by trees in the Chicago area, improving the average hourly air quality by 1.3%. For the Greenwood Community Forest north of Nottingham, Broadmeadow and Freer-Smith (1996) estimated an annual pollutant removal of 2.1 kg per hectare of forestry. Based on a literature review of previous studies, a summary of deposition velocities are also provided by Broadmeadow and Freer-Smith (1996) for a mixture of broadleaved and coniferous studies. McPherson *et al.* (1998) estimated that 30 year average annual deposition of SO₂ per 100 trees

³ Based on a literature review of previous studies, a summary of deposition velocities is provided by Broadmeadow and Freer-Smith (1996), with the majority of studies dealing with coniferous species.

was 0.8 kg, however, given the small area considered, it was not appropriate to estimate the overall improvement in air quality. Nowak et al. (1998) calculated the trees in Philadelphia had removed approximately 163 tonnes of PM₁₀ in 1994, improving air quality by 0.29%.

3. Epidemiological effects

Physical damage functions relating health (mortality and morbidity) to air pollution levels have been estimated over a number of years in different countries. The health impacts from air pollution should be the net effect controlling for all other factors. However, this is impossible even in the best designed studies, due to genetic variation, different behavioural patterns, different past exposures, and errors in the measurement of air pollution and diagnosis of causes of mortality and morbidity.

Particulate matter of less than 10 microns diameter (PM₁₀), from road transport and other sources, gives rise to a wide range of respiratory symptoms and are also linked to heart and lung disease since particulates carry carcinogens into the lungs. An epidemiological study by Dockery *et al* (1993) traced a sample of 8,111 adults between 25 and 74 years of age for between 14 and 16 years in 6 different locations. Another study by Pope *et al* (1995) had a sampled 552,138 over 7 years in 151 locations for sulphates and 50 locations in the USA for fine particles. From these studies, Ostro (1994) concluded that the dose-response (D-R) relationship for PM₁₀ is

$$\%dH_{MT} = 0.096.dPM_{10}$$

where dH_{MT} is the change in mortality. This coefficient is, as far as possible, net of other factors such as smoking. Thus a 1 $\mu\text{g}/\text{m}^3$ change in PM₁₀ concentrations is associated with a 0.1% change in mortality, or a 10 $\mu\text{g}/\text{m}^3$ change in PM₁₀ concentrations is associated with a 1% change in mortality. However, there is considerable variation in the different D-R study results such that the upper and lower bounds of the D-R estimate is given as $\%dH_{MT} = 0.130.dPM_{10}$ and $\%dH_{MT} = 0.062.dPM_{10}$, respectively (see Pearce and Crowards, 1996).

Epidemiological studies relate daily mortality data in a particular location to meteorological variables (temperature) and PM₁₀ levels. Thus for example, Schwartz (1993), for Birmingham, Alabama, related daily deaths to 3-day averages of PM₁₀, and estimated that a unit milligram per cubic metre ($\mu\text{g}/\text{m}^3$) rise in PM₁₀ would increase the rate of deaths in the elderly population by 0.08%. Variance in estimates from epidemiological models arise from:

- (a) choice of meteorological variables: e.g. Smith (1997) re-analysed Schwartz's data and included humidity as an additional variable, and found it an important factor. Thus, the sensitivity of the estimated PM₁₀ effect to the choice of meteorological variables remains an important issue.
- (b) choice of exposure measure: e.g. different combinations of current and lagged days used as PM₁₀ averages
- (c) existence of thresholds: the bulk of evidence for PM₁₀ is for values above 80 $\mu\text{g}/\text{m}^3$ whereas the proposed UK NAQS is 50 $\mu\text{g}/\text{m}^3$
- (d) uncertainty as to the interpretation of air pollution mortality data: whether it causes mortality displacement (individuals dying are those already sick

who would have died anyway: evidence for this is indirect, but is the scenario adopted by NAQS) or mortality amongst otherwise healthy individuals. Little is known about individuals who are dying since only aggregate statistics are used.

