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APPENDIX 3.3: Detailed Obesity Evidence Base

OBESITY

Synopsis of the Evidence

In England, 15% of children between 2 and 10 year of age are obese and the prevalence of obesity and overweight children is increasing steadily. Further to this, since 1990 there has been a dramatic increase in the incidence of type 2 diabetes which has occurred in tandem with the prevalence of sedentary lifestyles and obesity.

There are ways to address this trend by encouraging physical activity. In adults physical activity reduces the risk for all-cause mortality as well as for cardiovascular disease, obesity and diabetes and musculoskeletal conditions. Physical activity does not need to be vigorous and sustained to achieve health benefits.

Factors key to supporting physical activity are:

a) Walking and cycle ways to connect homes with schools, workplaces and shops.
b) Accessibility to playing in the park and sports facilities.
c) Removal of environmental barriers to permit residents in poorer areas to become physically active.

There is evidence that creation or enhancement of the above factors is effective in engaging people across the socio-economic and ethnic spectrum in levels of physical activity that improve their health.

Spatial planning can encourage physical activity by promoting walking and cycle ways, improving accessibility to open and green spaces and sports facilities, and removing environmental barriers to allow residents in poorer areas to become physically active.

The prevalence of obesity and overweight in children is rising steadily and if this trend continues, obesity may constitute the single most serious health threat to the current generation of children. According to the latest figures from the Health Survey for England (HSE) (2002-04 data combined), 15% of children aged 2-10 are obese (http://www.dh.gov.uk/en/Publicationsandstatistics/PublishedSurvey/HealthSurveyforEngland)

The incidence of Type 2 diabetes has increased dramatically since 1990 and this rise has occurred in tandem with the prevalence of sedentary lifestyles and obesity. There are ways to address this trend by encouraging physical activity (McGinnis JM 2002) and in the U.S.A a large clinical trial (Diabetes Prevention Program DPP) demonstrated that through diet and engaging in walking or other moderate intensity exercise for thirty minutes each day, participants reduced their body weight by 5% to 7% with a consequent reduction in risk for diabetes of 60% (National Institute of Diabetes and Digestive and Kidney Disease 2001). 45% of the participants in the DPP trial were from minority groups that suffer disproportionately from Type 2 diabetes, including African-American and Hispanic-Americans, demonstrating that exercise intervention is effective in dealing with inequalities in health (Kriska A 2002). The physical activity prescription used in the DPP was similar to public health recommendations (Centres for Disease Control 1996, Pate RR 1995) which call for an increase in moderate levels of physical activity, such a brisk walking for about thirty minutes on most days. Such activities are more likely to be maintained over the years by people of different ethnic/racial and economic groups than are high-intensity sports (Task Force on Community Preventive Services 2002). Planning changes are needed to build environments that are more favourable to physical activity and which will maximise the likelihood that walking will be maintained.

Physical activity is essential to the aim of addressing the problems of sedentary living and obesity amongst children and adults. Accessibility (determined by land-use patterns and the transport system) to playing in the park, sport and other facilities is key to supporting physical activity. Proximity to routine destinations is an important correlate of physical activity (D.A. Cohen 2006) and residents living in poorer areas have more environmental barriers to overcome to become physically active (W.C. Taylor 2006) which highlights the need for environmental justice. Active living requires that development plans incorporate walking and cycle ways to connect homes with schools, workplaces and shops whilst implementing appropriate control of motor traffic. The World Health Organisation (WHO 2006) has produced guidance for local governments and examples of case-studies whereby physical activity and active living may be promoted in urban environments. For example, the Sandness Municipal Council in Norway has made a systematic effort to identify and promote the interests of children in planning. Schools and children have registered hundreds of play areas and short cuts on digital maps and these areas are required to be safeguarded in planning activities.

Physical activity does not need to be vigorous and sustained to achieve health benefits. Improved health can be attained through the accumulation of shorter bouts of moderate-intensity activity (US Department of Health and Human Services 1996); however, greater levels of activity do produce greater health benefits.

