NOISE AND HEALTH – OVERVIEW AND RECOMMENDATIONS

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1. INTRODUCTION

In order to make properly informed choices between alternative noise management options, decision makers require good information about the effects of noise. Depending on noise levels, the specific character of the noise, and on the context in which the noise occurs, the possible effects can vary over a wide range. In practice, most decisions are made on the basis of real or reported ***annoyance*** effects, but noise annoyance can be a difficult concept to take into account when carrying out cost-benefit or cost-effectiveness analyses. There are a number of other noise effects which, providing that they could be estimated accurately, might be more easily compared against the costs and inconvenience of noise control. The potential for environmental and community noise to contribute directly to the development of more ***direct health effects*** with clinically measurable outcomes (such as cardiovascular disease, mental illness, and/or hearing loss) and which might be more easily weighed against the relative costs and inconveniences of alternative noise control options and strategies, is therefore a matter of great interest to regulators and standards organisations alike.

This paper is based on two separate reviews carried out by the presenting authors; a) an NPL study carried out for the then DOE in 1998, (*Porter N D, Flindell I H, Berry B F, Health effect-based noise assessment methods: a review and feasibility study, NPL report CMAM16, September 1998*) which found only limited scientific evidence for a number of theoretical health effects, and b) a more recent (unpublished) review carried out for the World Health Organisation European Office and intended to assist the WHO in deciding between future priorities for action. This review preceded a WHO technical meeting held in Bonn on 19-21 September 2002 and which was convened with the main objective of seeking international agreement on exposure-response relationships for each considered effect. This new WHO initiative was conceived with the idea that exposure-response relationships (where available) would be potentially more useful for decision makers than the single guideline values which exist at present (for the current WHO guideline values see; *Berglund B, Lindvall T, Schwela D, and Goh K, Guidelines for Community Noise, WHO, Geneva,*

2000). The Bonn meeting concluded, however, that the scientific data which is presently available remains insufficient to support reliable exposure-response relationships for any noise-health effects other than annoyance (steady-state) and hearing damage risk. It should be noted that (at least in theory) annoyance can be considered as a 'health effect' according to the 1968 WHO definition of good health which is; 'a state of complete physical and mental and social well-being and not merely the absence of disease or infirmity'.

In the UK, there are two policy areas where strategic overview of these issues may be particularly timely. First, local authority environmental health officers tend to be the first point of contact for members of the public making enquiries or registering complaints about environmental and community noise. Environmental health officers are obliged to work within a legal framework which is mainly focussed on public health, rather than on the public or private amenity issues which noise annoyance is mostly related to. The planning system can be used for noise management where new development is concerned, but planning officers will generally call upon their colleagues in environmental health for specialist advice in this area and the same problem regarding existing legislation also applies. Secondly, the recent Environmental Noise Directive (*Directive of the European Parliament and of the Council relating to the assessment and management of environmental noise', European Commission 2000/0194/(COD), Brussels, 18th July 2002*) includes as its third objective *'adoption of action plans….particularly where exposure levels can induce harmful effects on human health*…'. and to this end Member States will be required to assess *'harmful effects on human health*' by means of dose-effect relations (note that the WHO use the alternative term; *'exposure-response relationships*' to mean the same thing) which have yet to be decided or agreed.

Against this background, this paper overviews the potential health effects of noise and then summarises the available scientific evidence for each of these effects. In our opinion, future scientific progress depends on the development of plausible causative models which can be properly tested in experimental contexts with adequate controls and not merely on further statistical analyses of existing cross-sectional field study data. To this end, we outline a theoretical framework model for the effects of noise on health, where inputs, processes, and outcomes are identified separately in a way that might facilitate the further unravelling of underlying cause-effect relationships (that is; potential or theoretical relationships which might then be experimentally tested). Finally the paper draws some conclusions and makes recommendations for future progress in this important topic area.

