APPENDICES

PROOF OF EVIDENCE

HEALTH

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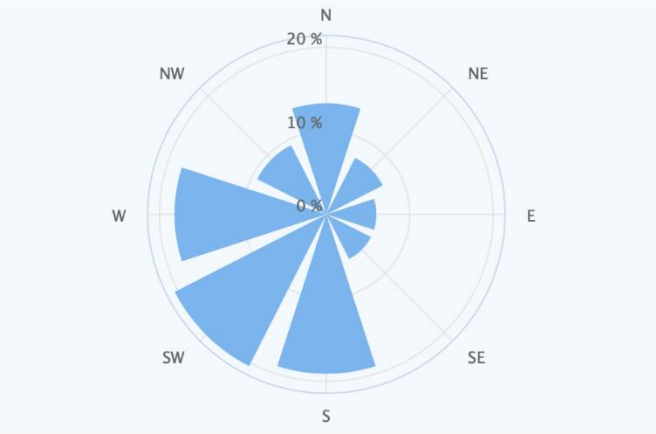
Appendix 1

Appendix 1

Wind rose for Didcot from *World-weather information United Kingdom / Didcot*[1]. accessed 28.1.2024.

This demonstrates that the prevailing winds are from the W, SW and S. This will mean that both air pollution and noise will be exacerbated in Appleford which is downwind of these prevailing directions.



[1] Hyperlink disabled~https://world-weather.info/archive/united_kingdom/didcot/

Appendix 2

Appendix 2

Reference regarding noise pollution

Ref. *European Environment Agency*[2] accesed 28.1.2024

Noise

Noise pollution is a major environmental health concern in Europe. It is caused by noise coming from a variety of sources and is widely present not only in the busiest urban environments but increasingly in once natural environments. The adverse effects on those exposed to noise pollution include threats to the well-being of human populations, the deteriorating health and distribution of wildlife on land and in the sea, the decreased abilities of our children to learn properly at school and the high economic price society must pay as a result.

The World Health Organization (WHO) has classified traffic noise, including road, rail and air traffic, as the second most important cause of ill health in Western Europe, behind only air pollution caused by very fine particulate matter[1,2].

Prolonged exposure to environmental noise can lead to negative cardiovascular and metabolic effects, reduced cognitive performance in children as well as severe annoyance and sleep disturbance[3]. Long-term exposure to environmental noise is estimated to cause 12.000 premature deaths and to contribute to 48.000 new cases of ischemic heart disease per year in the European territory. It is estimated that 22 million people suffer chronic high annoyance and 6.5 million people suffer chronic high sleep disturbance. As a result of aircraft noise, 12500 schoolchildren are estimated to suffer learning impairment in school.

EU Policy

Emissions of noise at source have been regulated in the EU for many years. Maximum noise limits for motor vehicles, household appliances and outdoor equipment date back to the 1970s.

[2] hyperlink disabled~www.eea.europa.eu/themes/human/noise/noise-2/

More recently, measures to control noise from operations and airports, and the regulation of industrial facilities' noise levels have broadened the control of environmental noise.

The introduction of the *Environmental Noise Directive*[3] (END) in 2002 sought to monitor the effectiveness of EU emission controls by requiring the assessment of environmental noise at Member State level. The Directive introduced two key indicators for annoyance and sleep disturbance, which, if exceeded, require action plans to be drawn up that are designed to reduce exposure and protect areas not yet polluted by noise.

WHO recommends that long-term exposure to noise from road traffic should not exceed 53dB during the day-evening-night period and 45dB during the night to avoid adverse consequences on health. The recommended values for rail are 54dB during the day-evening-night period and 44dB during the night, and for aircraft they are 45dB during the day-evening-night period and 40dB during the night.

Given the negative impacts on human health and the large number of people affected, environmental noise is therefore a significant concern for citizens and policy makers. Reducing environmental noise is a key objective under the Zero Pollution ambition and the Environmental Noise Directive (END). One of the headline targets of the zero pollution action plan is to reduce the number of people chronically disturbed by transport noise by 30% by 2030 compared with 2017.

EEA Activities

Noise emissions is a priority work area in the mandate of the EEA. Most of the current activities relate to data reporting and assessment as required by the END.

The EEA devised an electronic noise data reporting mechanism in 2007 in order to facilitate the reporting of noise data in line with the principles set out in the Shared Environmental Information System for Europe and in accordance with the specifications for a spatial data infrastructure for Europe. Recently a new *noise data reporting mechanism*[4] has been implemented following the new END reporting requirements outlined in the Commission Implementing Decision (EU) 2021/1967 of 11 November 2021.

In assisting with the implementation of the END, the EEA works closely with the European Topic Centre on Human health and the environment and with EEA's country network, Eionet.

[3] Hyperlink disabled~Https://environment.ec.europa.eu/topics/noise/environmental-noise-directive_en
[4] Hyperlink disabled~https://www.eionet.europa.eu/reportnet/docs/noise/

Data on noise exposure and associated action plans reported in accordance with the END are used to make regular assessments of environmental *Noise in Europe*[5].