APPENDIX 3.3: Detailed Obesity Evidence Base

In the U.S.A the Task Force on Community Preventive Services conducted a systematic review of community interventions to increase physical activity. It found sufficient evidence to recommend creation of or enhanced access to places for physical activity combined with informational outreach activities. This was effective as measured, for example, by percentage of persons exercising on three or more days of the week (Task Force on Community Preventive Services 2002a, 2002b). Lack of data is one of the main barriers to progress in examining the causal links between the built environment and physical activity levels (Transportation Research Board of the Institute of Medicine, 2005) There is a need for geographic and environmental data which will permit the linking of information on physical activity with details about the respondents' location and physical environment (Boarnet MG 2004, Handy 2004, Handy et al 2002).

There is a growing awareness that cities can be designed to fight obesity (Larkin M 2003) and there is increasingly a marriage of city planning and public health. Planners have the imperative of solving problems of congestion and lack of open-space and public health professionals are realizing that these problems contribute to the prevalence of obesity in the population. There are initiatives in the U.S.A with projects looking at ways communities can be designed to encourage more physical activity (e.g. Active Living by Design: http://www.activelivingbydesign.org and American Planning Association: http://www.planning.org/physicallyactive). The U.K also has several well developed programmes for promoting fitness-friendly environments such as the UK's Walking the Way to Health Initiative (http://www.whi.org.uk) which is a project of the British Health Foundation and the Countryside Agency.

The rising prevalence of obesity and Type 2 diabetes as well as the recognised preventive measure of increasing physical activity resulted in guidance from the National Institute for Health and Clinical Excellence (www.nice.org.uk). NICE has published a quick reference guide for local authorities and the public in the prevention of obesity in adults and children (NICE 2006). This encourages planners to facilitate links between health professionals and others to ensure that local policies improve access to opportunities for physical activity and healthy food.

Evidence such as that cited above as well as others (Kahn E 2002, Spence JC 2001, ACES 2006) have made the case for how land use planning can impact on the public health and healthy living. Researchers are now addressing how perceptions of the local environment affect people's preparedness to exercise (Rutten et al 2001) as well as the issues relating to access to facilities by socioeconomic status (Gordon-Larsen P 2006). Also, attention is being focused on vulnerable groups such as children (Active Living Research 2005), people with disabilities (Active Living Research 2006) and older adults (Cunningham GO 2004, King AC 1998).

Regular physical activity is associated with enhanced health and reduced risk for all-cause mortality (Lee et al 1995, Paffenbarger et al 1993, Paffenbarger et al 1994, Blair et al 1995). Beyond the effects on mortality, physical activity has multiple health benefits, including reducing the risk for cardiovascular disease, diabetes, obesity, selected cancers and musculoskeletal conditions (Bouchard et al 1994).

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APPENDIX 3.4: Detailed Cardiovascular and Respiratory Disease Evidence Base

CARDIOVASCULAR AND RESPIRATORY DISEASE AND RISK FACTORS WHICH MAY BE MODIFIED THROUGH SPATIAL PLANNING

Synopsis of the Evidence

- There is strong quantitative evidence that respirable particles (PM10) in urban areas have a short term effect on deaths from all causes (0.75% per 10 μgm^{-3} PM_{10} as 24 hour mean) and respiratory hospital admissions (0.8% per 10 μgm^{-3} PM_{10} as 24 hour mean). The primary source of PM_{10} in urban areas is motor traffic.

- Nitrogen dioxide is produced by motor vehicle exhausts and is an effect modifier. Increase in daily mortality associated with a 10 μgm^{-3} increase in PM10 was 0.8% for a city with high long term average NO_2 concentrations compared to 0.19% for a city with low long term average.

- Ozone gives rise to population exposure in both rural and urban areas. Deaths (all causes) and respiratory hospital admissions are +3% and +3.5% respectively for 50 μgm^{-3} eight hour mean O_3 concentration.