- (e) influence of different pollutants: there is substantial chemical coupling between the different pollutants, such that it is difficult to separate out a specific effect due to PM₁₀. For example, in one study of PM₁₀ data from Philadelphia, which also included ozone, sulphur dioxide (SO₂), NO₂, and carbon monoxide (CO), all 5 pollutants were statistically significant, but the coefficient for NO₂ was negative, probably as a result of multicollinearity among the covariates. In another study on Chicago, all 3 pollutants in the analysis, PM₁₀, ozone, and SO₂, were significant, but now the coefficient on SO₂ was negative.

There are many issues and problems, including econometric problems, in defining a D-R relationship. The appropriateness of the D-R relationship depends upon the functional form: evidence from Schwartz (1994) and Dockery *et al* (1993) suggest the mortality function is approximately linear, but more work is required to verify whether the functional relationship is linear or non-linear. In addition, there may be threshold levels which might give rise to a strong attenuation effect at low exposure levels of PM₁₀, although there is no evidence of this to date. Thus, the D-R relationship may over-estimate the health effects of concentrations of low PM₁₀. There is also the econometric question of whether the D-R model has adequately controlled for all the other variables affecting health status, such as smoking, diet, social status, income, indoor and outdoor concentrations of PM₁₀, etc. Failure to do so will result in omitted variable bias, and biased estimates of the intercept and coefficients on the remaining variables in the model. Indeed, one of the main difficulties in investigating the effects of air pollution is that a number of pollutants are usually present together in the atmosphere. Most epidemiological studies have been concerned with the effects of mixtures of pollutants. Laboratory studies on the other hand have concentrated on the effects of high concentrations of individual substances. Unless the D-R relationship between health and individual pollutants are separable, D-R functions may over-estimate health impacts. Some of the data sets used in the various D-R function studies also appear to correspond to a Poisson distribution: variation in the observed values may correspond to that expected from random events. There is also concern in D-R studies of the biological pathway by which the pollutant affects human health: this is a current controversy with respect to childhood leukaemia and the location of electric power lines; and also the D-R relationship between air pollution and damage to trees. Finally, there is the question of transferability of the D-R functions, from largely empirical studies of American cities, to areas across the UK, where age, health, and other profiles for the population may be different. The question of the transferability of D-R functions is an outstanding issue that has not been resolved.

The Committee of the Medical Effects of Air Pollutants (COMEAP) assessed available evidence on health effects of air pollution and identified dose-response functions that could be applied with reasonable confidence in the UK (Department of Health, 1998). Evidence for the effects of nitrogen dioxide and carbon monoxide on health was not considered sufficiently robust for quantification. The dose-response functions identified by COMEAP as quantifiable are presented in Table 1. Only two

types of health outcome are reported: increases in mortality and increases in respiratory hospital admissions. The data relating levels of air pollution to hospital admissions are also based on aggregate statistics. It is not known how many people are being admitted to hospital who would not otherwise have been admitted at all, or how many people are simply being admitted to hospital sooner than otherwise expected. Nor do studies distinguish between first admissions and readmissions. For the mortality effects of PM₁₀, the Department of Health (1999) estimates reflect the more conservative aspects of American evidence, and hence err on the side of discretion.

Table 1: Exposure-response coefficients

Pollutant	Health Outcome	Dose-Response relationship
PM ₁₀	Deaths brought forward (all causes)	+0.75% per 10 µg/m ³ (24 hour mean)
	Respiratory hospital admissions	+0.80% per 10 µg/m ³ (24 hour mean)
Sulphur dioxide	Deaths brought forward (all causes)	+0.60% per 10 µg/m ³ (24 hour mean)
	Respiratory hospital admissions	+0.50% per 10 µg/m ³ (24 hour mean)
Ozone	Deaths brought forward (all causes)	+0.60% per 10 µg/m ³ (8 hour mean)
	Respiratory hospital admissions	+0.70% per 10 µg/m ³ (8 hour mean)

Source: Department of Health (1999).

