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FEASIBILITY OF EPIDEMIOLOGIC RESEARCH ON NONAUDITORY HEALTH EFFECTS OF RESIDENTIAL AIRCRAFT NOISE EXPOSURE

Background, General Process Model and Potential Studies

Volume II of III Volumes

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This report examines the feasibility of conducting epidemiologic studies that would support inferences about effects of residential exposure to aircraft noise on nonauditory health. The type of aircraft noise of particular interest is that associated with supersonic and low altitude, high speed flight in Military Operating Areas (MOAs) and Military Training Routes (MTRs): both sonic booms and high peak level, rapid onset time subsonic noise. Potential studies considered are those with observation designs that are community-based or derived from audiometric databases. Since the primary goal of such studies is to improve the Air Force's ability to predict the effects on nonauditory health of noise exposure near MOAs and MTRs, such studies must provide: a demonstration of a causal chain from aircraft noise exposure to nonauditory adverse health consequences; and a reliable quantitative relationship between amount of noise exposure (dose) and degree of specific health consequences (effect).

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Foreword

This report was prepared under Contract F33615-86-C-0530 of the Noise and Sonic Boom Impact Technology (NSBIT) program. The NSBIT program is conducted by the United States Air Force Systems Command, Human Systems Division under the direction of Captain Robert Kull, Program Manager. Mr. Lawrence Finegold served as the contract monitor for this effort.

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Summary

This report examines the feasibility of conducting epidemiologic studies that would support valid inferences about effects of residential exposure to aircraft noise on nonauditory health. The type of aircraft noise of particular interest is that associated with supersonic and low altitude, high speed flight near Military Operating Areas (MOAs) and Military Training Routes (MTRs): both sonic booms and high peak level, rapid onset time subsonic noise. Potential studies considered are those with observational designs that are community-based or derived from audiometric databases. Since the primary goal of such studies is to improve the Air Force's ability to predict the effects on nonauditory health of noise exposure near MOAs and MTRs, such studies must provide:

- a demonstration of a causal chain from aircraft noise exposure to nonauditory adverse health consequences; and
- a reliable quantitative relationship between amount of noise exposure (dose) and degree of specific health consequence (effect).

Volume I of this report is an executive summary of the detailed findings presented in Volume II. Chapter 2 of this report (Volume II) presents background information about the essential requirements of valid epidemiologic study of aircraft noise effects on health. Chapter 3 presents and evaluates information produced by existing studies of noise-related health effects. Chapter 4 develops a general process model from this literature that identifies the links in a chain of reasoning needed to reach conclusions about the effects of aircraft noise on nonauditory health. Chapter 5 assesses the adequacy of a variety of research methods and sites at which community-based studies of aircraft noise effects on people could potentially be studied by epidemiologic methods. Chapter 6 explores the suitability of potential sites for epidemiologic study. Chapter 7 considers alternatives to community-based studies. Chapter 8 presents recommendations for epidemiologic study of the effects of aircraft noise exposure on health. A glossary of terms used in the report precedes the references and appendices at the end of Volume II. Volume III contains a tabular summary of the literature on the effects of noise on health.

1. Introduction

1.1 Purpose of this Study

The National Environmental Policy Act (NEPA) of 1969 requires the U.S. Air Force to predict the effects of its aircraft operations. Major changes in military operations require environmental impact statements, which in turn require prediction of the effects of those changes. The effects may include, among others, potential adverse health consequences of aircraft noise exposure on residential populations. The noise in question includes not only the familiar sort of overflight noise experienced in the vicinity of civilian and military airports, but also the unexpected noise of low altitude, high speed flyovers and sonic booms. The latter forms of aircraft noise exposure are generally produced in the course of training operations, most of which take place near MTRs (Military Training Routes) and MOAs (Military Operating Areas) intentionally sited to avoid high population density areas.

Despite a great deal of research on the effects of noise on health, credible and appropriate information to support predictions by Air Force environmental planners is still lacking. In particular, there is no accepted predictive model establishing a causal chain to link noise exposure with health consequences. As a result, there is no firm basis for predictions of the sort required by environmental impact statements.