2. OVERVIEW OF EXISTING EVIDENCE FOR NOISE-INDUCED HEALTH EFFECTS

2001 literature review and consultation

The review carried out for the WHO at the end of 2001 took into account most of the relevant scientific literature published since the 1998 NPL report and also included a short consultation of academic researchers, government research laboratories, environment managers and planners and many other UK 'stakeholders' with an interest in this general topic. The 2001 review concluded that although some published sources imply otherwise, the available scientific evidence remains generally insufficient to support reliable exposure-response relationships for any

noise-health effects other than annoyance (steady-state) and hearing damage risk (although some increases in sleep disturbance indicators have been associated with increases in noise level but the findings do not lend themselves to the development of rigorous exposure-response relationships). Furthermore it concluded that there is some scepticism about a number of other possible or potential effects amongst experts consulted in the UK.

From our consultation we found that the majority of stakeholders considered noise annoyance to be more a matter of public or private amenity rather than a direct 'health effect' in the same way as ischaemic heart disease is commonly understood to be a direct health effect. This suggests that, at least in the UK, cognitive or attitudinal noise effects or outcomes should be clearly differentiated from clinical or medically recognised effects or outcomes. There was some scepticism as to whether there really is sufficient evidence for postulated effects such as delayed learning effects in schoolchildren or for increased rates of clinical hypertension or ischaemic heart disease around airports. Experts were divided over whether aircraft noise really is a more serious problem than other forms of community noise. Noise levels can be relatively high very close to airports, but there are many more people exposed to road traffic noise (and other noise sources) overall. Recommendations for specific topics to be afforded the highest priority for more work included specific mechanisms underlying noise complaints (particularly related to perceived safety issues), biological mechanisms underlying endocrine response, vulnerable groups, intervention studies, individual noise sensitivity, improved measures of exposure, etc.

The following sections give more detail from our work on potential effects, evidence for effects and the scientific issues surrounding existing controversies.

Possible effects

The potential effects of noise include the following:

1. Direct masking effects e.g. speech interference, music appreciation, auditory tasks etc.
2. Cognitive responses e.g. distraction, annoyance, task interference,
3. Acute physiological responses e.g. endocrine and neurophysiological reactions (including transient sleep disturbance)
4. Behavioural responses e.g. adaptations and coping strategies, social disorder, complaints and community action
5. Chronic physiological responses e.g. hypertension, long-term stress reactions,
6. Clinical morbidity e.g. heart disease, hearing loss, psychiatric morbidity, immune system deficiencies, teratogenetic effects.

Evidence for actual effects

Table 1 gives a summary of the apparent strength of evidence for noise-induced health effects as reported in existing literature reviews (see list of references for typical examples). It is interesting to note that not all experts who were consulted in the UK accept that the available scientific evidence really is as convincing as is

claimed in some of these reviews. It is also interesting to note that some of these reviews seem to be merely repeating the findings of previous reviews without offering any more critical appraisal of the underlying factual evidence.

Table 1: Apparent strength of evidence for a causal association between noise exposure and health effect (from references given at end of paper)

Effect	Strength of Evidence for effect	Comments
Annoyance	Sufficient	
Performance by school children	Sufficient	
Sleep (although mainly a night-time effect)	Sufficient	
Ischaemic heart disease	Sufficient	
Hearing loss	Sufficient	Although unlikely at typical levels of community noise exposure
Hypertension	Sufficient/inconclusive	Reviews disagree
Performance	Limited	
Biochemical effects	Limited	
Immune effects	Limited	
Psychiatric disorders	Limited	
Psycho-social well being	Limited	
Birthweight	Limited/inconclusive	
Immune effects	Limited/inconclusive	
Forms of cardiovascular disease	Limited/inconclusive	
Congenital effects	Lack/inadequate	

Incomplete evidence

On the basis of existing research, we know most about effects 1 – direct masking effects and least about effects 6 – clinical morbidity (see list above). As we go from effects 1 to effects 6, possible reasons for this include the following:

- The underlying exposure-response relationships include an increasing number of different variables such as non-acoustic modifying factors and potential confounding factors – thus increasing the difficulty of obtaining definitive findings.
- The underlying mechanisms become increasingly less well understood.
- Research teams require increasingly multidisciplinary skills to be able to make significant progress.
- The underlying prevalence of effects probably decreases anyway (and is therefore harder to detect if it exists at all).