The Department of Health (1998) report also shows that the impact differs according to age of population (see Table 2). For PM₁₀ the mortality effects are clearly distinguishable by age of population. However, the information is not complete: hospital admissions are not reported by age, whilst the mortality effects of sulphur dioxide and ozone are also not reported. Moreover, the Department of Health (1999) report does not explain why all the age specific respiratory hospital admission effects for sulphur dioxide (reported in Table 2) are lower than the aggregate effects (reported in Table 1).

Table 2: Exposure-response coefficients by age of population

<i>Pollutant & age</i>	<i>Mortality</i>	<i>Respiratory hospital admissions</i>
<i>PM₁₀</i>		
All ages	1.2% increase per 10 µg/m ³	
< 65	0.5% increase per 10 µg/m ³	
> 65	1.8% increase per 10 µg/m ³	
<i>Sulphur dioxide</i> (daily mean)		
15-64 years		0.2% increase per 10 µg/m ³
65+		0.4% increase per 10 µg/m ³
<i>Ozone</i> (8 hour average)		
15-64 years		0.6% increase per 10 µg/m ³
65+		0.75% increase per 10 µg/m ³

Source: Department of Health (1999).

4. Economic costs of air pollution

Mortality

For mortality, the Department of Health (1999) adopted a willingness-to-pay (WTP) approach to assess the value people place on reductions in risk, i.e. the value of prevention of a statistical fatality (VPF) from air pollution. Since no direct work had addressed this problem they modified the DETR's WTP-based values for the prevention of a road fatality by the factors that influence people's WTP for avoiding particular risk, viz type of health effect (lingering or sudden), risk context (voluntary, responsibility, etc.) futurity (sooner or later), age, remaining life expectancy, attitudes to risk (younger people less averse to risk), state of health related quality of life, level of risk exposure, and wealth/income/socio-economic status.

Adjusting DETR's road VPF of £847,580 (1996 prices) for risk context produced an air-pollution base-line VPF of around £2 million. This value was then modified to account for the other factors such as age, impaired health state, futurity, etc. (Table 3).

Table 3: Adjustment of air pollution VPF by supplementary factors
(£ millions, 1996 prices)

<i>Factor</i>	<i>Calculation</i>	<i>VPF</i>	<i>Justification</i>
Age	£2 * 0.7	£1.400	WTP >65 years 0.7 mean value of population
Reduced life expectancy	£1.4 * 1/12	£0.120	Reduction of 1 year of average life expectancy beyond retirement age
Reduced life expectancy	£1.4 * 1/12 * 1/12	£0.010	Reduction of 1 month of average life expectancy beyond retirement age
Impaired health status	£0.120 * 0.7/0.76	£0.110	Lower quality of life (QoL) than average elderly population (0.76) and with COPD with rated QoL 0.4 (std. 0.2-0.7)
Impaired health status	£0.120 * 0.2/0.76	£0.032	Lower quality of life (QoL) than average elderly population (0.76) and with COPD with rated QoL 0.4 (std. 0.2-0.7)
Risk, wealth, income, socio-economic status			No adjustment advocated
Futurity	5yrs : 95% 10 yrs : 90% 15 yrs : 86% 20 yrs : 82% 25 yrs : 78%		Mortality occurs at some time in future after first exposure to air pollution. Thus, future risk reductions benefits are valued at current rates discounted by pure time preference rate (1%)

Source: Department of Health (1999).

The Department of Health estimated using this procedure that the WTP for a small reduction in risk per death brought forward had an upper-bound of £1.4 million and a lower-bound of £32,000 to £110,000 for 1 year, and £2,600 to £9,200 for 1 month delay in the probability of death from air pollution.

Morbidity

The benefits of reduced morbidity comprise reductions in

1. public costs e.g. cost to NHS;
2. private costs to households e.g. for medicines, etc.;
3. lost output of people prevented from working due to ill-health;
4. welfare costs (reflecting on the pain and discomfort of illness).

The Department of Health (1999) estimated NHS costs of £1400 to £2500 for a respiratory hospital admission; and about £1,500 to £1,700 for a cardiovascular admission, for admission to a standard medical ward; with some unquantified variance around these costs.