Although many documents offer guidance in the preparation of impact statements (e.g., CHABA, 1977, 1981, 1982; Galloway, 1981; EPA, 1973, 1974, 1981, 1982, USAF, 1984; Departments of the Air Force, the Army, and the Navy, 1978; NATO, 1988), they refer largely to studies which report inconclusive findings about the effect of noise on nonauditory health. Most of these documents explicitly note the lack of useful data and the need for further research.

Prediction of potential health consequences of military aircraft noise exposure has proven to be an especially difficult task for the Air Force for a number of reasons. One of the principal reasons is that the technical literature contains numerous reports that some interpret as suggesting that noise exposure (not necessarily produced by aircraft) can have adverse effects on health. The fact that many such reports deal with levels and circumstances of noise exposure very different from residential exposure to aircraft noise near MTRs and MOAs does not necessarily lessen the controversy. Other reasons that environmental planners cannot predict the health consequences of residential aircraft noise exposure include the lack of definitive empirical data to support or dispute the existence of a causal relationship, and the absence of a quantitative dose-response predictive model.

1.2 Fundamental Difficulties in Establishing Health Consequences of Noise Exposure

Although there are good reasons to believe that residential noise exposure created by Air Force operations does not pose a meaningful hazard to human health, the Air Force cannot cite conclusive evidence to support this view. A recent Air Force-sponsored epidemiologic study of the effects of sonic boom exposure on health (Anton-Guirgis et al., 1986) was largely inconclusive, and it is very likely that additional studies of a similar nature would prove similarly inconclusive. Furthermore, the requirements for a proof of safety (i.e., a claim that aircraft noise exposure--or for that matter, any environmental agent--does not create adverse health effects) are far more stringent than the requirements for demonstrating a potential association, especially a noncausal one, between aircraft noise exposure and some health condition (cf. Bross, 1985).

The lack of citable counter-evidence to claims of ill effects of residential aircraft noise exposure is also due in part to researchers' reluctance to undertake a study whose most likely outcome is a finding of no effect. Likewise, professional journals do not generally publish studies which attempt to prove a negative, nor studies which attempt to support the null hypothesis. Failure to detect an effect in any particular study would not in any event constitute logical proof that one might not have been detected by other means, or that an effect might not manifest itself under circumstances somewhat different from those studied.

1.3 Rationale for Current Approach

Definitive statements about the effects of aircraft noise on health require: (1) a demonstration of causality of some prespecified adverse health consequence, and (2) the availability of a reliable, quantitative relationship between degree of exposure and degree of effect. The strategy adopted in the current effort was to cede all benefit of doubt to the view that residential aircraft noise exposure might in fact adversely affect human health, and then to assess the feasibility of conducting studies which could logically support such a view.

If upon completion of the assessment it could be concluded that some form of epidemiologic study were capable of providing support for the adverse effects of aircraft noise on nonauditory health, such study could be recommended. On the other hand, if it could be concluded that certain types of epidemiologic study could not be meaningfully and conclusively conducted in this area, then the Air Force would have documented grounds for not employing such techniques.

Health effects of aircraft noise exposure may be studied in a variety of ways, ranging from highly controlled laboratory studies of infrahuman species, through clinical studies of individual or small groups of human patients, to studies of large samples by epidemiologic techniques. This report deals only with the latter form of study. It evaluates the feasibility of potential solutions to the theoretical and practical problems of conducting epidemiologic research to determine whether human health is affected by subsonic and supersonic aircraft noise.

1.4 Organization of Report

Chapter 2 of this report presents background information about essential requirements of valid epidemiologic study of aircraft noise effects on health. Chapter 3 presents and evaluates information produced from published studies of noise-related health effects. Chapter 4 develops a general process model from this literature that identifies the links in a chain of reasoning needed to reach conclusions about the effects of aircraft noise on human health. Chapter 5 assesses the adequacy of research methods and sites at which community studies of aircraft noise effects on people might be studied by epidemiologic methods. Chapter 6 explores the suitability of potential sites for epidemiologic study. Chapter 7 considers an alternative to community-based studies. Chapter 8 presents recommendations for epidemiologic studies of the effects of aircraft noise exposure on health. A glossary of technical terms used in the report precedes the references and appendices at the end of this volume. Volume III contains a tabular summary of the literature on the effects of noise on health.