◆ **Spatial planning can modify the total volume of traffic as well as congestion of traffic at certain locations. This may have a preventive effect on population CVD and respiratory disease by reducing air pollution.**

- There is evidence that vigorous physical activities such as brisk walking or cycling on most days of the week has a clear association with reduced risk of CHD in middle aged and older people.

◆ **Spatial planning has the potential to provide opportunities for walking to work, cycling and access to leisure facilities.**

- The epidemiological evidence for association between noise exposure and CVD is not strong.

- There is evidence that following flooding residents are more likely to report exacerbations of asthma, coughs and respiratory infections.

◆ **Spatial planning can prevent flood risk to residential properties.**

CARDIOVASCULAR AND RESPIRATORY DISEASE

Health Risks Associated with Respirable Particles in ambient air

There is a body of epidemiologic evidence which demonstrates that there are small health risks associated with population short-term (acute) and long-term (chronic) exposure to ambient levels of PM_{10} particles. The evidence base is sufficiently robust to permit quantitative risk estimates The DOH's Committee on the Medical Effects of Air Pollutants have reviewed the available epidemiological evidence and published exposure-response coefficients (COMEAP 1998) for short term effects of air pollution on health. These are:

- Deaths, all causes : +0.75% per 10µg/m^3 PM_{10} (24 hour mean)
- Respiratory hospital admissions: +0.8% per 10µg/m^3 PM_{10} (24 hour mean)

The estimates of the exposure-response relationships are based on the results of time-series studies. These studies examine the relationship between daily levels of pollution and the risk of adverse health effects, on the same day or subsequent days, adjusting for climate and other factors.

The APHEA-2 mortality study (Air Pollution and Health: a European Approach) covered a population of more than forty three million people living in European cities studied for more than five years in the 1990's. The all-cause daily mortality increased by 0.6% (95% CI 0.4-0.8) for each 10µgm^{-3} increase in PM_{10}. (Katsouyanni K et al, 2001). The National Mortality, Morbidity and Air Pollution Studies (NMMAPS) in the USA investigated 50 million inhabitants of metropolitan areas during 1987-94 and all-cause mortality increased by 0.5% (95% CI 0.1-0.9) for each 10µgm^{-3} increase in PM_{10}. (Samet JM et al, 2000).

In Europe, a World Health Organisation (WHO) task group conducted a meta-analysis of time series studies. (Anderson HR et al, 2005). Estimates of PM_{10} effects on all cause mortality were taken from 33 separate European cities or regions and all cause mortality increased by 0.6% (95% CI 0.4-0.8) for each 10µgm^{-3} increase in PM_{10}. The summary estimates for cardiovascular and respiratory mortality separately were 0.9% (95% CI 0.5-1.3) and 1.3% (95% CI 0.5-2.0) respectively for each 10µgm^{-3} increase in PM_{10}.

The APHEA-2 hospital admission study covered a population of thirty eight million living in eight European cities during the 1990s. Hospital admissions for chronic obstructive lung disease (COPD) and asthma for people of more than sixty five years of age were increased by 1% (95% CI 0.4-1.5) per 10µgm^{-3} increase in PM_{10}, and admissions for cardiovascular disease (CVD) were increased by 0.5% (95% CI 0.2-0.8) per 10ugm^{-3} increase in PM_{10}. (Le Tertre A et al, 2003). In the USA effects on people of more than sixty five years of age who were studied in ten cities were an increase of 1.5% (95% CI 1.0-1.9) on COPD admissions and 1.1% (95% CI 0.9-1.3) on CVD admissions per 10µgm^{-3} increase in PM_{10}. (Zanobetti A, 2000).

It is important to realise that these outcomes are likely to only apply to patients who already have severe, pre-existing disease (e.g. Chronic Obstructive Pulmonary Disease and ischaemic heart disease). In these circumstances the increment in level of an air pollutant acts as the precipitating factor.

