

## Conceptual differences between experimental and epidemiological approaches to assessing the causal role of noise in health effects

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### ABSTRACT

Prima facie evidence for detrimental physical and mental health effects of noise has been extensively established in cross-sectional research. However, people living in high versus low noise areas may differ in many (confounding) ways: air pollution, age, socio-economic status, education, and lifestyle. A key scientific issue in this field is the causal link (if any) between noise exposure and the health effects. Broadly, there are three approaches to this issue: (1) the direct assertion that the cross-sectional research is sufficient to establish a causal link; (2) the experimental approach; and (3) the epidemiological approach. The experimental approach suggests, at the simplest level, the comparison between otherwise comparable (through matching or random assignment) groups given forced exposure to noise or control conditions. The epidemiological approach suggests measurement and then statistical control of the confounding factors to assess the extent to which noise remains a predictor of health after all confounders are controlled in the statistical model. These approaches differ radically in terms not only of statistical versus methodological control of confounding factors, but also in terms of preparedness to make claims regarding the nature of the exposure variable (e.g., specific noise versus all sounds), and the confounding versus causally linked nature of variables (e.g., is reaction a confounder or a causal link between noise and health?). The success of these approaches depends on the varacity of the assumptions and the strength of their use. Inconsistent results and the failure to resolve the issue of the extent to which noise produces health effects, arise in part from the failure to appreciate the different strengths and weaknesses of these distinct research traditions. This paper recommends that within the limitations of field research, which dictate the epidemiological approach, a key change to this approach is to allow assumptions in relation to underlying causal variables.

### INTRODUCTION

For the purposes of this paper, which addresses different methodological approaches to the issue of the effects of noise on people, it is necessary to initially identify the possible, likely, and established effects of noise. These are hearing loss (see Ward 1993), stress (e.g. Evans et al. 1995; Ising et al. 2004), cognitive impairments (Haines et al. 2001), sleep disturbance (Griefahn et al. 2006; Griefahn & Spreng 2004), community reaction or negative emotional reactions (often restricted to annoyance, but including more: Job 1988; Schulte-Fortkamp & Fiebig 2006; van Kamp et al. 2004), and a variety of physical and mental health effects.

A number of reviews of methodology have been published, considering the evidence for the proposed causal link from noise exposure to health effects. These have drawn divergent conclusions from supporting evidence for the health effects of noise (e.g. Ising & Kruppa 2004; Job 1996) to going further and identifying the noise levels

at which the various health effects occur (e.g. Berglund & Lindvall 1995; Schwela 1999) to challenging the evidence for the effects (e.g. Flindell & Cartwright 1999). The present review does not consider each study in detail, but rather considers the principle differences between the methodologies. Consideration of the core research methodologies (experimental and epidemiological) is of value for two reasons. First, they differ substantially in their strengths and weaknesses. Second, the divergence of opinion on the issue of noise effects often corresponds with the methodological background; with more support generally proposed from the experimental camp, than from the epidemiological camp.

It should be acknowledged that other methodologies have been applied to the research question of the effects of noise (such as case studies: e.g. Feldmann & Pitten 2004). While adding to the weight of evidence, and sometimes providing worthwhile hypotheses for further research, these studies do not compellingly address the issue of causality.

## **EXPERIMENT AND EPIDEMIOLOGICAL METHODOLOGIES**

### **The problem**

Many studies attest to the association (observed correlation) between noise exposure and each of the effects listed in the introduction above. The problem may be seen clearly with a broad example. A number of studies have shown positive correlations between noise exposure and blood pressure. Alternatively, in two group comparison studies people living in noisy areas near busy roads have been shown to be more likely to have high blood pressure than those living in quiet areas. The point of the latter example is only to highlight that the limitations below do not only apply to studies based on statistical correlation (as in the misleadingly simple warning: correlation is not causation). Rather, these limitations apply to all observational studies. An observational study is one in which two or more variables (in this case noise and health) are measured (observed) and the relationship between them is assessed. (In contrast, a manipulation is the hallmark of an experiment. In this case one of the two variables is deliberately manipulated and the other is measured.)

The problem for the simple observational study is that it cannot directly establish a causal connection. A real correlation/association between A and B, could occur for one of five reasons: A causes B (directly or indirectly), or B causes A (directly or indirectly), or some third factor, C, causes both A and B, or some third factor C, causes A and co-occurs with B, or C causes B and co-occurs with A.

For example, the observed association of noise and blood pressure may occur because:

1. Noise (A) causes higher blood pressure (B).
2. Lower socio-economic status or education (C) causes higher blood pressure (B) and in virtue of allowing less choice of living location – a quiet, expensive area is unaffordable– co-occurs with noise.
3. Lower education is associated with more alcohol consumption, more smoking and less medical attention, causing high blood pressure, and lower education is associated with living in higher noise areas.

Numerous other interrelationships may also be hypothesised. The above are just a few examples.