2. Review of Basics

This chapter reviews fundamentals of noise exposure and epidemiologic inference. The chapter provides background for Chapter 3, which examines existing evidence of the association between noise exposure and health.

2.1 Review of Noise Exposure Basics

The independent variable of concern in the present feasibility study is aircraft noise. It is axiomatic that no useful dosage-effect relationships, no causal inferences, and no definitive conclusions about the effects of aircraft noise on health can be developed without some meaningful quantification of noise. A fully satisfactory metric for purposes of investigating health consequences of aircraft noise exposure would be simple, accurate, precise, direct, individual- and source-specific, and inexpensive. The following subsection make explicit some of the reasons that such an ideal metric is not now available, and why a full satisfactory metric is unlikely to emerge from conventional (place-oriented) noise measurements.

2.1.1 Difficulties in Measuring Aircraft Noise for Epidemiologic Purposes

Noise--that is, sound that is too expensive or too inconvenient to control--is energy, not a material substance. Unlike material agents and some other forms of energy such as ionizing radiation, noise is not a pathogen that leaves a residue in people that can be measured after the fact. Furthermore, at levels of concern for the case of residential exposure, it produces no obvious stigmata, such as tissue damage or other biological markers.

The most persuasive quantification of noise as an independent variable for epidemiologic analyses requires direct measurement of personal noise "exposure"[1] *as it occurs in real time.*

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[1]Sound exposure at a specified location is the time integral of sound intensity. Sound exposure level is 10 times the logarithm to the base 10 of the ratio of sound exposure to a reference exposure of 400 μPa^2-s. Intensity is the rate of flow of sound energy per unit area per second. At distances from sound sources that are of interest in community analyses, sound intensity is directly proportional to the square of sound pressure. In logarithmic form, squared sound pressure is called sound level and expressed in units of decibels. Thus, sound exposure is usually represented as the time integral of squared sound pressure. This process is often referred to informally as "energy summation." Sound exposure in decibel notation is most often expressed as average (equivalent) sound level over a specified time interval. Single events are usually described by sound exposure level (SEL) in which the reference time interval is 1 s. Cumulative sound exposure is usually described by hourly or 24-h levels.

(As discussed in greater detail elsewhere in this chapter, attempts to reconstruct personal noise exposure after the fact often leave much to be desired.) Exposure is perhaps the most convenient quantity, not only because it combines both intensity and duration into a single index, but also because exposure can represent the total body burden of acoustic energy without regard for its distribution over time.

If one wishes to compare health effects of varying amounts of noise exposure, or to construct dosage-effect relationships for different groups of individuals, one must be able to argue convincingly that a noise measure reflects some aspect of individual exposure. As discussed later, this is almost always an impractical or unaffordable goal in studying the health consequences of long-term residential exposure of large populations to aircraft noise.

In principle, measurement of sound exposure is straightforward: instrumentation has long been available for measuring essentially any characteristic of sound of interest, and simple descriptors of magnitude, spectral content, and duration abound. One is immediately faced with a number of decisions about what aspects of noise are worth measuring, however, when one considers measurement of community noise exposure for epidemiologic purposes. Noises produced by most community sources vary simultaneously in the time, amplitude, and frequency domains in ways that defy simple description.

For example, an aircraft flyover generates a noise signature which, with respect to an observer at a particular point on the ground in a residential community, first increases at some rate (depending on altitude, speed, and flight track geometry) above the ambient noise distribution, then decreases. The vagaries of atmospheric propagation superimpose random perturbations in level on top of this haystack-shaped time pattern. At the same time that the signal is changing more or less rapidly in level, it is also changing in spectral content due to the directivity of different noise sources on the aircraft and to propagation effects (e.g., atmospheric absorption, refraction, diffraction, etc.)

A second observer located elsewhere in the same neighborhood would hear a noise signature of somewhat different temporal pattern and spectral content, depending not only on the geometry of the situation, but also upon other largely unpredictable factors such as propagation and masking effects. It is futile to aspire to any great detail or precision in wide area measurements under these conditions of spatial nonhomogeneity and temporal variability of exposure.