The same data are available for download via the *EEA data service* [6] and can be viewed interactively on the EEA *Noise Observation and Information Service for Europe* [7], which also presents noise contour maps for major sources and cities in Europe.

Country specific fact sheets [8] illustrating END data and estimating the likely health impacts due to noise in EEA member countries have also been published.

The EEA has also used END data to make an assessment of the likely extent of areas yet to be affected by noise pollution in a report entitled *Quiet Areas in Europe* [9]

The EEA published an *Outlook to 2030* [10] to explore the feasibility of reaching the noise objective outlined in the EC zero pollution ambition.

The EEA develops a number of integrated and cross-cutting environmental assessments. *The viewer on combined health impacts from road traffic noise and air pollution* [11] in urban areas presents information on the areas that are better or worse in terms of these two pollutants.

References

1. Hänninen, O., et al., 2014, 'Environmental Burden of Disease in Europe: Assessing Nine Risk Factors in Six Countries', Environmental Health Perspectives 122(5), pp. 439-446 (DOI: 10.1289/ehp.1206154).

2. WHO and JRC, 2011 *World Health Organization, Geneva, Switzerland* [12] accessed 5 May 2014.

3. WHO, 2018, *WHO environmental noise guidelines for the European region* [13], World Health Organization, Regional Office for Europe, Copenhagen accessed 7 December 2018.

[5] Hyperlink disabled~https://www.eea.europa.eu/publications/environmental-noise-in-europe
[6] Hyperlink disabled~https://www.eea.europa.eu/ds_resolveuid/DAT-152-en
[7] Hyperlink disabled~http://noise.eionet.europa.eu/
[8] Hyperlink disabled~https://www.eea.europa.eu/themes/human/copy_of_noise-fact-sheets
[9] Hyperlink disabled~https://www.eea.europa.eu/publications/quiet-areas-in-europe
[10] Hyperlink disabled~https://www.eea.europa.eu/publications/outlook-to-2030/outlook-to-2030-can-the
[11] Hyperlink disabled~https://www.eea.europa.eu/themes/human/noise/viewer-on-combined-health-impacts
[12] Hyperlink disabled~http://www.who.int/quantifying_ehimpacts/publications/e94888/en/
[13] Hyperlink Disabled~http://www.euro.who.int/en/health-topics/environment-and-health/noise/publications/2018/environmental-noise-guidelines-for-the-european-region-2018

Appendix 3

Appendix 3

Reference relating to air pollution

WHO Regional Office for Europe. Review of evidence on health aspects of air pollution – REVIHAAP Project: Technical Report [Internet]. Copenhagen: WHO Regional Office for Europe; 2013. C, Proximity to roads, NO2, other air pollutants and their mixtures. Available from: https://www.ncbi.nlm.nih.gov/books/NBK361807/

C Proximity to roads, NO_2, other air pollutants and their mixtures

Question C1

There is evidence of increased health effects linked to proximity to roads. What evidence is available that specific air pollutants or mixtures are responsible for such increases, taking into account co-exposures such as noise?

Answer

Motor vehicles are a significant source of urban air pollution. Adverse effects on health due to proximity to roads were observed after adjusting for socioeconomic status and after adjusting for noise. Elevated health risks associated with living in close proximity to roads is unlikely to be explained by $PM_{2.5}$ mass since this is only slightly elevated near roads. In contrast, levels of such pollutants as ultrafine particles, carbon monoxide, NO_2, black carbon, polycyclic aromatic hydrocarbons, and some metals are more elevated near roads. Individually or in combination, these are likely to be responsible for the observed adverse effects on health. Current available evidence does not allow discernment of the pollutants or pollutant combinations that are related to different health outcomes, although association with tailpipe primary PM is identified increasingly.

Exhaust emissions are an important source of traffic-related pollution, and several epidemiological and toxicological studies have linked such emissions to adverse effects on health. Road abrasion, tyre wear and brake wear are non-exhaust traffic emissions that become relatively more important with progressive reductions in exhaust emissions. Toxicological research increasingly indicates that such non-exhaust pollutants could be responsible for some of the observed adverse effects on health.

Rationale

In 2010, the Health Effects Institute published their authoritative report *Traffic-related air pollution: a critical review of the literature on emissions, exposure, and health effects*, which formed the basis of the current assessment. Motor vehicles emit large quantities of carbon dioxide, carbon monoxide, hydrocarbons, nitrogen oxides, PM, and substances known as

mobile source air toxics, such as benzene, formaldehyde, acetaldehyde, 1,3-butadiene and lead (where leaded gasoline is still in use). Furthermore, secondary by-products, such as ozone and secondary aerosols (for example, nitrates and inorganic and organic acids), are formed farther away from roads, but these are not considered here.

Pollutant emissions from vehicles are related to vehicle type (such as light- or heavy-duty vehicles) and age, operating and maintenance conditions, exhaust treatment, type and quality of fuel, wear of parts (such as tyres and brakes), and engine lubricants used. Important non-combustion PM emissions associated with motor vehicles include wear particles from road surfaces, tyres and brakes, as well as resuspended road dust.

Non-combustion emissions contain such chemical compounds as trace metals and organics. Traffic emissions are the principal source of intra-urban variation in the concentrations of air pollutants in many cities, but this can vary both by time and location.