Broadly, there are two ways to attempt to resolve these alternative interpretations: experiment and epidemiological study.

## EXPERIMENTAL SOLUTION

The experimental approach addresses this issue most directly by avoiding the problem of measurement of two variables in the first place. Rather, one variable is deliberately manipulated. The advantage of this approach arises from the removal of the possible role of other variables (C in the above discussion) producing an artifactual relationship between noise and outcome. In order to ensure no potential confounding variable is re-introduced it is necessary to avoid the groups exposed to the manipulated (e.g. high versus low) levels of noise being allowed to differ in other ways. Thus, subjects should be randomly assigned or matched, and other variables controlled. To achieve this level of control typically requires a well controlled environment in a laboratory.

The weaknesses of this approach lie in ecological validity. The key question is – Are the conditions of the laboratory, the level and chronicity of noise, and the measures of outcomes valid representations of real life?

## EPIDEMIOLOGICAL SOLUTION

Among the various features of the epidemiological approach, three stand out as most relevant to the present discussion. First, the appropriate temporal sequence is the *sine qua non* of causality. Thus, the approach also typically requires, at a minimum, that a temporal relationship be identified such that noise exposure precedes the health effects. In practice, this temporal requirement is often ignored. Second, potential confounding features are addressed by being measured and statistically controlled in the analysis of the key relationship under investigation. Third, the simplicity of the approach arises in part from the absence of assumptions about the nature of the underlying causal connection. The second feature represents the strength of this approach. Careful epidemiological analyses reveal critically informative relationships (e.g. between noise exposure, sensitivity, and depression: Stansfeld 1992; see also Babisch 1998, for a methodological commentary).

As an example of how the critical variables can be missed, Meecham and Shaw (1979) compared mortality rates in high and low aircraft noise exposure zones around Los Angeles Airport. Among their findings were increased mortality rates for cirrhosis of the liver and stroke. However, the study failed to control for many possible confounding differences between the populations compared, and Frerichs et al. (1980) found that when the mortality rates were adjusted for age, gender and race the previously reported differences disappeared. It seems, in this study, the supposed effect is clearly explicable in terms of factors other than noise exposure.

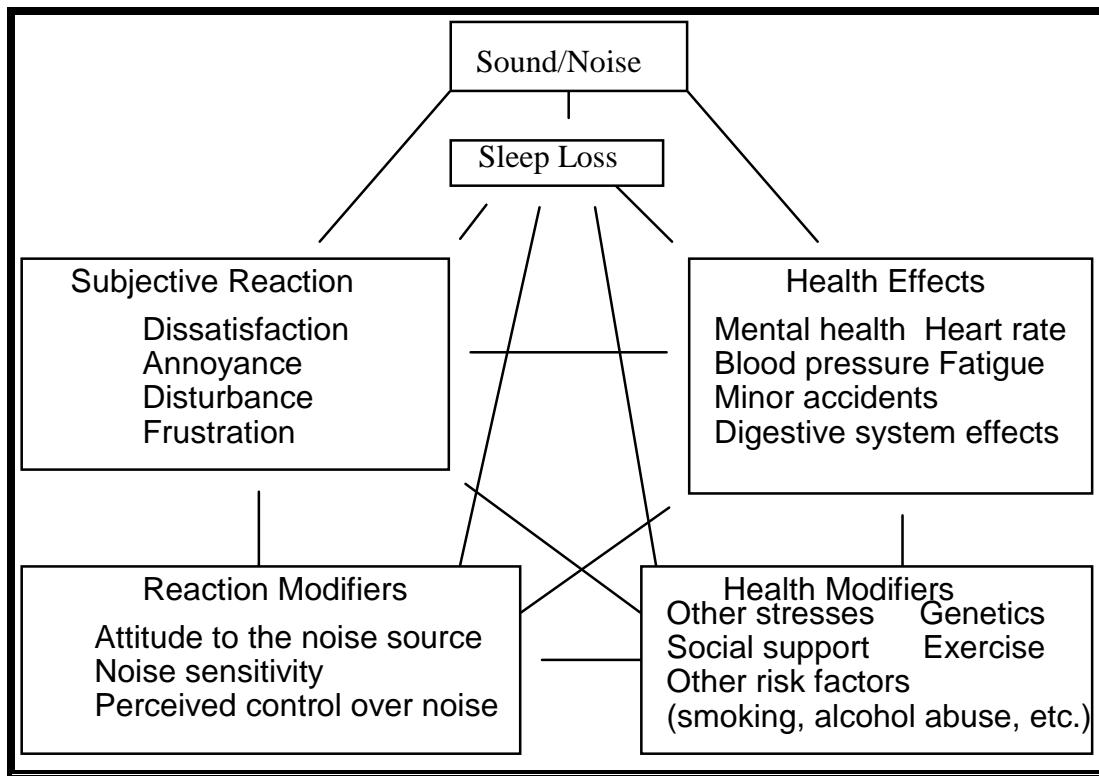
Even this strength is vulnerable: critical related/confounding variables may be poorly measured, not feasible to measure, and/or go unrecognised. In this paper it is argued that the third feature introduces key strengths and key weaknesses to the epidemiological method. The study of the effects of noise draws out the weaknesses.