Moreover, environmental noises that are heard in residential neighborhoods are not limited to those produced by aircraft alone. Noises from familiar outdoor neighborhood sources may propagate throughout communities over long distances, creating differing personal exposures in areas that reflect different combinations of local and distant noise sources. It is difficult under most residential circumstances to distinguish the contributions of specific noise sources to the composite exposure produced at particular points.

Even a cursory review of aircraft noise measurement procedures reveals a paradoxical embarrassment of riches, however; there are at least as many ways of measuring aircraft noise as there are purposes for making the measurement. Means have been developed over the last several decades for quantifying virtually all aspects of the acoustic emissions of aircraft, including instantaneous peak levels, total energy normalized to various periods, single event, integrated, averaged, frequency-weighted, and tone-corrected flyover noise at specified distances and locations. Pearsons and Bennett (1974) and Schultz (1982) have cataloged dozens of ways of measuring aircraft and other environmental noises.

Confusion is a common reaction to an initial encounter with the alphabet soup of environmental noise measurement schemes. Inconsistency in terminology for variant measures of the same interactions of physical parameters adds to the confusion,[2] as does incomplete specification of measurement procedures.[3] In the absence of a theoretical basis for deciding which metric(s) best predict(s) which human effect(s) of noise exposure, it is difficult to make reasoned choices among metrics which reflect most of the physical ways in which sounds can covary in time, frequency, and amplitude.

As is often the case when practical decisions must be made about measurement procedures, simplifying assumptions are unavoidable. The elementary decisions that must be made before noise intrusions produced by community noise sources can be represented in quantitative terms are: (1) how to deal with the distribution of energy over frequency (i.e., spectral content) of a noise intrusion; and (2) how to represent the duration and number of noise intrusions over a specified time. Consensus was reached a decade ago on a set of assumptions that permits construction of a family of measurements adequate for most regulatory purposes (EPA, 1974). A frequency weighting network which resembles the inverse of human auditory sensitivity (the A-weighting network) is now universally accepted for measuring nonimpulsive sounds, at least as a starting point for more elaborate measurement schemes. Simple energy integration (10 log duration) is the process adopted to account for duration and number of events.

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[2]For example, short term fluctuations in squared sound pressure level over time (on a scale of tens of milliseconds or less) are described by measures of "impulsiveness" or rise and decay time; longer term variations in sound pressure level over time (on a scale of tens to hundreds of milliseconds or more) are described by measures of "onset time"; yet longer term variations over time (on a scale of seconds to thousands of seconds) are discussed in terms of intermittency or continuity; and even longer term variability in level over time (on a scale of days or longer) is described by "cumulative" or "annualized worst day/week/month" metrics.

[3]A problem often encountered when interpretations of published measurements of noise exposure are attempted is inadequate specification of what has been measured. It is common in the occupational noise effects literature, for example, to encounter statements of the form "The exposure of workers in the high noise group was 90-100 dBA, while that of the nonexposed workers was 70-80 dBA." It is unclear from this description what has been measured and what it represents. Are the cited A-weighted sound pressure levels peak or average values? Are they spatially or temporally averaged throughout the work place? How long were various workers exposed to how much of the noise? Do they represent intermittent or continuous exposure? How may they be compared with similar-appearing figures reported in other studies?

These assumptions suffice to represent the total A-weighted energy of a time-varying flyover, normalized to a nominal 1 s period, as a "Sound Exposure Level" (SEL). SEL values are expressed in decibel notation (ten times the logarithm of the ratio of the squared sound pressure to an agreed upon reference level of 20 μPa). SELs can be logarithmically summed over specified time periods to produce Equivalent Levels (represented symbolically as L_{eq}). Hourly equivalent levels can be summed independently for daytime (0700-2200) and nighttime hours into a measure known as the Day-Night Average Sound Level (represented symbolically as L_{dn} but usually abbreviated as DNL) in which noise exposure occurring during nighttime hours is treated as though it were of a magnitude ten times greater than noise exposure occurring during daytime hours.

The availability of a consistent set of units in which to measure environmental noise exposure has been a great convenience for regulatory purposes, since consistency of measurement permits expression of noise effects criteria in common terms. Consistency also provides a rational basis for controlling noise emissions of a variety of sources. Regulatory purposes are not research purposes, however; measurement procedures developed to make administrative decisions are not necessarily adequate for epidemiologic study of the effects of noise exposure on health.