Scientific issues

Our work suggested that existing controversies mostly relate to the following scientific issues:

- Increasingly inconclusive findings for effects from (1) to (6),
- Questions regarding clinical and socio-economic significance of each effect,
- Relative importance of modifying factors in research methodology e.g. cross sectional studies cannot 'prove' cause and effect, differences between laboratory and field studies,
- Individual differences in sensitivity and susceptibility,
- Measurements of noise exposure,
- Combined effects with other stressors,
- Effects of interventions,
- Relative annoyance of aircraft noise versus other sources and understanding of factors giving rise to differences,
- Monetary evaluation of the social costs of noise.

It seems to us that not that enough account is taken of the fact that the amount of energy present in most community noise is far too small to cause any direct damage to the tissues through physical disruption or dissipative effects. To make progress in this area, it may be more appropriate to think of most adverse noise effects as being indirect in the sense that they arise not from physical disruption caused by the energy present in the noise, but rather from some maladaptive way in which the human organism responds the presence of the noise, or even in some cases, to the information contained in the noise. In this way, we can begin to explain the differences between, for example, the beneficial effects of music in willing listeners and the potentially extremely disruptive effects of exactly the same music in unwilling listeners. The differences lie purely in terms of cognitive appraisal of the meanings contained in the musical sounds understood in the context within which the sounds occur.

3. PRELIMINARY MODEL OF EFFECTS

Framework model

Figure 1 presents a framework model for the effects of noise on health, which in this case was developed specifically to illustrate daytime aircraft noise but which is likely to be more generally applicable. The model follows a general rule that inputs lead only to outcomes in terms of moving from left to right in the figure. The inputs to the model are shown in light blue and include the source noise characteristics, the acoustic context (e.g. the residual and background noise environment which may include other sources of noise) in which it is set, and the situational context (e.g. those non acoustic situational factors that can influence people's opinions such as previous history of noise exposure and public relations at the site, other environmental stressors etc.)

These inputs are then processed through underlying physical, physiological and cognitive mechanisms. The first order processes are shown in red. These are divided into physical processes, autonomic processes and cognitive appraisal.

Physical processes may include masking effects caused by background sounds exceeding the sound level of wanted sounds within the relevant time-varying frequency spectra. The location of these physical processes can be considered as taking place anywhere up to the sensitive basilar membrane in the inner ear. Physical processes are unaffected by the situational context and are largely independent of the human listener, except in terms of the basic time-frequency analysis capabilities of the human auditory system.

Autonomic processes relate to the largely subconscious response of the autonomic nervous and endocrine systems. The autonomic nervous system performs basic functions more or less automatically, without conscious intervention of the higher brain centres. The function of the nervous system is to control the actions and reactions of the body and its adjustment to the environment. It also activates the metabolic system (chemical processes in the body) to better deal with any anticipated situation likely to arise. Autonomic reflex responses can be divided into fast acting/short lasting neural reflexes and slow acting/long lasting endocrine responses which release hormones into the body. These include adrenaline (also known as epinephrine which is termed a catecholamine) and cortisol (also known as hydrocortisone which is a steroid hormone).

Cognitive appraisal relates to internal mental processes mediating between the stimulus and response – including recognition of the sound. The inputs to the cognitive appraisal system include all three; the source, acoustic context and situation context. The process is necessary slower than for the fast acting autonomic neural reflexes, but can be faster than slow acting endocrine release.