No estimates were provided for private costs and lost output. Lost output would be small, and indeed zero for those >65 who were retired. However, the Department of Health (1999) report did not mention that there would be some lost 'black economy' output as a consequence of the illness of these individuals (loss of casual part-time jobs, inability to undertake own home improvement jobs, loss of services e.g. in terms of looking after grand-children, etc.). These might amount to 10% of wage rate individual obtained whilst in employment.

The Department of Health (1999) estimated WTP to avoid a hospital admission of 8 to 14 days. Their procedure arrived at an intuitive average of different WTP estimates relating WTP to avoid a deterioration in Quality of Well Being (QWB)⁴ (1=normal; 0=dead) necessitating hospital admission. WTP was estimated to increase as QWB scores declined, and decrease as length of hospital admission increased. The Department of health (1999) report concluded that:

“We consider giving the estimate as a range from £170 to £735 (at 1996 prices) best reflects the uncertainties. A single “mid” estimate could be derived using the mean QWB score of 0.6 and a mid-point of 11 days for duration (although this is not necessarily more likely than another duration between 8 and 14 days). This would give an estimate of about £530 (1996 prices).” (page 99).

Thus, assuming a mid-point for duration of hospital admissions of 11 days, and a change in QWB score from 0.6 to 0.47, this value of £530 per hospital admission avoided (up-dated to May 2002 prices) was employed to value avoidance of hospital admissions from reductions in PM₁₀ through adsorption by trees.

5. Study methodology

This study assesses the improvement in health and reduced economic cost of pollution due to tree absorption for Britain as a whole. As many assumptions are required and the science on which they are based provides a number of uncertainties this is a difficult task. It is particularly evident in the case of ozone that the complexities involved are such that the net absorption for this pollutant was not estimated.

i) Data sources

The UK National Air Quality Information Archive provides data on the relevant pollutants. The air quality information is provided in terms of daily average gravimetric units ($\mu\text{g}/\text{m}^3$) for both sulphur dioxide (1996) and PM₁₀ (predicted 2004 total particles). Data on the type and spatial distribution of woodland was provided by the Forestry Commission on a 1km² basis and does not include the many types of woodland within other land uses⁵. The average rainfall data was collected from the Met Office web site on a weather station basis.

ii) Net pollution absorption

Estimation of the dry pollution deposition from trees is conducted using the following equation:

$$ABSORPTION = FLUX * SURFACE * PERIOD$$

where:

$$FLUX = \text{deposition velocity (m s}^{-1}\text{)} * \text{pollutant concentration (}\mu\text{g}/\text{m}^3\text{)}$$

⁴ QWB is a scale for measuring health related quality of life.

⁵ See Hewitt (2002) for a more in depth study. However, at the time of writing no research output was available.

SURFACE = area of land considered (m²) * surface area index (m² per m² of ground area)

PERIOD = period of analysis (days) * proportion of dry days * proportion of in leaf days

Table 4 provides a summary of the deposition velocities and surface area statistics adapted from those available in the literature. Most of the statistics are based on US studies, where the deposition rate may have slight differences in the British context (Nowak *et al.*, 1998 and Broadmeadow and Freer-Smith, 1996). For example, their estimation is based on average wind profiles. Edge effects and the size and the difference in canopy space of trees in urban and rural locations have also not been considered. Other factors such as rainfall patterns and on-leaf periods have been adjusted for within the methodology (assumed May to September inclusive to the on-leaf period).

The net effect was determined by the woodland pollution absorption minus that of heather or grass seen as the alternative land use. This does not include, for example, the edge effects where such trees are more effective at absorbing pollution. Based on regional average Met Office data, days with over 1mm of rain were deemed to be rainy days and deposition velocity set to zero. Although the extent of dry deposition when it rains is unclear, this assumption may be too restrictive. For example, stomata will still be open taking in SO₂. Hence, pollution absorption was also estimated assuming that dry deposition continues on rainy days. Pollution concentration is based on average daily concentrations in terms of µg/m³, which is available for 1km grid-square and will provide the areal unit for analysis. Area prepared for felling was excluded from this analysis as it was unclear what the net effect would be in comparison to heather or grass.