APPENDIX 3.4: Detailed Cardiovascular and Respiratory Disease Evidence Base

Two epidemiological studies in the United States have shown long term effects and that those living in less polluted cities live longer than those living in more polluted cities. The measure of particulates employed was $PM_{2.5}$ defined analogously to PM_{10} (i.e. smaller particles). COMEAP considers that this evidence demonstrates that it is more likely than not that a causal association exists between long term exposure to particles and although the results of the US studies may be transferable to the UK, the size of the impact could differ.

COMEAP reviewed these two studies (COMEAP, 2001) and also a reanalysis of their data by the Health Effects Institute. The COMEAP review reported that:

The 'Six Cities; study (Dockery DW et al, 1993) examined the mortality experience of over 8000 adults living in six cities in relation to measurements of air pollution in the cities in which they lived. They were followed up for 14 to 16 years between 1974 and 1991. All cause mortality rates, adjusted for sex, age, smoking, education, occupational exposure and body mass index, were shown to be associated with levels of fine particles (measured from 1979 to 1985) and sulphate (1979 to 1984). The ratio of the adjusted mortality rate in the most polluted city to that in the least polluted city (using fine particles as a measure) was 1.26 (95% confidence intervals 1.08-1.47). Air pollution was positively associated with death from lung cancer and cardiopulmonary disease but not other causes.

The ACS study (Pope CA et al, 1995) made use of a cohort of 552,138 adults living in 151 cities across the United States. The cohort was followed for 7 years from 1982 to 1989. Again, there was an association between all cause mortality (adjusted for age, sex, race, smoking, occupational exposure, education, body mass index and alcohol use) and sulphates (measured in 1980 in all 151 cities) or fine particles (measured from 1979 to 1983 in 50 of the cities). The adjusted relative risk for the most polluted areas compared with the least polluted areas (as indicated by fine particles) was 1.17 (95% confidence intervals 1.09-1.26). Positive associations were found with cardiopulmonary mortality and, for sulphates only, with lung cancer.

The HEI reanalysis (Krewski D, 2000) also looked at whether the associations between fine particles and mortality varied in different subgroups. Those with pre-existing heart or lung disease are one plausible susceptible subgroup but the relative risk was not increased in this group in the ACS study and the increase seen in the six cities study was not statistically significant. Similarly, the increased relative risk in those with reduced FEV1 in the six cities study was not significant. There was also no difference in relative risk between smokers and non-smokers. It seemed that only cardiovascular mortality was affected in both studies. It might be suggested that the apparent lack of an effect on respiratory mortality might be due to misclassification between cardiovascular and respiratory deaths. Schwartz (1994) has shown that cardiac deaths associated with short-term exposure to air pollution more often have respiratory disease as a contributing cause of death. It is unknown whether this is the case for the long-term effects.

The cohort studies (Dockery DW et al, 1993; Pope CA et al, 1995) were unable to take account of different durations of exposure or of the differences in historical levels of air pollution in the cities. Particle levels measured over relatively brief periods (6 years at most (Dockery DW et al, 1993)) were used to represent the pollution experience of cities.

Stratification of age in the ACS study indicated that risks were not reduced in those under 50 as would be expected if more than 40 to 50 years of exposure were needed before the onset of an effect.
The studies examine mortality in relation to spatial rather than temporal differences in pollution so there are additional uncertainties when applying them to assess the impact of changing pollution over time. The mix of pollutants in the air may vary between countries and this may be important for transferability of the results.

The ACS study (Pope CA et al, 1995) represented the $PM_{2.5}$ levels in terms of a single value of long-term average concentration in each of the cities studied. It used a median concentration, based on measurements from 1979-1983, as its index of long-term annual average $PM_{2.5}$. The relative risk for fine particles and mortality in the ACS study (Pope CA et al, 1995) was 1.17.

A multi-disciplinary team co-ordinated by the Institute of Occupational Medicine (IOM) (Hurley JF et al, 2000) has analysed the possible implications of the effects of long-term exposure to particles on the UK population. The IOM report used life-table methods to link the results of the US cohort studies with the age and mortality experience of the UK population. This allowed an estimate of the possible loss of life expectancy due to long-term exposure to fine particles subject to certain assumptions. The calculations are for changes in all-cause mortality in those over 30 (as in the cohort studies).