These first order processes in turn lead to observable acute outcome responses shown in green. There are masking, cognitive and physiological responses.

The *physical processes* of sound interaction can lead to masking of the sound, causing detection and identification problems. Masking can reduce the comprehension of speech, warning signals and auditory cues.

Cognitive responses refer to psychological reactions such as reported annoyance, distraction, and interference with cognitive tasks. These can follow on from autonomic processes mediated through becoming consciously aware of the effects of endocrine activity, or more commonly from cognitive appraisal of the inputs.

Acute physiological responses include endocrine and neurophysiological reactions resulting from the activation of the metabolic system. These are usually as a result of the autonomic processes, but may be moderated by cognitive appraisal such as fear or recognition associated with recognising the character or true nature of the source.

Finally the longer-term outcomes are shown in dark blue, the chronic responses which are divided in the model into possible behavioural, physiological and clinical outcomes.

Behavioural responses may include a whole range of adaptations, development of coping strategies, social disorders, complaints, learning

effects, activity disturbance and community action. These mainly arise from the effects of masking and cognitive responses.

Chronic physiological responses include hypertension, long-term stress reactions, and effects on memory and can result from both cognitive and physiological responses. These effects are not necessarily of sufficient magnitude or importance to be deemed of clinical or socio-economic significance.

Clinical morbidity relates to those outcomes that result in the presence of illness or disease. These may include heart disease, hearing loss, psychiatric morbidity, immune system deficiencies, teratogenetic effects etc. The precise boundaries between the chronic physiological and clinical outcomes may be subject to debate and relies on a classification of clinical or societal significance.

It should be noted that variables that are often shown as confounders or modifying variables in more specific models but are just different input variables in any fully general framework model, and are not therefore shown separately.

In **Figure 1**, the links that represent cause-effect relationships are shown in both red and green. The red lines represent the most commonly researched cause effect chain. The potential effects on learning may be both behavioural (through learning behaviour) and chronic physiological (through possible or theoretical effects on neurone development and memory systems). One of the problems of a number of existing models of these phenomena is that the precise direction of postulated cause-effect relationships is not always clear. In this model, all cause effect arrows flow in one direction only to simplify the development of scientifically testable hypotheses. The model starts from inputs and then proceeds through internal mechanisms which lead to observable acute outcomes that in turn may lead to observable chronic outcomes. The model acknowledges previous models, which may of course be equally plausible within their defined limits of applicability.

Figure 2 adds two further components to the previous figure to show the boundaries of cause-effect mechanisms lying either inside or outside the noise exposed individual; (yellow lines) and the possible effects of intervention shown in green.

Cause effect mechanisms lying outside the person boundary (yellow line) are those which can be explained purely by reference to the known physical masking of wanted sounds by unwanted masking noise (such as direct interference with speech communications). Similarly, cause-effect mechanisms lying inside the person boundary can also be affected by the biological character of the individual and as such may be sensitive to individual differences and susceptibilities and the possible effects of situational factors where the more direct physical mechanisms lying outside the person boundary would not be.

The green arrows show those areas at which intervention could (in theory) affect the process. Intervention options may have direct physical effects on source noise levels, the acoustic context or on various situational factors. There are possible intervention strategies which might have no effect on physical inputs, but which might nevertheless influence individual attitudes and opinions and thereby modify the various cognitive processes and outcomes shown in the model. These strategies can include improved public relations and/or various offsetting actions that make environmental contributions in other areas. It also possible that specific events such as last year's terrorist activity in New York could influence certain cognitive aspects of the model.