2.1.2 Inadequacies of Common Noise Metrics

Perhaps the most obvious inadequacy of common metrics of environmental noise exposure for epidemiologic research purposes is that they are inherently place-oriented metrics that may bear no specifiable relationship to personal exposure. Worse yet, outdoor exposure levels are the only ones that can be measured or predicted at reasonable expense and defended as even arguably representative of the exposure of residential populations. Granting for the sake of argument that the outdoor noise exposure of a residential neighborhood can be expressed with useful precision in numeric form, how much can one predict about the noise exposure produced inside particular residences? How much can one predict from outdoor neighborhood noise measurements about personal noise doses of individual household residents?

At first glance, it might appear that indoor and outdoor noise exposure would differ only by a constant (the transmission loss of a typical residential structure) and a small error term. If true, this difference would introduce some uncertainty into predictions of indoor exposure levels based on knowledge of outdoor levels, but conventional statistical methods could treat this uncertainty as just another source of bias or error variance.

Unfortunately, matters are not this simple. Even if one could estimate indoor exposure levels with satisfactory accuracy and precision, one could not lay claim to a persuasive metric of personal exposure for two additional reasons. First, the indoor noise environment of a residence

bears little necessary relationship to the outdoor noise environment of a neighborhood, because the indoor noise environment contains its own noise sources: household equipment and appliances, radio and TV, and other sounds of human habitation. Thus, the level of the indoor noise environment of residences may be higher or lower than the level of the outdoor noise environment at different time periods throughout the day.

Second, household residents are not stationary objects; they move about within homes and leave home entirely for long periods during the day and night. At such times, the correlation between personal noise exposure and outdoor neighborhood noise exposure is essentially zero. Additional detail about these realities of environmental noise exposure may be found in Appendix A.

2.1.3 Alternatives to Empirical Measurements of Outdoor Noise Exposure

Three alternatives to direct measurements of community-wide outdoor noise exposure are conceivable for epidemiologic purposes: individual dosimetry, estimation of exposure by assumption, and reliance upon a surrogate measure of exposure. All of these alternatives have limited value for inferring source-specific dosage-effect relationships, but for different reasons.

2.1.3.1 Individual Dosimetry

Dosimetry is a superficially attractive alternative offering at least the possibility of quantifying noise exposure as heard by individuals rather than as incident on physical locations. A small amount of dosimetric data about individual noise exposure has in fact been published (e.g., Namba and Kuwano, 1979). However, individual dosimetry cannot provide the sort of information needed to derive dosage-effect relationships between aircraft noise and health effects for two major reasons.

First, simple dosimeters (those which measure only integrated energy) are unable to discriminate energy produced by different noise sources. Thus they nonselectively register not only noises from multiple environmental sources, but also self-generated noises. Such noises can generate significant apparent exposure. The long-term average at-ear level of a person's own speech exceeds 75 dBA; artifacts such as brushing contacts of the microphone can also produce spuriously high levels. Even in airport neighborhoods, non-aircraft noise sources make major contributions to total exposure at some times of the day. Greater sophistication is required of dosimeters useful for epidemiologic research purposes, including the signal processing capability for discriminating artifactual from realistic noise exposure.

Second, continuous collection and processing of dosimetric data with existing instrumentation over a prolonged period comparable to the latent period for expression of health

consequences of aircraft noise exposure--say, 5 to 10 years--is a prohibitively expensive proposition. The maintenance requirements alone (monitoring, data reduction, record keeping, calibration and repair, etc.) over any significant period and numbers of cases are formidable.

2.1.3.2 Estimation of Exposure from *A Priori* Information

Credible reconstruction of historical residential noise exposure is possible only under very limited circumstances. The most obvious examples are reconstruction of aircraft noise exposure in neighborhoods near large civil airports or military airbases for which land use compatibility studies have been completed. In such cases, records of numbers, types, and times of day of flight operations are often available for a number of years. It has also become common within the last 15 years for civil airports to publish noise exposure contours for airport environs against which operational information can be checked for consistency, and for military airbases to conduct formal analyses of aircraft noise impacts in Air Installation Compatibility Use Zone (AICUZ) studies using software tools such as NOISEMAP (Beckmann & Seidman, 1978).

