An initial investigation into the potential link between air pollution and asthma using geographical information system based technique

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Abstract

One of the central research concerns about air pollution and respiratory disease is whether air pollution is contributing to the induction of asthma thereby increasing its incidence.

This study, used a Geographical Information System (GIS) to compare the distribution of nitrogen dioxide, a known traffic sourced bronchoconstricting agent, with the postcode distribution of emergency asthma admissions into local hospitals.

The potential of GIS as a modelling tool for combining air quality monitoring and epidemiological studies on a rapid basis is demonstrated. Further development of this approach may provide an invaluable aid to public health monitoring.

A significant correlation of 0.882 was found between asthma admission and NO2 levels for adults while the correlation for children was 0.496. The adult figure is considered significant for an epidemiological study.

Health and the atmospheric environment

Relationships between the atmospheric environment and its impact on human health have been well established. Well-known examples include the 4,000 excess deaths caused by the notorious London smog of 1952 an episode of illness only exceeded in the UK this century by the influenza pandemic of 1918; and the adverse effects of atmospheric lead and alkyl-lead compound concentrations on the neurophysiological behaviour in children [1]. Recent international research [2] suggests that episodes of air pollution and short term fluctuations in pollutant levels may give rise to temporary

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reductions in lung function and, more disturbingly, increased cardiorespiratory mortality.

**Increase in the prevalence of asthma**
The American Thoracic society define asthma as “a disease characterised by an increased responsiveness of the trachea and bronchi to various stimuli and manifested by widespread narrowing of the airways ...” [3] this condition causes extreme breathlessness amongst sufferers, which in a worst case scenario can lead to death. Symptoms of asthma may be brought on by a variety of trigger factors and the disease has been associated with housing conditions (dampness, heating systems and ventilation), occupation, environmental factors and lifestyle [4].
The prevalence of asthma, notably in the young, has been seen to rise steadily over the last few decades, although the actual level of increase is uncertain, due to lack of long term data. Current research suggests, about 1 in 10 UK primary school children are diagnosed as asthmatic and a further 5 to 10% have recurrent respiratory symptoms that are suggestive of asthma but are undiagnosed [5,6]. Studies have also shown that the increases in asthma are not attributable to increases in diagnosis [7,8]. In the UK asthma is now the most common reason for the admission of children into hospital and is the cause of an estimated 2000 premature deaths a year. In economic terms, the cost of asthma in the UK to the NHS has been recently estimated to be of the order of £400 million [8]. Thus asthma has become a topic of great social and political concern; fuelled by the possibility that traffic emissions could be contributing to this increase. A number of UK studies [5,7,10] have compared childhood asthma prevalence in the same geographical areas separated by time intervals. These studies all used questionnaire surveys completed within a geographical location, each showing an increase in prevalence. Fleming [8] also performed a study in adults using a representative sample of approximately 3000 people across England and Wales comparing 1971-2 and 1981-2 reports. He found the prevalence in men had increased from 11.6 to 20.5 per 1000 people, and for women the increase was from 8.8 to 15.9 per 1000 people. Otherwise little research has been done on the prevalence of asthma in adults, yet American cohort studies suggest that two thirds of children with asthma will continue to have asthma attacks as adults. Other similar studies have been carried out in the developed world [11,12] showing similar increases in prevalence as in the UK, however in the third world only the ‘westernised’ populations rather than the ‘traditional’ populations have been showing similar increases [13]. This increase in prevalence cannot be attributed purely to changes in social conditions or in the willingness and ability of doctors to diagnose asthma, and hence it is possible that there is an associated environmental factor.

**Traffic related pollutants**
The most likely environmental parameter affecting asthma suffers is air pollution of which the greatest concern surrounds the increase in road vehicle related pollutants. Between 1979 and 1991 there was a 75% increase in traffic on UK motorways and a 33% increase in traffic on ‘A’ classification roads. Department of Transport forecasts expect total traffic to increase between 27 and 47% by the year 2000 and 83 to 142% by 2025 compared with 1988 [14]. Vehicle derived emissions have risen as a result of these traffic increases, for example a survey of Nitrogen Dioxide concentrations for the
periods July to December 1986 and July to December 1991 showed an average increase of 35% across the 240 study sites [15].

Key Atmospheric Pollutants thought to contribute to asthma
Although not generally associated with vehicle exhaust emissions, the presence or absence of Sulphur Dioxide, $SO_2$, a potent bronchoconstricting agent, is a good indicator of general atmospheric quality. Urban Nitrogen Oxides, $NO_x$, are generally vehicle sourced, and in conjunction with $SO_2$ can indicate differences between general air quality and air pollution from transport sources. Although $NO_x$ gases are bronchoconstrictors the concentrations required to cause unequivocal dysfunction are above ambient atmospheric conditions. However “pollution incidents” can provide the concentrations of $NO_x$ gases necessary to trigger an “asthmatic attack”. Ozone, $O_3$, is also a potent bronchoconstrictor in low concentrations [16]. Although a secondary pollutant, the primary factors associated with tropospheric $O_3$ production are from a road transport source.