The results apply to the population of England and Wales alive in 2000 followed to the end of their lives assuming a $1\mu g/m^3$ drop in annual mean $PM_{2.5}$ is maintained for the rest of their lifetime. The baseline assumes current mortality rates remain unchanged (sensitivity analyses have shown that changing this assumption has little effect on the results). In a statement on the long-term effects of particles on mortality (COMEAP 2001) COMEAP also provided a separate rough estimate of the life years gained, if the effect of short-term exposure on mortality is calculated for a $1ugm^{-3}$ drop in annual mean PM_{10} rather than $PM_{2.5}$. This includes the under 30s and assumes an effect on all ages.

The answers range from 0.2 to 4.1 million life years gained over the rest of the lifetime of the population alive today. This could be expressed as 1 day to 1 month per person per $\mu g/m^3$ $PM_{2.5}$ on average but it should be noted that the gains if life expectancy are unlikely to be evenly distributed across the population.

To put this reduction into context, a series of measurements of $PM_{2.5}$ made at 3 urban background sites in London during 2000-2001 gave an average of $18\mu\ gm^{-3}$. A reduction of $1ugm^{-3}$ $PM_{2.5}$ represents around a 5% reduction from this level (COMEAP 2001).

In addition to studies on hospital admissions and mortality the effects of air pollution on respiratory morbidity have also been investigated. Lung function and symptoms of bronchitis in adults was negatively associated with PM_{10} in different communities in Switzerland (Zemp E 1999). Associations were also reported between lung function and symptoms of bronchitis in children and fine particles in children in US and Canadian communities. (Raizenne M. 1996, Dockety D.W. 1996). Also lung function growth has been shown to be affected by exposure to air particulates. (Gauderman W.J. 2000). The evidence base of air pollution and health is very extensive and a review of the major studies is given by Brunekreef and Holgate (Brunekreef B 2002).

APPENDIX 3.4: Detailed Cardiovascular and Respiratory Disease Evidence Base

Health Effects Associated with Nitrogen Dioxide in Ambient Air

Nitrogen dioxide is produced with nitric oxide by motor traffic. Epidemiological studies have tended not to show that nitrogen dioxide contributes much to health effects associated with ambient air pollution (Department of Health, 1999). COMEAP reported on acute and chronic effects of nitrogen dioxide in 1998 (Department of Health, 1998) and commented that:

The effects of NO_2 appear to be dependent on a wide range of modifying influences and are difficult to assess. All cause mortality is increased by approximately 3.5% per 100μgm^{-3} on days with elevated NO_2. There is little evidence that admissions to hospital are increased by increased levels of NO_2 for all respiratory causes but there are apparent increases in admissions for COPD and asthma. A large proportion of all studies on the acute effects of NO_2 emphasize the heterogeneity of response between groups defined either by personal characteristics or by coexistent exposures. COMEAP quotes a risk coefficient for respiratory hospital admissions of +2.5% per 50μgm^{-3} NO_2 increase in 24 hour average NO_2. There is a lack of consistency in the results of studies examining the chronic effects of NO_2 on respiratory symptoms and lung function in children and therefore COMEAP has not yet estimated risk coefficients for these effects.

Since 1998 the APHEA study which relates time-series of routinely collected mortality and hospital admissions to daily air pollution data (Katsouyanni K, 2001) has found that NO_2 is an effect modifier and that the estimated increase in daily mortality associated with a 10μgm^{-3} increase in PM_{10} was 0.8% for a city with high long term average NO_2 concentrations compared to 0.19% for a city with low long term average.

Health Effects Associated with Ozone in Ambient Air

Ozone is created by the action of sunlight on nitrogen dioxide in the presence of volatile organic compounds and ambient concentrations show yearly, seasonal and diurnal variation. Ozone and its precursors may be transported over long distances giving rise to population exposure in both rural and urban areas.