The practical difficulties of constructing estimates useful for epidemiologic purposes include:

- accounting for the influence of non-aircraft noise sources in communities near airports (commonly highways and local street traffic);
- accounting for nonscheduled (e.g., military, general aviation) operations; and
- the potentially time consuming and painstaking labor of reconciling operational information from various sources (tower records, OAG publications, airport reports, noise contours, etc.).

If good records are available and the influences of nonscheduled operations and non-aircraft noise sources can be determined, it is possible to retroactively estimate outdoor DNL values for well-defined areas with useful precision. Ninety-five percent confidence intervals approximately 5 dB wide can be expected under optimal conditions (e.g., in proximity to major civil airports.)

The reasons that such estimates can be made with useful precision are that:

- the numbers of operations at major civil airports are very large. For example, international airports serving 20 million or more passengers per year often accommodate 1,000-2,000 operations per day. Relatively large errors in retrospective estimates of numbers of daily flight operations (say, errors on the order of 10-100 flights per day) in such cases will lead to errors of only tenths of units of estimated DNL values.
- total aircraft noise exposure is dominated by the emissions of a small number of well-known aircraft types. These aircraft are generally jet transports for which engine types, power settings, speeds, altitudes, and so forth vary little for well-defined approach and departure profiles, and for which detailed acoustic measurements made under controlled conditions are available.

- approach and departure paths and profiles are stable over periods of years, and fairly narrow. Flight track dispersal, especially over communities near runways, is minimized by air traffic control and navigational aids, and is generally well known from multiple observations.
- variability in operations is low. Apart from minor weekend/weekday and seasonal variability, commercial aircraft operations are generally regular and stable over long periods.
- distances from aircraft to exposed residential areas are relatively short. Thus, variability in noise levels due to atmospheric propagation is small.

However, Air Force environmental planners are not generally concerned with predicting health consequences of the sorts of aircraft noise exposure common in large civil airport environs. Even the airbase case differs markedly from the large civil airport case. Records of flight operations and fleet composition at Air Force bases are rarely as complete or available for periods of as many years as at large civil airports; Air Force bases tend to have fewer daily aircraft operations; variability in operations is likely to be considerable; and flight track dispersal and operational conditions may be more variable than at large civil airports.

Of greater concern for present purposes, however, is estimation of noise exposure near MTRs and MOAs. Flight operations in such areas are often characterized by the following conditions:

- very small numbers of operations (sometimes only a few per day or a few per month);
- sketchy or nonexistent records of actual use patterns of route segments over long periods;
- great potential variability and little documentation of operational parameters (speed, altitude, power settings);
- considerable uncertainty about flight track dispersal (especially for MOAs); and
- multiple aircraft types.

Furthermore, since MTRs and MOAs are intentionally situated at considerable distances from populated areas, noise exposure estimates are generally needed for areas which are distant from flight tracks by several miles or more. Although the average exposure estimates can be made with reasonable confidence, the vagaries of long range acoustic propagation can introduce enormous uncertainties (as much as ± 20 dB) about short-term noise exposure levels[4].

[4]A simple analogy is helpful in understanding the meaning of this degree of uncertainty in quantification of noise exposure. Because of the logarithmic nature of decibel scales, a range of ± 20 dB does not merely imply a range of 40 units, but rather a ratio of 10,000:1. What inferences could be drawn from a study of the effects of water consumption on health if one could not determine whether study participants had drunk 1 glass of water or 10,000 glasses of water on a given day?

Accurate and precise historical reconstruction of noise exposure produced in distant communities by MTR and MOA aircraft operations are often impractical for all of these reasons.

Supersonic Noise Exposure Estimation

Difficult as reconstruction of the noise of subsonic flight operations is, reconstruction of sonic boom exposure is yet more problematical except in a few special cases. A brief explanation of Air Force procedures for monitoring supersonic flight is helpful for understanding why this is so.