Perhaps the most current major topic regarding air pollution and asthma is with particulate matter such as smoke and dusts ($PM_{10s}$). While not regarded as the major pollutants they once were before the Clean Air Act of 1956; the effects of diesel powered vehicle emissions are now causing concern [4,17]. Schwartz [18-20] has shown that rising particulate concentrations causes an increase in emergency admissions for respiratory disease, and that a $10\mu g m^{-3}$ rise in $PM_{10s}$ is associated with a 1% increase in mortality for all causes.

Other than considering these atmospheric pollutants in isolation it is possible that a synergistic effect between two or more of these pollutants exists. It is known that $SO_2$, $NO_2$ and $O_3$ interact and potentiate the effect of allergens in hay fever sufferers, [21] however the way in which pollutants interact and their combined role in the aetiology of chronic respiratory disease is poorly understood.

Geographical Information Systems (GIS)
In a GIS each set of information exists as a map layer or coverage. These layers can be combined to produce new maps using overlay functions which can be used to define information about the relationships between the original map layers. For example the distribution of pollutant concentrations could be held on one layer, and a distribution of medical conditions held on another, and these two maps could be combined to produce a third. The GIS processing was performed the ‘raster based’ package, ‘IDRISI’.

Survey region
This survey area is based in Stockport, a metropolitan district situated in the south east corner of the Greater Manchester County in the North West of England covering 12,605 hectares. Stockport is a closely defined geographical region, with a socio-economically well-defined population of 291,000 people. Earlier health studies have shown an increased number of emergency admissions due to asthma in wards of Stockport, previously unassociated with poor social class and standards of housing, the traditional factors associated with asthma prevalence. These studies have suggested the possibility of the influence of an environmental factor, as there is evidence that people who live along one of Stockport’s main traffic corridors with asthma suffer increased
morbidity [22]. Furthermore, the topography of Stockport is very varied, with a range of hill and valley sites which may present variations in atmospheric concentrations with height, notably during temperature inversions.

Sources of Data for the GIS study
Previous studies using GIS to assess the links between air quality and health have often made assumptions about spatial variations in air quality, and have been carried out without recourse to air quality data. This is often because GIS users, have lacked the expertise and access to technology to undertake an air quality monitoring programme. Consequently these studies have relied upon atmospheric dispersion models, often based upon gaussian distribution from a single point source [23]. For this study a sampling network of 16 sites using triethanolamine (TEA) based diffusion tubes [24] was created in the Stockport area to measure NO₂ concentrations over monthly periods. The pollutant monitoring was carried out between November 1993 and March 1994. Körner episode statistics (hospital inpatient admission data) for asthma was obtained from Stockport Health Authority. This consisted of sex, age, postcode of residence, date of admission and medical diagnosis for each patient. The postcode can be located to 100m on an Ordnance Survey grid square. Emergency asthma admissions was used to reduce mis-classification, and to provide more objective data than using a self reported symptoms questionnaire. In addition adult patients were interviewed by the hospital asthma nurse to verify that the diagnosis of asthma was correct. The patient data is temporally consistent with the atmospheric sampling. Both the pollution data and the asthma patients postcodes were plotted in relation to a digitised outline of Stockport for visualisation and interpretation of the data. The cell size within the GIS, i.e. smallest definable area, used in the digital map creation was a 33m grid, which offered sufficient resolution for both patient and pollution data.

Due to the relatively small number (164) of emergency asthma admissions over the survey period the patient data investigated as a whole rather than at discrete time intervals. Consequently, it was decided to produce, by interpolation, a compilation NO₂ pollution map to cover the whole survey period. Over this period NO₂ concentrations did not vary significantly at each test site, and variations were within the errors attributable to diffusion tube monitoring. Spatial interpolation of the pollution data, was achieved by algorithms within IDRISI. The postcodes of the asthma patients were then digitised onto a second map layer and overlaid on the pollutant data map. An example of the resultant overlaid map as shown for adult admissions and NO₂ concentrations can be seen in fig 1. A similar procedure was performed for the admission of children. The asthma patient home locations, and the associated NO₂ concentration, were extracted by the GIS for statistical analysis as shown in table 1. The number of asthma admissions was correlated with the NO₂ concentration around their postcode address with the number of patients in each area being adjusted against a standardised area based on pollution levels; in accordance with epidemiological techniques. The correlation coefficient value for the area standardised adult asthma distribution against NO₂ levels was 0.886, and for area standardised childhood asthma distribution against NO₂ levels, 0.496. As there appeared to be a strong correlation in adult admissions the non-area standardised values were correlated against NO₂ concentrations, which give a correlation coefficient of 0.353.
Table 1 - Adult asthma admission distribution according to NO\textsubscript{2} levels around their home.