COMEAP concluded that for the purposes of health impact assessment only data for respiratory admissions and all cause mortality should be used and published exposure response coefficients (Department of Health 1998):

Deaths (all causes) and respiratory hospital admissions +3% and +3.5% respectively per 50μgm^{-3} 8 hour mean 03 concentrations.

Physical activity in the prevention of Cardiovascular Disease

The amount and type of physical activity required for the primary prevention of coronary heart disease is not known precisely. There is evidence from a number of studies (Wannamethee SG, 2001) that leisure time physical activity is associated with reduced risk of CHD in middle-aged and older people of both sexes. Perhaps the strongest evidence comes from an ongoing cohort of men in the USA (Sesso HD, 2000) which demonstrates that vigorous activities are associated with a reduced risk of CHD but that moderate or light activities have no clear association with the risk of CHD. Vigorous activities equate to activities such as brisk walking and recreational cycling on most days of the week. Such activity may favourably affect CHD risk even in the presence of other CHD risk factors.

Noise and Cardiovascular Disease in the Community

Concerns have been expressed that long term exposure to community noise may have adverse health affects. There is some evidence that noise is a non-specific stressor that arouses the autonomous nervous system and the endocrine system. (Spreng 2000a, Spreng 200b) and that the arousal of the endocrine and autonomic nervous system in turn affects classical biological risk factors for cardiovascular disease (e.g. blood pressure) (Babisch 2002).

In a community cardiovascular survey in the early 1970s, into cardiovascular health and aircraft noise around Schiphol airport in Amsterdam, over 5,800 people were screened for cardiovascular symptoms using a questionnaire and medical examination (Knipschild, 1977, Knipschild & Oudshoorn 1977). It was found that in areas with "more" aircraft noise a significantly greater number of people were receiving medical treatment for heart trouble ($p<0.05$), were receiving medical treatment for hypertension (high blood pressure – $p<0.001$) or were taking cardiovascular drugs ($p<0.01$). These changes could not be explained by potential confounding factors such as age, sex, smoking habits and so on. This work has been criticised, however, because of the poor response rate (of the people contacted only 42% took part in the survey) (Green et al , 1982). It was also not possible to control for socio-economic factors, and socio-economic status was lower in the group exposed to the greatest aircraft noise (Knipschild, 1977).

In a study on GP contacts (Knipschild, 1977a), total contact rate was found to be significantly higher ($p<0.001$) in the group exposed to the greatest aircraft noise. As with the cardiovascular screening survey socio-economic variables were not accounted for, nor were other confounding factors, such as the prevalence of smoking, considered.

In a study on school children, (Cohen et al, 1980) found that children attending a school near the Los Angeles International airport (mean peak classroom noise: 74 dB) were more likely to have higher systolic and diastolic blood pressure than children from "quiet" schools (mean peak noise: 56 dB). Socio-economic factors, race, mobility etc. were taken into account. Living in a quiet home but attending a noisy school was not found to affect the results. A longitudinal study (where participants are followed through time), conducted 1 year after the original study failed to confirm these findings (Cohen et al, 1981). The authors attribute this to the high drop out rate, with a large proportion of the children from the noisy school not being re-tested. Although the original study did demonstrate a statistically significant difference in blood pressure readings between the two groups of children, the readings from the high-noise exposed children were still within the normal range (Voors et al, 1976).

APPENDIX 3.4: Detailed Cardiovascular and Respiratory Disease Evidence Base

In Slovak Republic, children between the ages of 3 – 7, attending kindergartens subject to high traffic noise (>60 dBA) have been shown to have higher mean systolic and diastolic blood pressure and lower mean heart rate than children attending quiet kindergartens (<60 dBA). The location of the kindergarten in relation to noise exposure was found to be more important than home noise exposure estimates (Regecova, Kellerova, 1995). In this study account was taken of confounding factors and statistical analysis showed that noise was the most important variable ($p<0.001$).