Table 4: Deposition velocities and surface area statistics (m s⁻¹)

	Deposition velocity		Surface area	
	On-leaf	Off-leaf	On-leaf	Off-leaf
PM₁₀				
Conifer	0.0080	0.0080 ^a	9	9
Deciduous	0.0050	0.0014	6	1.7
Heather or grass	<i>0.0010^b</i>	<i>0.0010</i>	2.5	1.7
SO₂				
Conifer	0.0816	0.0816	9	9
Deciduous	0.0526	<i>0.0100</i>	6	1.7
Heather or grass	<i>0.0100</i>	<i>0.0100</i>	2.5	1.7

Note:

a. With the exception of larch, there is no off-leaf period so the on-leaf deposition velocity is stated.

b. Figures in italics are educated guesses made by the authors in the absence of information in the literature.

iv) *Health effects*

The change in health effect, mortality or morbidity for a given pollutant is calculated as

$$dH_{MT} = DR * POLLUT * POP * RATE * 1/100$$

where:

dH_{MT} = change in mortality due to forestry foliage;

DR = dose response coefficient (i.e. 0.075);

$POLLUT$ = net reduction in pollution due to forestry foliage;

POP = population of the 1km²; and

$RATE$ = morbidity or mortality rate for the 1km².

The factor 1/100 converts percentages to absolute numbers.

The resident population for each 1km² will be estimated using 1991 Population Census⁶. An estimation of 200m grid squares was aggregated to 1km².

Information on mortality rates was derived from the Office of National Statistics (2000). Mortality rates by county were applied to estimate the number of baseline deaths, from which to estimate the change attributable to air pollution absorption by woodland. More geographically specific mortality data, e.g. by 1 km², was not available.

Information exists on hospital admissions for specific causes (OPCS, 1987) e.g. diseases of the respiratory system, and within this general category, respiratory diseases that might be caused or exacerbated by air pollution (i.e. respiratory diseases not caused by air pollution can be excluded). However, whilst this information exists at a national level, and by health authority areas, it is not ascribed to the area of residence of the individual, and not on a 1km² grid square basis. Therefore, for morbidity, the national rate of hospital admissions due to air pollution was applied to the 1km² areas.

This is consistent with COMEAP and EAHEAP procedures. They applied baseline death rates and respiratory hospital admissions for PM₁₀ and SO₂ of 1074 deaths and 830 hospital admissions per 100,000 population (<http://www.aeat.co.uk/netcen/airqual/reports/healthrep/hchpt2.html>) (25/03/2002).

The health effects of PM₁₀ and SO₂ were treated as additive, although this has not been definitively established. Air pollution is a mix of different compounds. Evidence points to PM₁₀ as being the main problem. SO₂ is more problematic. However, in terms of air pollution, SO₂ is correlated more highly with NO_x than with PM₁₀.

The COMEAP dose-response functions may underestimate the benefits of air quality improvements, because it places greater weight on the proximity in time between air pollution and mortality and morbidity. That is, the function looks at the immediate effect of air pollution, and underestimates the long-term impact of air pollution.

⁶ Source: The 1991 Census, Crown Copyright ESRC/JISC purchase. The surface data used in this work were generated by David Martin, Ian Bracken and Nick Tate, and obtained from Manchester Computing.

v) *Reduction in economic costs*

The reduction in economic costs was estimated by merely multiplying the number of deaths brought forward and hospital admissions by the costs noted above adjusted to 2002 prices. Given the uncertainty as to the period which deaths are brought forward, two costs were used reflecting an average period of one month and one year. With the average period of one month as the lower bound.

6. Results

Tables 5 and 6 provide a summary of the results by the Britain as a whole and by country and region, with the former excluding days of more than 1mm rain. Trees can be seen to absorb large quantities of pollutants, for example, between 391,664-617,790 metric tonnes of PM₁₀ and 714,158-1,199,840 metric tonnes of SO₂ per year. Using the methodology adopted here the impact in terms of net health effects, of having trees compared to another land use, has been estimated to be between 65-89 deaths brought forward and between 45-62 hospital omissions. Although these numbers are significant, a larger health effect was expected. However, these results can at least be partially explained due to a mismatch caused by the lack of correspondence between people and trees. A large number of 1km² contain trees but no people, whereas the highest populated areas tend to have little if any trees (at least not recorded on the data set provided). Furthermore, the spatial effect of pollution absorption by trees is not well understood and it may be that the 1km² spatial extent used is not appropriate. Current science provides little guidance on this and there is a clear need for research combining dispersion and absorption by trees.