The Health Effects Institute report summarized that measurements of outdoor air quality on roadways indicate that concentrations of ultrafine particles, black carbon, particle-bound polycyclic aromatic hydrocarbons, nitric oxide, NO_2, carbon monoxide, benzene, and formaldehyde are high and variable compared with ambient concentrations measured at background locations. Furthermore, concentrations around roadways may represent direct influences from road traffic and from background concentrations. The concentration gradient also seems to be a function of the reactivity of specific pollutants, such as NO_2, nitrogen oxides and ozone. Hitchins et al. (2000) reported a 50% decrease in $PM_{2.5}$ and ultrafine particles within 100–150 m of a road. A decay to background concentrations within as little as 50 m has been described for $PM_{2.5}$ mass concentration (Tiitta et al., 2002), although $PM_{2.5}$ tends to be more spatially homogeneous than ultrafine particles. Roorda-Knape et al. (1998) found that concentrations of black smoke, $PM_{2.5}$, NO_2, and benzene decreased to background concentrations within 100–150 m of a roadway (Roorda-Knape et al., 1998).

In an environment with greater volumes of traffic, Zhu et al. (2002) found that ultrafine particles, black carbon, and total PM counts decreased rapidly in the first 150 m and then levelled off. $PM_{2.5}$ was found to be elevated only modestly (that is, in the range of 20%) near roadways. Zhu et al. (2006) suggested that distance-decay gradients extend to at least 500 m on the downwind side during night-time hours. Some studies concurrently measured such pollutants as NO_2 and volatile organic compounds (Roorda-Knape et al., 1998; Weisel et al., 2005) and carbon monoxide (Zhu et al., 2002; Zhang et al., 2005), to assess pollutant mix. Zhu et al. (2002) found that the decay of concentrations with distance on the downwind side of a highway was similar for ultrafine particles, black carbon and carbon monoxide – that is, a 60% to 80% decrease from roadside concentrations within 100 m. Gilbert et al. (2003) also found that NO_2 concentrations decayed with distance around a busy highway in Montreal, the greatest decrease occurring within the first 200 m.

In general, distance-decay gradients have different characteristics on upwind and downwind sides of an expressway (Roorda-Knape et al., 1998; Zhu et al., 2002; Gilbert et al., 2003; McConnell et al., 2006b). On the upwind side, concentrations drop off to near background levels within 200 m and, in the case of particles, probably within 100 m or less. On the downwind side, concentrations do not generally reach background levels until 300–500 m. In some studies, this was extended to up to 1500 m for NO_2 (Gilbert et al., 2003; Jerrett et al., 2007) and 800 m for ultrafine particle number counts (Reponen et al., 2003).

Zhou & Levy (2007) pooled estimates from more than 30 studies and characterized the decay with distance from the road source for various combinations of reactive and nonreactive pollutants in areas of either high or low background pollution. Further simulations, using dispersion models, were employed to augment the empirical results. Overall, the distance-decay gradients demonstrated a heterogeneity that could be explained by background concentrations, pollutant characteristics, and local meteorological conditions (such as wind speed). Based on dispersion simulations for elemental carbon, the distance-decay gradient was in the range of 100–400 m from the source. For ultrafine particle counts, the gradient was 100–300 m; NO_2 had gradients of 200–500 m. Also, metals (Peachey et al., 2009) and polycyclic aromatic hydrocarbons (Schnelle-Kreis et al., 1999) have shown a distance-decay gradient for roads. While this chapter was being prepared, Karner, Eisinger & Niemeier (2010) published a systematic compilation of the proximity measurements of multiple pollutants classified by category, which is a useful addition to this discussion.

In conclusion, there are a number studies showing higher levels of pollutants in proximity to roads. In general $PM_{2.5}$ does not exhibit the sharp distance-decay gradient evident for carbon monoxide, NO_2 or ultrafine particles. The Health Effects Institute Panel identified an exposure zone within a range of up to 300–500 m from a highway or a major road as the area most highly affected by traffic emissions – the range reflecting the variable influence of background pollution concentrations, meteorological conditions, and season. Metals usually attributed to brake and tyre wear, with such metals as copper, iron, antimony, tin, barium and zinc being higher close to roadways, compared with urban background (Querol et al., 2007). These metals were previously only seen in industrialized areas (Lee, Garland & Fox, 1994). Importantly, Ostro et al. (2011) found association between $PM_{2.5}$ road dust and mortality.