In case-control studies undertaken in Berlin relative risks greater that 1 for the incidence of myocardial infarction were found only in men who lived adjacent to streets where daytime noise levels exceeded 70 dB(A) outdoors (Babisch W et al 1994). However assessment of cardiovascular risk associated with prolonged exposure to traffic noise at home through a 10 year follow-up cohort study of middle-aged men in Caerphilly and Bristol found no statistically significant noise effects (Babisch W. et al 1999). More recently, meta analyses of case-control and cohort data from the epidemiological studies in Caerphilly and Speedwell in the United Kingdom (Babisch 2003) and from Berlin (Babisch 2005) have been undertaken on the relationship between road traffic noise level and the incidence of myocardial infarction (Babisch 2006). A dose response relationship was observed when the incidence of myocardial infarction was compared by exposure category i.e. <60 dBA, 61-65 dBA, 66-70 dBA, 71-75 dBA and 76-80 dBA but none of the exposure category estimates achieved statistical significance.

The epidemiological evidence for association between noise exposure and cardiovascular disease is not strong as indicated by reviews (Babisch 2000, Kempen 2002, Passchier-Vermeer et al 2000).

RESPIRATORY DISEASE ASSOCIATED WITH FLOODING

Dales et al. (1991a and b) examined the effects on health in adults and children (between the ages of 5 and 8) exposed to home dampness and moulds (defined as damp spots, visible mould or mildew, water damage and flooding). In adults, lower respiratory symptoms (any cough, phlegm, wheeze or wheeze with dyspnoea – laboured or difficult breathing) were reported more frequently in those living in homes with dampness or mould (Dales et al., 1991a). The relationship remained after adjusting for a number of socio-demographic and exposure variables (adjusted odds ratio [OR] 1.62; 95% confidence interval [CI] 1.48 – 1.78). In children, the prevalence of all respiratory symptoms was higher in homes with reported dampness or moulds. The adjusted odds ratios ranged from 1.32 (95% CI 1.06 – 1.39) for bronchitis to 1.89 (95% CI 1.58 – 2.26) for cough (Dales et al., 1991b). Although these studies were conducted as questionnaire surveys and no fungal spore measurements were taken, previous studies have suggested that respondents' perceptions of home dampness correlate with measured spore concentrations (von Wageningen et al., 1987) and that where a 'water problem' existing for more than three days this was associated with an increase in measured spore levels (Gallup et al., 1987).

Research conducted in the UK by the Flood Hazard Research Centre (FHRC), has suggested that respondents at focus group meetings following flooding in the northeast of England (June 2000) were likely to report asthma, chest infections, coughs, colds and flu believed to be due to the flooding (Tapsell et al., 2002), although the specific symptoms reported vary by the area studied. In a Defra/EA study (Defra/EA, 2003) respondents were asked to report on physical health effects following flooding. Respiratory symptoms (namely colds, cough, sore throats and flu) were reported by 20% of the respondents. Reacher et al. (2004), in a study of the health impacts experienced as a result of flooding in Lewes found a significant association between flooding and self-reported worsening of asthma in adults (relative risk [RR] 3.1; 95% CI 1.2 – 4.4) but not for respiratory illness in children or adults.

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APPENDIX 3.5: Detailed Winter (cold) and Summer (heat) mortality Evidence Base

Heat and Cold Related Mortality and Morbidity

Synopsis of the Evidence

Urban areas generate a 'heat island' effect and London can be up to 8% warmer than rural areas and night temperatures in the city can remain above 19°C. Mortality increases in hot weather and elderly people are particularly vulnerable; in the 1995 heatwave in London there was a 16% excess in deaths for all ages and those aged over 85 had a 20% excess mortality. Climate change will further exacerbate this problem.

Measures can be incorporated into the layout of a development to reduce the heat island effect.

In England approximately a third of excess deaths in winter (18 excess deaths per 100,000 adults) are related to low indoor temperatures and 90% of these occur in those more than 65 years of age. Poor home insulation and fuel poverty contribute to the problem.

Measures can be incorporated into building design to improve insulation.