A key strength of the resistance to making assumptions regarding causal sequences is that the study remains unbiased by the variability of assumptions the researchers may be prepared to make. At the extreme end, this means that in a pure epidemiological study, we would assess the effects of all sound (not noise) on health. Thus, we would measure all sound received by our subjects (regardless of it being noise – unwanted sound or desired sound – music or conversation). We would seek associations between sound exposure and health effects, resisting any assumptions regarding the negative effects of unwanted sound versus wanted sound. Clearly, given the extensive evidence for negative emotional effects of noise (not sounds), such an as-

assumption free approach may miss real noise-health relationships by combining noise and desired sound in the exposure side of the equation. It is acknowledged that this is an extreme end point of epidemiology, and of various statistical methods (path analysis, etc.) if applied purely- without assumptions.

## THE PSYCHOLOGICAL APPROACH

A potential solution to the assumption related limitation of the epidemiological approach is to allow for assumed relationships between variables, based on previous research. For example, the exposure variable must be noise not sound. Numerous other relationships may exist at a psychological level. These possibilities cannot be ignored in interpretation of statistical results. However, in considering the interrelationships of possible effects, myriad possibilities exist, each with at least strong suggestive evidence or logic (see Figure 1, in which each line represents one- or often two with bi-directional causality- of the possibilities).



**Figure 1:** A model of the causal connections between noise, community reaction, modifiers and health effects

### Psychological Approach: Some assumptions appear well founded.

The primary assumption suggested here as the psychological method, is that the causal connection from noise to health effects lies through psychological reaction to the noise via the stress effects caused by or entailed in this reaction. Thus, the assumption asserts that noise causes community reaction which causes or itself entails stress. The stress causes the health effects, consistent with established effects of other stressors (Sarafino 1994) and with the stress-personality relationship influencing the health outcome (Job 2008). The connection between stress and health effects may itself occur directly or through other indirect mechanisms such as stress related sleep loss, or stress induced changes in other health related behaviors such

as drug, tobacco or alcohol use, diet and exercise. These mechanisms are themselves important research issues for determination.

The value of this psychological approach will depend on the validity of the underlying assumptions. The following assumptions are offered, based on the literature as briefly reviewed above and elsewhere (e.g., Job 1996). Apart from assumption 1, these may be the subject of ongoing debate, although in the present authors' views the evidence is quite strong.

1. Noise causes negative psychological/emotional reactions.
2. Psychological reactions cause and/or entail stress (see Hatfield et al. 2001).
3. The stress arising from noise has direct and/or indirect adverse effects on health (e.g., by compromised immunity: Ader & Cohen 1993; Job 2008; or by stress induced changes in cholesterol: Brennan et al. 1992; Lercher & Kofler 1993).
4. Noise causes sleep loss and disruption to sleep architecture.
5. The sleep disruption and loss arising from noise has direct and/or indirect adverse effects on health (e.g., via stress- see Carter et al. 1993 or via compromised immunity: Palmblad et al. 1979).

## **SUMMARY AND CONCLUSIONS**

Experimental manipulation is the compelling method for establishing causal relationships, but many suffer from limited ecological validity, making field research a key element of resolution of applied research questions such as the extent of health effects of noise.

Field research approaches to establishing the existence or (non-existence) of a causal connection between noise and relevant health effects may be divided into those which make no assumptions regarding the underlying causal mechanism(s) by which noise may produce health effects (the epidemiological approach) and those which make, often unstated, assumptions regarding the causal mechanism (e.g., the underlying psychological reaction to the noise causes stress and health effects). While the epidemiological approach demands rigorous measurement of the two critical variables, and if possible, other related variables, and the assumption free approach has obvious appeal, this limits the research to assessment of noise or even sound exposure as the only pertinent independent variable. If, on the other hand, a correct assumption is made regarding the underlying causal mechanism, then the measurement of this variable (e.g., as suggested here negative emotional reaction to the noise, or sleep loss) allows closer examination of the relationship between this critical variable and putative health effects. The present analysis suggests that:

- (1) The causal mechanism assuming approach is only as good as the assumptions it makes;
- (2) There is good evidence for the assumption of certain underlying causal mechanisms;
- (3) Most importantly, the epidemiological approach is not simply aided by the assumption free approach, but rather may fail to identify real relationships.
- (4) The strongest evidence comes from the combined application of rigorous experimental and epidemiological research, with the later able to make effective use of well founded assumptions regarding the causal sequence already established by experimental data (the psychological approach).

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