Many studies have shown excess health risks in proximity to roads – after adjustment for a range of possible confounders, including socioeconomic status – for such outcomes as: cardiovascular mortality (Gehring et al., 2006), respiratory mortality and traffic intensity in a 100-m buffer (Beelen et al., 2008a), myocardial infarction (Tonne et al., 2007), cardiovascular disease (Hoffmann et al., 2006), coronary artery calcification (Hoffmann et al., 2007), cardiac function-left ventricular mass index (van Hee et al., 2009), asthma (Morgenstern et al., 2007, 2008; Gauderman et al., 2005; McConnell et al., 2006a; Gordian, Haneuse & Wakefield, 2006; Kim et al., 2008), wheeze (McConnell et al., 2006a; Ryan et al., 2005; Venn et al., 2005; Gauderman et al., 2005; van Vliet et al., 1997), asthma hospitalization (Edwards, Walters & Griffiths, 1994; English et al., 1999; Lin et al., 2002; Wilhelm et al., 2008), lung function reduction (Sekine et al., 2004; Kan et al., 2007; Gauderman et al., 2007; Schikowski et al., 2007), birth weight (Brauer et al., 2008), childhood cancer (Savitz & Feingold, 1989; Pearson, Wachtel & Ebi, 2000), and lung cancer (Beelen et al., 2008b). Therefore, the observed excess risk in proximity to roads cannot solely be explained by socioeconomic status; although associations between traffic proximity and health impacts have been observed in locations where both high and low socioeconomic status occur in close proximity to roads (Généreux et al., 2008), its influence cannot be ruled out.

Some studies have examined the effects of air pollution and noise at the same time. Those who have done so found that excess risks of air pollution in the proximity of roads generally remained after adjustment for noise for cardiovascular mortality (Beelen et al., 2008a; Gan et al., 2012), hypertension and diabetes mellitus (Coogan et al., 2012), hypertension (Fuks et al., 2011; Sørensen et al., 2012), and cognitive performance of primary schoolchildren (van Kempen et al., 2012). Therefore, these studies show effects of air pollution that cannot be explained by noise.

Generally, few epidemiological studies have examined the health effects of multiple air pollutants in proximity to roads. For those studies that have examined multiple air pollutants, it is not clear whether or not these pollutants are coming solely from roads and/or traffic or not. Some studies have examined the effect of multiple pollutants in the proximity of roads, but their small number, the generally high correlation among different pollutants, and the inconsistent results do not provide a good basis to draw firm conclusions.

The only epidemiological study identified that evaluates the short-term effects of multiple air pollutants in proximity to roads and farther away is by Roemer & van Wijnen (2001). These investigators obtained data from a sample of Amsterdam residents (n = 4352) who lived "along roads with more than 10,000 motorized vehicles per day" (actual distance from the roads not specified) from 1987 to 1998, and these were compared with the general population. Ambient-pollutant data from "traffic-influenced" sites and "non-influenced" sites (criteria not specified) were obtained for black smoke, PM_{10}, and gaseous pollutants (carbon monoxide, NO_2, SO_2 and ozone). They found higher levels of NO_2, nitric oxide, carbon monoxide and black smoke at the traffic-influenced measurement sites compared with the background sites, confirming combustion engines as the source of these air pollutants. Black smoke and NO_2 were associated with mortality (RR: 1.38 and 1.10, respectively, for an increase of 100 µg/m^3 on the previous day). Effect estimates were larger in the summer and in the population living along busy roads. Only 10% of the total Amsterdam population resides along busy roads. Nevertheless, they were still able to show associations between black smoke, NO_2, and daily mortality for this subpopulation. These associations were stronger than they were in the total population.

Other studies have examined the effects of multiple pollutants in the proximity of roads for respiratory health and allergic disease outcomes (Brunekreef et al., 1997; van Vliet et al., 1997; Nicolai et al., 2003; Kim J et al., 2004; Gauderman et al., 2005; Morgenstern et al., 2008; Rosenlund et al., 2009a; McConnell et al., 2010; Gehring et al., 2010; Clark et al., 2010; Gruzieva et al., 2012, 2013; Schultz et al., 2012; Willers et al., 2013), birth weight (Brauer et al., 2008), pre-eclampsia and preterm birth (Wu et al., 2011), fatal myocardial infarction (Rosenlund et al., 2006, 2009b), lung cancer (Nyberg et al., 2000) and mortality (Beelen et al., 2008a). However in their analyses these investigators used either a *proximity to road* measure or a specific pollutant(s), but never the effects of (multiple) pollutants within proximity to roads. Even so, and assuming that often the population may have been near roads, no consistent picture emerged that specific pollutants and/or a mixture may be responsible for the observed health effects.

COMEAP recently concluded that the epidemiological evidence for associations between ambient levels of air pollutants and asthma prevalence at a whole community level was unconvincing; a meta-analysis confirmed a lack of association (Gowers et al., 2012). In contrast, a meta-analysis of cohort studies found an association between asthma incidence and within-community variations in air pollution (largely traffic dominated). Similarly, a systematic review suggested an association between asthma prevalence and exposure to traffic, although only in those living very close to heavily trafficked roads carrying many trucks, suggesting a possible role for diesel exhaust.