The counties with greatest health effect (over one death brought forward) are Strathclyde, Surrey and Hampshire. Surrey and Hampshire both have above average pollution levels, population densities and proportions of deciduous trees. In the case of Strathclyde its pollution levels and population density area are slightly below average but it has an above average proportion of conifer and deciduous trees. Indeed, the absorption levels are very high. Other important areas include Manchester, Lothian, Mid-Glamorgan and Outer London. These are notable in that they all have population densities above average. Greater Manchester and Outer London also have high pollution levels. Mid-Glamorgan and Outer London also have above average tree levels in terms of conifers and deciduous respectively.

A monetary measure of the net costs forgone or net benefits of having trees, instead of another land use, was also estimated. The total net benefit for Britain has been estimated to be somewhere between £222,308 and £11,213,276. This is clearly a broad range and is dependant on the extent of dry deposition on days with more than 1mm rain and how much early the deaths brought forward occur, with 1 year being assumed for the upper bound. This broad range may, however, have been set too narrow and the net effect of other pollutants absorbed, such as Ozone, has not been included.

Pollution absorption in this study was modelled at a 1 km² level. Data from the National Inventory of Woodland and Trees (Smith, 2002) is accurate at this level, and distinguishes between broadleaved and conifer tree by age of trees. It is also the most detailed level at which estimates of PM₁₀ and SO₂ are available (based on

extrapolation from samples). However, most studies estimating the capture of particulate matter by trees have assessed trees in urban areas often in proximity to road traffic as a generator of PM₁₀. Most PM₁₀ is captured by trees close to source. Thus if pollution absorption was considered at a more detailed level below 1km², then the localised effects on health may increase or decrease depending on the location of population in relation to woodland and the source of PM₁₀. However, to estimate such effects would require more detailed data on woodland, pollution and population, some of which is not currently available. Conversely, the effects of absorption, particularly for smaller particles, may be wider than 1km². Many factors affect the dispersion of PM₁₀ from source to sink, and accounting for possible health benefits at a wider level than 1km², would significantly increase the inaccuracies caused by not modelling the dispersion of pollutants.

7. Conclusion

The review of the literature has shown pollution absorption by trees to be sizeable. The health effects of pollution are also large delaying many deaths and preventing hospital omissions from poor air pollution. This research has endeavoured to investigate the link between pollution absorption and health effects, considering both PM₁₀ and SO₂. Ozone was also seen to be an important pollutant but was excluded from this analysis due to the complexity of the link between the effects of vegetation and ozone formation and absorption.

The research has attempted to estimate the net health effects and the reduction in economic costs due to the current tree resource in Britain. Given the current lack of understanding of the link between pollution dispersion and tree absorption of pollutants the research has been based on a scale of 1km². The results have found net pollution absorption by trees to have reduced the number of deaths brought forward by air pollution by between 65-89 deaths brought forward and between 45-62 hospital omissions, with the net reduction in costs estimated to range somewhere between £222,308 and £11,213,276. Aggregating the data initially on a county basis, the population of Hampshire, Strathclyde and Surrey have benefited the most, with the net improvement in air quality also being important within Greater Manchester, Lothian, Mid-Glamorgan and Outer London.

Given the magnitude of this task and the limitations in terms of the resources available, many simplifying assumptions have been made, with perhaps the most notable being the spatial extent of the benefits from pollution absorption. In most 1km² in Britain there is a mismatch in that areas that have the most people have little trees and vice-versa. The results of Beckett *et al.* (2000a) suggest, however, that in the case of the finer PM_{2.5} particles absorption of trees within rural areas may be of wider importance. However, the science concerning this issue is as yet unclear and there is a need for further research. Issues such as the difference in absorption due to location (size of the trees and temperature at which the absorption occurs), wet deposition, edge effects of forests and the link between dispersion of pollution and absorption have not been considered. These were omitted due to the resources available for this project, however, it should also be noted that in the case of the latter two issues the resource requirements for their inclusion for the whole of Britain would be large. Many issues have been considered including rainfall, pollution levels, tree type, population, as well as regional differences in mortality rates.