<table>
<thead>
<tr>
<th>NO\textsubscript{2} concentration (\mu g m\textsuperscript{-3})</th>
<th>Number of adult asthmatic patients admitted to hospital</th>
<th>Number of asthmatic children admitted to hospital</th>
</tr>
</thead>
<tbody>
<tr>
<td>39.5</td>
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<td>0</td>
</tr>
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<td>1</td>
</tr>
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<td>3</td>
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<td>0</td>
</tr>
<tr>
<td>83.5</td>
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</tr>
</tbody>
</table>

Figure 1: Overlay map showing adult asthma admissions and NO\textsubscript{2} concentrations for Stockport November 1993 - March 1994

Key - White line = Stockport boundary
Cross - postcode address of asthma patient

NO\textsubscript{2} Concentrations
- Black Area - < 40 \mu g m\textsuperscript{3}
- Dark Grey Area - 41 - 50 \mu g m\textsuperscript{3}
- Grey Area - 51 - 60 \mu g m\textsuperscript{3}
- Light Grey Area - 61 - 70 \mu g m\textsuperscript{3}
- White Area - > 70 \mu g m\textsuperscript{3}

NB this image is compressed from original image for reproduction purposes.
While the area standardised adult asthma distribution against NO\textsubscript{2} levels was significant, the correlation for childhood admissions was much lower. This was probably due to a smaller data set being available for the childhood admission case thus any interpolation would use fewer points than for the adult map. For both adult and childhood data sets there were no asthmatic admissions associated with the lower NO\textsubscript{2} concentration levels; if there had these would have had a strong weighting from the standardisation process due to them having small areas, even though the low pollution bands are spatially larger than the high pollution bands.

The $\chi^2$ test was used to test the hypothesis that the relationship between patient standardised hospital admission distribution, for adults, and NO\textsubscript{2} concentrations occurred by chance gave a value of 284; demonstrating that there was an insignificant probability of such a distribution occurring by chance. The notable positive correlation for the relationship between adult patients and NO\textsubscript{2} when the patient numbers are standardised by pollution coverage area does suggest that there appears to be a positive relationship between increased NO\textsubscript{2} levels and asthma providing some evidence to support the hypothesis that traffic levels and asthma have a positive relationship. Using the dates of admission into hospital, a comparison was made, on a daily basis, of the actual number of cases admitted compared to the expected number of expected adult and children admission cases. A $\chi^2$ test produced a probability of 1 in 1000 that such a distribution occurred by chance for both, suggesting that hospital admissions could be stimulated by pollution events. As the diffusion tube survey only give a monthly mean concentration, pollution events will effectively become lost. Ideally, this study should therefore be repeated using shorter timescale reporting instrumentation.

**Discussion**

Without a known direct mechanism which would cause increased asthma occurrence when NO\textsubscript{2} levels increase there is the possibility that other causal factors are associated for example the effects of other traffic pollutants and obviously this preliminary study will lead to further areas of investigation. A further factor that requires consideration is that within GIS the mode of spatial analysis used can have marked effects on the outcome and that this correlation could be merely a product of standardising patient levels by areas of pollutant concentration. Further work such as that carried out by Openshaw [25] on ‘intelligent spatial analysis of data’ to reduce the uncertainties of the spatial analysis techniques within GIS need to be considered.

The key outcome of this study is that although a preliminary study, it has shown that there is validity in the GIS approach to an air pollution modelling problem. Even allowing for the fact the GIS spatial interpolation of NO\textsubscript{2} values, made no direct allowances for elevation, mixing height, and other meteorological factors which effect local NO\textsubscript{2} distribution. Because the interpolation used the difference in measured values between individual points, indirectly these factors were reflected in the model. The GIS approach was able to provide rapid analysis of the data to provide apparent correlation between asthma admissions and NO\textsubscript{2} levels, demonstrating that the traffic pollution / asthma link is worthy of further study.
Conclusions

Despite the limitations with this preliminary study into the use of GIS for interpreting health and pollution data; notably the length of the survey, it does display some of the potential that GIS can offer both epidemiologists and air pollution scientists in the interpretation of spatially held information. Because GIS can adjust the application of formulas during a query operation on spatially held information it cuts the time required to model the relationships between air pollution and other spatially held information dramatically. It has also produced evidence to support the hypothesis that traffic and asthma have a positive relationship. Within GIS it is already possible to use the results of more extensive air pollution modelling than the interpretation used in this study, and the visual interpretation that a GIS offers means that there is a much greater potential to extract potentially valuable data.

The town of Stockport is about to undergo an extensive period of road modernisation, which will result in the establishment of several major road networks in areas currently free of major traffic systems. Continuation and expansion of this study over a number of years, for example testing for other pollutants such as SO₄, O³, VOCs and PM₁₀ and using GIS to check socio-economic variation across the borough and relating them to asthma distribution; will make it possible to monitor the population within distinct time periods to determine increased traffic effects and any potential change in human health.

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References

1. First report of the Quality of Urban Air Review Group, Department of the Environment 1993.