A critical review of the literature on the health effects of traffic-related air pollution (HEI, 2010b) included toxicological evidence of the impact of traffic-mixture exposures. Such evidence stems from controlled exposures of animals in areas of high traffic density, real-world exposure design in which subjects spend time in a polluted location (compared with equivalent activities in a location with relatively clean air), and individuals occupationally exposed to traffic and populations (animals or human beings) naturally exposed to polluted

urban environments.[2] Of the small number of studies reported (compared with the much larger literature on specific components of traffic emissions), the main cardiorespiratory findings in humans were that short-term exposures can bring about decrements in lung function and enhanced responses to allergens in adult subjects with asthma (Svartengren et al., 2000; McCreanor et al., 2007), as well as positive and negative effects on vascular function in healthy subjects (Rundell et al., 2007; Bräuner et al., 2008). On-road animal studies, utilizing compromised or allergic rodents, observed mild pulmonary inflammation (Elder et al., 2004), significant alterations in lung structure and elastic properties (Mauad et al., 2008), and systemic inflammation and effects on vascular function and autonomic control of the heart (Elder et al., 2004, 2007). Effects on reproductive and neurological health – specifically, compromised sperm quality in toll booth employees (de Rosa et al., 2003) and neuropathological lesions in dogs exposed to high concentrations of ambient pollution in Mexico City (Calderón-Garcidueñas et al., 2002) – were interpreted with caution as a result of data limitations. Finally, observations of genotoxic effects were limited to one study that reported higher mutagenicity from total suspended particulates in an area with intense moving traffic than in an area with limited traffic (Bronzetti et al., 1997).

In a recent review of the adverse effects on health of black carbon, the WHO Regional Office for Europe (2012) evaluated the toxicological evidence of effects of diesel exhaust in controlled human exposure experiments. It concluded that there are not enough clinical or toxicological studies to allow an evaluation: of the qualitative differences between the health effects of exposure to black carbon or those of exposure to PM mass (for example, different health outcomes); of a quantitative comparison of the strength of the associations; or of (identifying) any distinctive mechanism of black carbon effects. The review of the results of all available toxicological studies suggested that black carbon (measured as elemental carbon) may not be a major directly toxic component of fine PM, but it may operate as a universal carrier of a wide variety of combustion-derived chemical constituents of varying toxicity to sensitive targets in the human body, such as the lungs, the body's major defence cells and, possibly, the systemic blood circulation.

Recent noteworthy toxicological evidence on the effects of traffic-mixture exposures include increased respiratory symptoms, decreased peak expiratory flow and an inflammatory response in the upper airways in mild asthmatic adults exposed for 2 hours in a road tunnel (Larsson et al., 2010). Studies of acute (20 minutes to 2 hours) effects of real-life traffic exposure on healthy volunteers have been unremarkable and are limited to a small increase in the percentage of blood neutrophils (Jacobs et al., 2010), modest effects on peak flow, exhaled nitric oxide and airway resistance (Zuurbier et al., 2011a, b). A study by Strak et al. (2012) was specifically designed to evaluate the contribution of different pollutants. They increased exposure contrasts and reduced correlations among pollutants by exposing healthy volunteers at five different locations, including two traffic sites. Changes in particle number concentrations, NO_2, and nitrogen oxides during five-hour exposures were associated with increased exhaled nitric oxide and impaired lung function. These associations were robust and insensitive to adjustment for other pollutants. PM mass concentration or other PM characteristics, including elemental carbon and trace metals, were not predictive of the observed responses. Results for several other health end-points, including markers of cardiovascular effects, have not yet been published.

Two toxicological studies have investigated acute cardiovascular health effects in volunteers with type 2 diabetes. Passengers on 90–110 minute car rides on a busy road demonstrated a decrease in high-frequency heart rate variability and an increase in the ratio of low-frequency to high-frequency components compared with pre-ride measurements (Laumbach et al.,

2010). Chronic exposure to urban air pollution (in chambers 20 m from a street with heavy traffic in downtown Sao Paulo) exacerbates the susceptibility of low density lipoprotein to oxidation, atherogenesis and vascular remodelling in hyperlipidemic mice (Soares et al., 2009), and in Swiss mice it presents as coronary arteriolar fibrosis and elastosis (Akinaga et al., 2009).

Toxicological reproductive outcomes have been investigated in subjects occupationally exposed to traffic. Findings include abnormal sperm count, mobility and morphology (Guven et al., 2008) and a significantly higher percentage of spermatozoa with damaged chromatin and DNA fragmentation (Calogero et al., 2011) in toll-gate workers. In male traffic policemen, lower free testosterone (Sancini et al., 2011) and higher luteinizing hormone (Tomao et al., 2009) and follicle-stimulating hormone (Tomei et al., 2009) plasma levels were reported. Studies on female traffic police observed significantly higher plasma free testosterone (Tomei et al., 2008) and follicle-stimulating hormone levels during the proliferative phase of the menstrual cycle (Ciarrocca et al., 2011).

Evidence continues to accumulate on the role that oxidative stress has as a mechanism through which traffic-related air pollution causes adverse effects on human health. The validity of urinary excretion of 8-oxo-7,8-dihydro-2-deoxyguanosine (8oxodG) as a biomarker was recently demonstrated in a meta-analysis (Barbato et al., 2010). Oxidative damage to DNA and the formation of bulky adducts are two mechanisms by which traffic-related air pollution could lead to mutagenesis and, ultimately, cause cancer. Bulky DNA adducts have been detected among traffic-exposed workers (Palli et al., 2008) and – together with micronuclei – in cord blood after maternal exposures to traffic-related air pollution, suggesting that transplacental environmental exposures could induce DNA damage in neonates (Pedersen et al., 2009).