Air Force pilots are required to log their supersonic operations on Air Force Form 121. Air Force Regulation 55-354 provided the initial authority for collection and storage of this information in a computer-based repository, currently implemented on an unclassified computer at the Pentagon as a "Sonic Boom Inquiry Data Base" (Kamerman et al., 1986). The earliest data available in the repository date from February of 1968. Approximately 22,000 records of supersonic operations are recorded annually in the repository, for a total of roughly half a million records to date. Records in the database include not only Air Force operations, but also those of Navy, Marine, National Guard, and NASA supersonic operations.

The database *does not contain information about sonic booms per se*, but rather about supersonic flight. An unknown proportion of the supersonic operations recorded in the database produced sonic booms that were audible on the ground. Operational information in the database is sparse, amounting in most cases to little more than the aircraft tail number and a pilot's estimate of his location, Mach number, altitude, and time of supersonic flight. No information is recorded about acceleration, aircraft attitude or maneuvering, all of which can affect the magnitude of sonic booms. In many cases, the location, Mach number, and altitude information is approximate. As a result, the ground positions of potentially audible sonic booms from supersonic flights may not be known to a precision any greater than 1 degree of latitude and longitude.

Since the logging of supersonic flight is not fully automated, compliance is uneven. Reporting of some types of supersonic operations (e.g., T-38 training missions, SR-71 flights) is believed to be quite accurate. Under-reporting of other types of operations (e.g., by test pilots, in over-water and other MOAs) is likely, except when concerted efforts are made over limited periods of time to improve compliance (cf. Kamerman et al., 1986, p. 26). Filing of reports can also be sporadic. Long periods pass when few are filed from some locations, followed sometimes by filing of hundreds of reports before scheduled inspections. Kamerman et al. (1986) note that the database "is not always accurately maintained for routine training flights...," and that "the completeness of reporting is extremely variable for fighter aircraft in general."

Except in isolated cases discussed later, it is effectively impossible to reconstruct the

number or absolute intensity of sonic booms audible at any point on the ground over extended periods with either the accuracy or precision needed for epidemiologic purposes.

Difficulties Common to Both Subsonic and Supersonic Noise Estimation

Any choice of a metric for quantifying noise exposure in an epidemiologic study implies at least a tacit theory about the aspect of noise that is responsible for an adverse health outcome. For example, if one hypothesizes that noise exposure creates adverse health effects by startling people, then a noise metric that reflects peak individual event levels, rise times, and numbers of events would seem most appropriate. On the other hand, if one believes that adverse health consequences are produced by a process related to long-term adverse attitudes toward noise sources (e.g., annoyance), then some other metric of noise exposure, perhaps a cumulative measure of total exposure, might be more appropriate.

The lack of a clear understanding of the mechanisms by which noise exposure might produce adverse health outcomes (cf. Chapter 4) is a major impediment to selection of an appropriate metric of noise exposure for epidemiologic purposes.

2.1.3.3 Reliance upon Surrogate Measures of Noise Exposure

Given the difficulties noted above in quantifying aircraft noise exposure *per se*, it is reasonable to explore whether it might be possible to rely, for epidemiologic purposes, upon measurements of a quantity arguably related to noise exposure. The most obvious quantity is noise-induced sensorineural hearing loss.

Reliance upon hearing loss as a surrogate for noise exposure is not without risk since the etiology of hearing loss is not always determinable without clinical evaluation. For example, certain types of hearing deficits, such as conductive and mycin-related losses, may be completely unrelated to noise exposure. Noise-induced hearing loss is generally a progressive effect of long-term, continuous exposure to very high noise levels. Hearing damage risk criteria for occupational noise exposure, for example, are written in terms of decades of exposure to continuous, daily noise levels on the order of 90 dBA. The initial loss of high frequency (above 4 kHz) hearing sensitivity that is typical of noise-induced hearing loss may not be noticed for many years, even in routine pure tone audiometric screening, until the loss extends to lower frequency regions important for speech intelligibility.

