Exhibit F
Burden of Disease from Environmental Noise

The health impacts of environmental noise are a growing concern among both the general public and policy-makers in Europe. This publication provides technical support to policy-makers and their advisers in the quantitative risk assessment of environmental noise, using evidence and data available in Europe. It contains the summary of synthesized reviews of evidence on the relationship between environmental noise and specific health effects, including cardiovascular disease, cognitive impairment, sleep disturbance, tinnitus, and annoyance. For each outcome, the environmental burden of disease methodology, based on exposure–response relationship, exposure distribution, background prevalence of disease and disability weights of the outcome, is applied to calculate the burden of disease in terms of disability-adjusted life-years. The results indicate that at least one million healthy life years are lost every year from traffic-related noise in the western part of Europe. Owing to a lack of exposure data in south-east Europe and the newly independent states, it was not possible to estimate the disease burden in the whole of the WHO European Region. The procedure of estimating burdens presented in this publication can be used by international, national and local authorities in prioritizing and planning environmental and public health policies.
The WHO European Centre for Environment and Health, Bonn Office, WHO Regional Office for Europe coordinated the development of this publication.

KEYWORDS

NOISE – ADVERSE EFFECTS
ENVIRONMENTAL EXPOSURE
ENVIRONMENTAL HEALTH
RISK ASSESSMENT
PUBLIC HEALTH
HEALTH STATUS
EUROPE

ISBN: 978 92 890 0229 5

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Schergigsvej 8
DK-2100 Copenhagen Ø, Denmark
Alternatively, complete an online request form for documentation, health information, or for permission to quote or translate, on the Regional Office web site (http://www.euro.who.int/pubrequest).

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Edited by Frank Theakston, layout by Dagmar Bengs, printed by www.warlich.de

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### LIST OF ACRONYMS AND ABBREVIATIONS

<table>
<thead>
<tr>
<th>Acronym</th>
<th>Definition</th>
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<tbody>
<tr>
<td>ADL</td>
<td>Activity of daily life</td>
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<tr>
<td>AF</td>
<td>Attributable fraction</td>
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<td>AR</td>
<td>Attributable risk</td>
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<tr>
<td>CI</td>
<td>Confidence interval</td>
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<tr>
<td>CLAMES</td>
<td>Classification and Measurement System of Functional Health</td>
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<tr>
<td>DALY</td>
<td>Disability-adjusted life year</td>
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<td>DW</td>
<td>Disability weight</td>
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<tr>
<td>EBD</td>
<td>Environmental burden of disease</td>
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<td>EEA</td>
<td>European Environment Agency</td>
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<td>EEG</td>
<td>Electroencephalogram</td>
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<td>EMG</td>
<td>Electromyogram</td>
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<td>END</td>
<td>Environmental noise directive (2002/49/EC)</td>
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<td>EOG</td>
<td>Electrooculogram</td>
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<tr>
<td>ETC LUSI</td>
<td>European Topic Centre on Land Use and Spatial Information</td>
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<td>EU</td>
<td>European Union</td>
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<tr>
<td>EUR-A</td>
<td>WHO epidemiological subregion in Europe: Andorra, Austria, Belgium, Croatia, Cyprus, the Czech Republic, Denmark, Finland, France, Germany, Greece, Iceland, Ireland, Israel, Italy, Luxembourg, Malta, Monaco, the Netherlands, Norway, Portugal, San Marino, Slovenia, Spain, Sweden, Switzerland and the United Kingdom</td>
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<tr>
<td>GBD</td>
<td>Global burden of disease</td>
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<td>HA</td>
<td>Highly annoyed people</td>
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<tr>
<td>HSD</td>
<td>Highly sleep disturbed people</td>
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<tr>
<td>ICD-9</td>
<td>International Statistical Classification of Diseases and Related Health Problems, ninth revision</td>
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<tr>
<td>ICD-10</td>
<td>International Statistical Classification of Diseases and Related Health Problems, tenth revision</td>
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<tr>
<td>$L_{Aeq,th}$ or $L_{eq,th}$</td>
<td>A-weighted equivalent sound pressure level over $t$ hours</td>
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<tr>
<td>$L_{den}$</td>
<td>Day-evening-night equivalent sound level</td>
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<tr>
<td>$L_{dn}$</td>
<td>Day-night equivalent sound level</td>
</tr>
<tr>
<td>$L_{night}$</td>
<td>Night equivalent sound level</td>
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<tr>
<td>NIHL</td>
<td>Noise-induced hearing loss</td>
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<td>NOISE</td>
<td>Noise Observation and Information Service for Europe</td>
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<tr>
<td>NYHA</td>
<td>New York Heart Association</td>
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<tr>
<td>OR</td>
<td>Odds ratio</td>
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<tr>
<td>OSAS</td>
<td>Obstructive sleep apnea syndrome</td>
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<tr>
<td>PAR</td>
<td>Population attributable risk</td>
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<td>PSG</td>
<td>Polysomnography</td>
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<td>REM</td>
<td>Rapid eye movement</td>
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<td>SWS</td>
<td>Slow wave sleep</td>
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<td>WHO</td>
<td>World Health Organization</td>
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<td>YLD</td>
<td>Years lost due to disability</td>
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<td>YLL</td>
<td>Years of life lost</td>
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Summary of evidence linking noise and cardiovascular disease