Ambient PM – particularly that derived from vehicles – has high oxidative potential (Kelly, 2003), and a clear increment in roadside particulate oxidative potential has been found that appears to be associated with metals arising from engine abrasion (iron, manganese and molybdenum) or brake wear (copper and antimony) (Schauer et al., 2006; Thorpe & Harrison, 2008). The roadside increments of particulate oxidative potential are significant and the metal components identified as determinants of this oxidative activity have established toxicity in human beings (Kelly et al., 2011). These results are potentially important as they highlight the contribution of traffic non-exhaust pollutants that are not regulated currently.

Appendix 4

Appendix 4

Vienna Declaration of 2022

Building forward better by transforming to new, clean, safe, healthy and inclusive mobility and transport

Annex 1 Key facts and figures on transport, health and environment[7]

1. The present annex highlights key facts and figures to provide a solid basis for member States of the United Nations Economic Commission for Europe (ECE) and World Health Organization (WHO) European region to support their efforts in advancing the transport system in their own countries for the better and to accelerate the transformation towards sustainable transport and mobility, building forward based on an "Avoid–Shift–Improve" strategy in mobility and transport policies.
2. Despite the technological progress made, current transport system and mobility patterns remain unsustainable. Traffic is still the source of several challenges in many countries, while mobility and transport play an essential role in our societies and economies. The sector provides access to jobs, education, services, amenities and leisure, while contributing to economic growth, jobs and trade. At the same time, it has a growing impact on the environment and human health.
3. THE PEP builds its objectives, strategies and actions on the latest scientific evidence and data available by analysing and highlighting the current state of mobility- and transport-related environmental and health effects in the region. This information should serve as a starting point for the further transformation of the sector towards zero emissions, health promoting mobility and safe and efficient transport in the decade to come. There is an urgent need for this transformation, as global increases in population, overall welfare and trade are expected to induce growing volumes of transport and mobility.
4. Across the ECE and WHO European region, motorized vehicles continue to play a significant role in transport. Considerable differences exist across the region but also between urban areas, where the share of trips carried out by walking, cycling and in public transport is increasing, and rural areas, where the car is still dominant and, all too often, no multimodal mobility option is provided.
5. Due to the COVID-19 pandemic, contrasting trends can be observed. On the one hand, rapid integration of new digital services may lead to less transport and the modal share of active mobility has increased. On the other hand, public transport has come under pressure and suffered significant decreases in passenger numbers and modal share.
6. Traffic-related air pollution, noise and road traffic accidents significantly contribute to the disease burden in the region, with a disproportionate burden concentrated in certain geographic areas and among less affluent social groups. Cars and related infrastructure such as parking spaces use up a large amount of the already very limited space available in urban areas.
7. Emissions of the main air pollutants have declined in recent decades, resulting in generally improved air quality. However, a large proportion of the European urban population remains exposed to levels of air pollution that exceed WHO Air Quality Guidelines. This makes air pollution the single largest environmental risk

in Europe. For the whole of the European region, WHO estimates that 509,000 premature deaths per year are attributable to ambient air pollution, measured as particulate matter of 2.5 microns or less in aerodynamic diameter ($PM_{2.5}$) in 2016. Another pollutant of concern typically associated with vehicle exhaust emissions is nitrogen dioxide (NO_2). The European Environment Agency estimates that 417,000 premature deaths and over 4.8 million years of life lost are attributable every year to $PM_{2.5}$, while 55,000 premature deaths and 624,000 years of life lost are attributable to NO_2 based on data from 2018 covering 41 countries. Policies to address transportrelated air pollution should focus not only on limiting exhaust emissions, but also on reducing non-exhaust emissions (such as tyre and brake abrasion), which are also a significant cause of air pollution mostly through the production of particles.

8. At least 20 per cent of the inhabitants of the ECE and WHO European region live in areas with road traffic noise levels that are harmful to health. In urban areas in most countries, this figure exceeds 50 per cent.

9. More than 110,000 people are killed on the roads every year in the ECE and WHO European region. On average, this means that one person dies every five minutes. Millions more are seriously injured in road accidents. Road traffic injuries are the number one cause of death globally among young people aged between 5 and 29 years.

10. In addition, road transport is responsible for about a quarter of energy-related greenhouse gas emissions, thus contributing to climate change and global temperature rise.

11. Car dependency, restricted use of public space and lack of safety for cyclists and pedestrians contribute to physical inactivity and to a sedentary lifestyle, which increase the risk of non-communicable diseases and obesity. Physical inactivity is estimated to cause about 1 million deaths each year in the WHO European region alone. Obesity also causes approximately 1 million deaths each year. However, physical activity, for example cycling or walking, has very important health benefits.

12. The external costs of road transport are not reflected in current market prices. The total bill for traffic congestion, pollution and accidents, for example, has been estimated at €502 billion per year for States members of the European Union alone. The benefits of a shift towards more active mobility and public transport arise mainly from increased life expectancy, increased productivity and lower health-care costs r elated to non-communicable diseases. This shows that there is a strong case for investing in and promoting walking and cycling in cities and beyond.

13. Inequalities related to transport and urban sprawl can be found in exposure levels and negative health impacts from air pollution, noise and road safety hazards. Furthermore, the benefits from transport are also unequally distributed. Not all socioeconomic groups have equal access to healthy transportation, public transport networks and recreational or green areas.