Relationship of model to current knowledge

Let us now relate parts of the model to the information in previous sections:

- Annoyance: much is known about reported annoyance as a cognitive response, but very little is known about how this might develop into a chronic behavioural response OR how important reported annoyance really is in an overall quality of life context.
- Performance by school children: there is some evidence regarding cognitive effects but little is known about how this might develop into longer term chronic effects or be compensated for by other mechanisms.
- Hearing loss: this is unlikely to be a problem at typical community noise exposure levels in developed countries, although it could be important elsewhere.
- Heart disease: the aetiology of this clinical outcome is not well understood even if there is sufficient evidence for such an effect (as claimed in some reviews).
- Moving from left to right in the model, the possible causative links are increasingly less well understood.
- There are uncertainties over the clinical and societal importance of the various possible chronic effects.
- The effects of intervention are not fully understood.

4. CONCLUSIONS AND RECOMMENDATIONS

The physical energy in noise is mostly insufficient to cause any direct physical harm to the tissues of the body. Those health effects that exist are most probably related to the consequences of maladaptive responses of the body to noise stimuli. It is unlikely that much further progress will be made in this field without research being designed around hypothetical and testable models. Our chief message is that by developing biologically plausible theoretical models with postulated causative links flowing from inputs through processes to outcomes, we may be able to realistically predict effects from basic information regarding input variables. Because of the large number of different variables which are likely to be involved, (additional to noise levels alone however measured), it seems unlikely that any simple 2 variable exposure-response relationships will be adequate.

Future research can most usefully be addressed to investigating particular links within the overall model. For these we may need more sophisticated exposure-response relationships that make explicit the likely consequences of alternative intervention strategies, and a better understanding of the underlying mechanisms linking cause and effect. Research needs to be targeted. For epidemiological studies, it is necessary to identify the most and least susceptible groups and for laboratory studies, we need to address ethical issues and representativeness of real-life experience.

6. REFERENCES

A large number of references were used for this work, but are not listed here for reasons of space. Useful summaries of the literature can be found in the following references:

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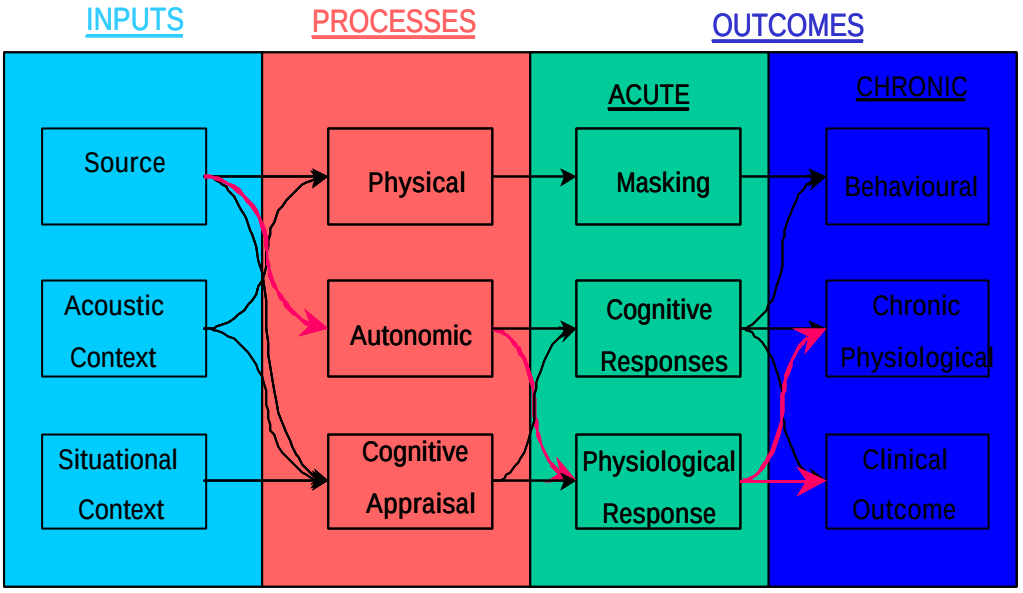

Figure 1: A framework model for the effects of noise on health, applicable to aircraft noise during the daytime

Figure 2: A framework model for the effects of noise on health, applicable to aircraft noise during the daytime showing individual differences and stages of intervention