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Table 5: Summary of the health effects and benefits by Region and Country (days with more than 1mm rain excluded)

Country and Region	Mean conifer per 1km ² (hectares)	Mean deciduous per 1km ² (hectares)	PM10 (kg)	SO2 (kg)	Mean population per 1km ²	Deaths brought forward (per year)	Hospital omissions (per year)	Total benefits (£) Lower bound ¹	Total Benefit (£) Upper bound ²
Wales	11	6	44,585,375	91,327,084	133	5.43	3.42	18,284	680,303
Scotland	19	7	194,041,451	234,687,708	62	16.75	10.67	56,524	2,100,437
England	5	7	153,037,112	385,143,467	741	43.20	30.44	147,501	5,417,522
Britain	10	7	391,663,938	711,158,259	312	65.37	44.53	222,308	8,198,262
English Regions									
East Midlands	4	6	7,909,195	32,599,311	234	2.20	1.51	7,490	275,860
East of England	5	6	18,045,989	48,859,306	259	4.34	3.04	14,806	544,124
London	1	9	628,145	2,613,927	4176	2.69	2.29	9,435	338,043
North East	13	5	22,102,722	39,511,654	294	2.90	1.90	9,811	363,399
North West	6	6	13,490,437	28,593,247	465	6.00	3.87	20,268	752,015
South East	5	13	33,772,286	89,279,419	370	12.16	9.02	41,789	1,525,048
South West	4	8	26,083,395	49,977,795	186	4.12	2.64	13,900	516,208
West Midlands	4	6	15,265,272	48,557,856	368	4.55	3.24	15,571	571,153
Yorkshire and Humberside	5	6	15,739,671	45,150,952	313	4.24	2.91	1,443	531,671

Notes:

1. Lower bound based on deaths brought forward only one month.
2. Upper bound based on deaths brought forward 1 year.

Table 6: Summary of the health effects and benefits by Region and Country

Country and Region	Mean conifer per 1km ² (hectares)	Mean deciduous per 1km ² (hectares)	PM10 (kg)	SO2 (kg)	Mean population per 1km ²	Deaths brought forward (per year)	Hospital omissions (per year)	Total benefits (£) Lower bound ¹	Total Benefit (£) Upper bound ²
Wales	11	6	73,381,012	1,645,94,297	133	9.07	5.69	27,553	1,011,889
Scotland	19	7	320,220,195	4,282,89,724	62	16.75	10.67	56,524	2,100,437
England	5	7	224,188,853	6,069,56,316	741	64.59	45.31	220,437	8,100,950
Britain	10	7	617,790,060	1,199,840,337	312	89.41	61.67	304,513	11,213,276
English Regions									
East Midlands	4	6	11,295,032	50,690,930	234	3.31	2.27	11,251	414,549
East of England	5	6	25,116,531	73,850,590	259	6.27	4.39	21,398	786,626
London	1	9	855,056	3,713,872	4176	3.73	3.18	13,078	468,619
North East	13	5	33,352,990	65,554,115	294	4.34	2.84	14,685	544,146
North West	6	6	21,576,115	48,947,215	465	9.67	6.23	32,673	1,212,583
South East	5	13	47,188,309	1,33,530,342	370	17.64	13.06	60,625	2,213,126
South West	4	8	38,587,913	79,633,675	186	6.25	3.99	21,095	783,673
West Midlands	4	6	22,559,039	77,443,781	368	6.97	4.95	23,837	874,653
Yorkshire and Humberside	5	6	23,657,868	73,591,796	313	6.40	4.40	21,795	802,975

Notes:

1. Lower bound based on deaths brought forward only one month.
2. Upper bound based on deaths brought forward 1 year.