14. The conditions and circumstances in which people live determine their state of health and level of physical activity. The settings in which people live (cities, workplaces, schools, etc.) should make healthy choices the easiest ones: active transport (walking and cycling) in this case.

15. Countries differ in economic and sociocultural circumstances, population density, local climate, geography and topography. These differences need to be taken into account when developing tailor-made approaches and solutions for the challenges posed by transport at the regional, national and local levels.

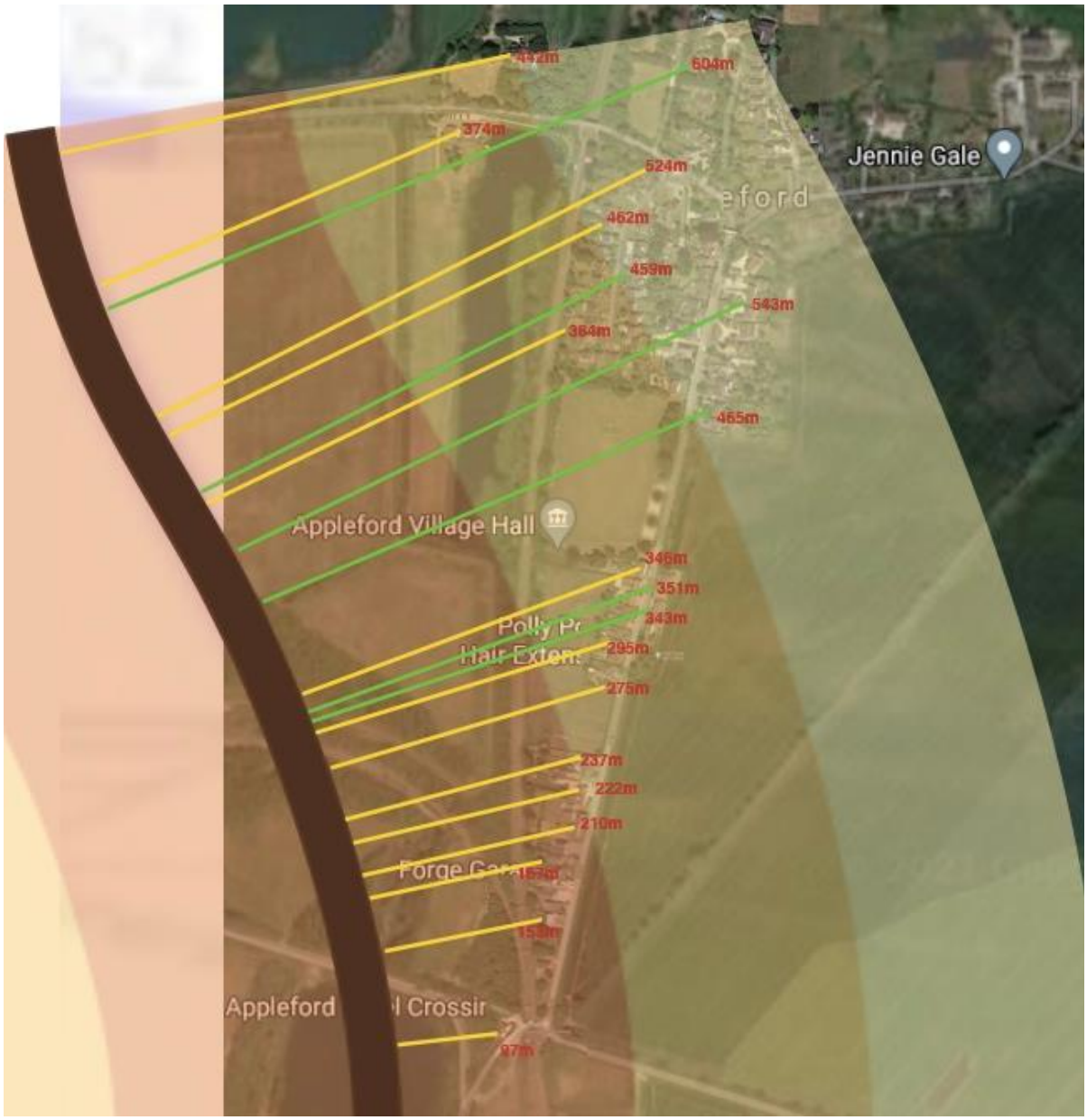
16. To allow for effective monitoring of the impacts of transport, harmonized data on transport, environment and health is crucial. There are significant gaps in data availability and quality, which need to be filled for a better understanding and comparison of data between countries.

17. Transforming the transport and mobility sector requires a multidisciplinary approach. Therefore, collaboration between decision-makers and experts in transport, environment, health, spatial planning and economy is crucial when designing transport-related policies that deliver benefits to environment, health and climate simultaneously. Moreover, international, cross-sectoral and multilevel (countries, regions and cities) cooperation is needed to drive the change to sustainable, environmentally friendly and healthy transport.

Appendix 5

Appendix 5

Distances from proposed road and alternative route to homes in Appleford



These distances are thought by local experts to be somewhat over-estimated.