Epidemiological studies on the relationship between transportation noise (particularly road traffic and aircraft noise) and cardiovascular effects have been carried out on adults and on children, focusing on mean blood pressure, hypertension and ischaemic heart diseases as cardiovascular end-points. The evidence, in general, of a positive association has increased during recent years (18–20). While there is evidence that road traffic noise increases the risk of ischaemic heart disease, including myocardial infarction, there is less evidence for such an association with aircraft noise because of a lack of studies. However, there is increasing evidence that both road traffic noise and aircraft noise increase the risk of hypertension. Very few studies on the cardiovascular effects of other environmental noise sources, including rail traffic, are known. Numerical meta-analyses were carried out assessing exposure–response relationships in quantitative terms (21,22) and the issue has been addressed in various WHO projects. The exposure–response curves presented here refer to the data collected for these projects, to illustrate the processes of a quantitative risk assessment.

Biological model of causation

Non-auditory health effects of noise have been studied in humans and animals for several decades, using laboratory and empirical methods. Biological reaction models have been derived, based on the general stress concept (17,23–30). Noise is a nonspecific stressor that arouses the autonomous nervous system and the endocrine system (9,11–14,31,32) (C. Maschke & K. Hecht, unpublished data, 2005). A neuro-endocrinological definition of stress is that it is a state that threatens homeostatic or adaptable systems in the body (16,33,34). Increased allostatic load is associated with various diseases, including ischaemic heart disease (35). The epidemiological reasoning is based on three facts. First, experimental studies in the laboratory have been carried out for a long time and revealed an increased vegetative and endocrine reactivity during periods of exposure (1,36–70). However, the question regarding long-term effects of chronic noise exposure cannot be answered from short-term experiments. Second, animal studies have shown manifest disorders in species exposed to high levels of noise for a long time (71–83). However, effects in humans and animals cannot be directly compared, particularly because two pathways may be relevant – the direct effect due to nervous innervation and the indirect effect due to the cognitive perception of the sound; the latter is certainly different in humans. Furthermore, noise levels in animal studies were higher than in ambient situations. Third, occupational studies have shown health disorders in workers chronically exposed to noise for many years (20,84–98). However, noise levels were higher than in the ambient environment. Epidemiological research has therefore been carried out with respect to community noise levels to test the hypothesis and to quantify the risk.

Among other non-auditory health end-points, short-term changes in circulation, including blood pressure, heart rate, cardiac output and vasoconstriction, as well as stress hormones (epinephrine, norepinephrine and corticosteroids), have been studied in experimental settings for many years (32,99). Classical biological risk factors have been shown to be elevated in subjects that were exposed to high levels of noise (44,54,79,100–111).
Meta-analysis - road traffic noise and myocardial infarction

To determine the most up-to-date and accurate exposure–response relationship between community noise and myocardial infarction, a meta-analysis was carried out (21,121). By 2005, a total of 61 epidemiological studies had been recognized as having either objectively or subjectively assessed the relationship between transportation noise and myocardial infarction. Nearly all of the studies referred to road traffic noise or (commercial) aircraft noise, and a few to military aircraft noise. Most of the studies were of the cross-sectional type (descriptive studies) but observational studies such as case-control and cohort studies (analytical studies) were also available. The study subjects were children and adults. Confounding factors were not always adequately considered in some older studies. Not many studies provided information on exposure–response relationships, because only two exposure categories were considered.

All epidemiological noise studies were evaluated with respect to their feasibility for inclusion in a meta-analysis. The following criteria for the inclusion in the analysis/synthesis process were applied: (a) peer-reviewed in the international literature; (b) reasonable control of possible confounding (stratification, model adjustment, matching); (c) objective assessment of exposure (sound level); (d) objective assessment of outcome (clinical assessment); (e) type of study (analytical or descriptive); and (f) multi-level exposure–response assessment (not only dichotomous exposure categories).

Based on the above criteria, five analytical (prospective case-control and cohort) and two descriptive (cross-sectional) studies were suitable for derivation of a common exposure–response curve for the association between road traffic noise and the risk of myocardial infarction. Two separate meta-analyses were undertaken by considering the analytical studies and descriptive studies separately. The analytical studies comprised those that were carried out in Caerphilly and Speedwell with a pooled analysis of 6 years follow-up data (122,123) and the three Berlin studies (124,125). The descriptive studies comprised the cross-sectional analyses that were carried out on the studies in Caerphilly and Speedwell (126). All studies referred to the road traffic noise level during the day (L_{day,16h}) and the incidence (analytical studies) or prevalence (descriptive studies) of myocardial infarction as the outcome. The study subjects were men. In all analytical studies the orientation of rooms (moderator of the exposure) was considered for the exposure assessment (at least one bedroom or living room facing the street or not). In all descriptive studies the traffic noise level referred to the nearest facades that were facing the street and did not consider the orientation of rooms/windows (source of exposure misclassification). The individual effect estimates of each study were adjusted for the covariates given in these studies. This means that different sets of covariates were considered in each study. Nevertheless, this pragmatic approach accounts best for possible confounding in each study and provides the most reliable effect estimates derived from each study.

The common set of covariates considered in the descriptive studies were age, sex (males only) social class, body mass index, smoking, family history of ischaemic heart disease, physical activity during leisure time and prevalence of pre-existing diseases. The common set of covariates considered in the analytical studies were
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