The alternative route suggested would be 175m further west at the crossing of the sidings which would allow more room for the construction of mitigating barriers such as mounds between the road and dwellings. Potentially most importantly, it would be very significantly further away from the most vulnerable dwellings at and close to the Appleford Crossing (level crossing).

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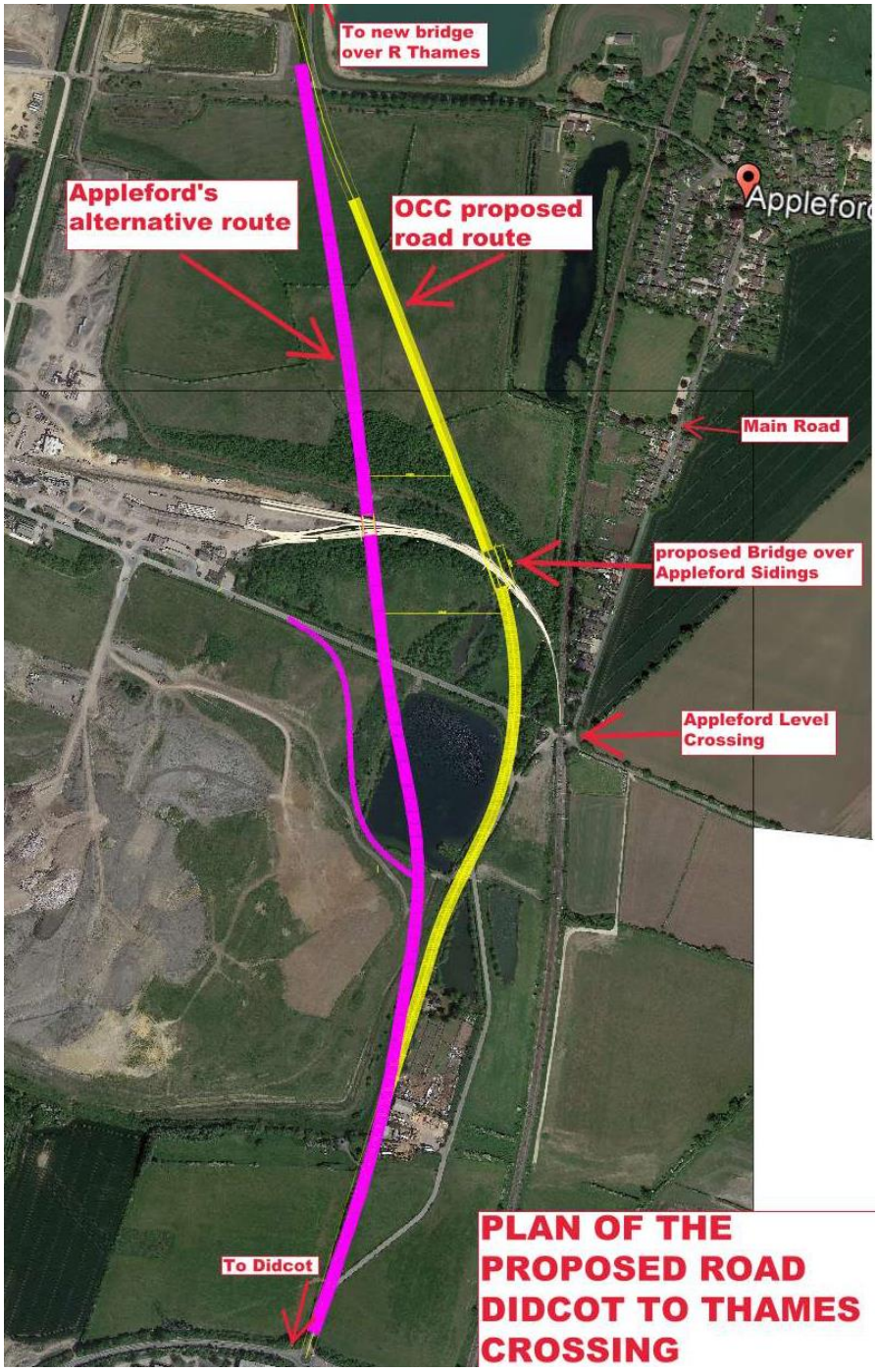

Appendix 6

Appendix 6

Issue of lack of Health Impact Assessment

In his advice regarding this application to Oxfordshire Public Health on 8th Dec 2021, the UK Health Security Agency included this paragraph:

"Reducing public exposures to non-threshold pollutants (such as particulate matter and nitrogen dioxide) below air quality standards has potential public health benefits. UKHSA support approaches which minimise or mitigate public exposure to non-threshold air pollutants, address inequalities (in exposure), and maximise co-benefits (such as physical exercise) and encourage their consideration during the design, environmental and health impact assessment, implementation, and post-implementation monitoring stages."

It is acknowledged that providing a definitive health impact assessment for such a project is not an easy thing to do. However, surely it is the responsibility of the local authority, and by extension of the applicant, to minimise the impact on the health of the local community. It is difficult to see how the applicant has complied with this advice, in particular to minimise public exposure to air pollutants in the design of the route.