Presbycusis is the term used for the common pattern of high frequency hearing loss in older people. It is not often clear whether an individual's presbycusis is an effect of the accumulation of many years of noise-induced hearing losses ("sociocusis"), or whether it simply reflects normal aging processes such as loss of vascularization of the capillary bed which nourishes the hair cells of the inner ear. More to the point, however, noise induced hearing loss is not a

plausible consequence of residential circumstances of exposure to aircraft noise, since neither the duration nor absolute level of aircraft noise intrusions is sufficient to generate measurable hearing damage. Thus, reliance upon noise-induced hearing loss as a surrogate for measurement of noise exposure is unwarranted in the residential settings of concern to Air Force environmental planners.

The most plausible setting in which noise-induced hearing loss might serve as a surrogate for direct measurements of noise exposure is the occupational setting, in which noise exposure levels are more likely to cause hearing damage. Even in this setting, however, noise-induced hearing loss is not necessarily a good predictor of noise exposure. As just noted, hearing loss is not caused exclusively by noise exposure, nor is an individual's total noise exposure necessarily dominated by occupational exposure. There are also large individual differences in sensitivity to hearing damage. These issues are revisited in greater detail in Chapter 7.

2.1.4 Summary of Difficulties of Acoustic Measurement for Epidemiologic Purposes

The foregoing discussion of problems of acoustic measurement of aircraft noise exposure for epidemiologic purposes may be summarized as follows:

- The most persuasive form of direct measurement is continuous, long-term, source-specific individual dosimetry. This form of measurement of acoustic exposure is the only one consistent with the individual as the preferred unit of analysis in epidemiologic study (v.i.), and the only one capable of discriminating aircraft noise from other exposure sources.
- Given that dosimetric measurement of aircraft noise exposure is unlikely to be economically or technically feasible on a large scale, the next best form of quantification of exposure is wide area, outdoor measurement. Such measurement is a distinct second best, however, since it is inconsistent with epidemiologic analyses of individual effects, and invariably introduces the ecologic fallacy (the assumption that outdoor noise measurements reflect individual noise exposure levels) into study designs.
- The third best method of quantifying aircraft noise exposure is by estimate and assumption. For some types of retrospective studies this is the only form of quantification of exposure that is possible. Reconstruction of historical noise exposure patterns can only rarely be accomplished with the accuracy and precision needed to support valid epidemiologic inference, and introduces the possibility of serious misclassification bias.
- The least satisfactory method of quantifying noise exposure for epidemiologic purposes is by measurement of a surrogate variable such as hearing loss.

In short, the problems of acoustic measurement for epidemiologic studies of aircraft noise effects on health are consequential, intractable in some cases, and unlikely to find practical solutions in many cases.

2.2 Review of Basics of Epidemiologic Inference

The most common objective of epidemiologic studies is to define cause-effect relationships by associating particular exposures with potential biologic or health effects which are highly unlikely to be attributable to extraneous differences between exposed and nonexposed populations. An etiologic association between noise exposure and disease is most simply established in dichotomous fashion, by showing a statistically significant difference in incidence of a given disease in exposed and nonexposed populations.

In reality, imputation of causality in human health research is complex. Causal inference in epidemiology tends to be derived from inductive reasoning. Hypotheses are formed from intuition or other *a priori* information; deductive logic is used to infer predictions from the hypotheses; and empirical observations are compared with deduced predictions. Hypotheses that are not falsified are confirmed, in the sense that they are accepted as plausible explanations until they are falsified and replaced by hypotheses that provide better accounts of observations (Rothman, 1986). Despite philosophic injunctions that inductive logic cannot establish conclusive verification of cause and effect (in the sense of proof in mathematics), causal inferences are nonetheless made to support decisions and actions with regard to critical health problems.

Hill (1965) emphasized this responsibility for making causal judgments when he set forth criteria for distinguishing causal from noncausal associations:

> "All scientific work is incomplete--whether it be observational or experimental. All scientific work is liable to be upset or modified by advancing knowledge we already have, or to postpone the action that it appears to demand at a given time."

At a minimum, assessments of causality should be based upon the degree to which the data meet criteria that are generally accepted for judging causality in observational studies. Furthermore, these standards for inferring causality should only be applied to studies which have been conducted with scientific rigor in terms of design, accuracy of data, avoidance of bias and appropriateness and accuracy of analysis. Six of these criteria set forth by Hill (1965) are noted.

2.2.1 Conditions Required for Inference of Causality in Epidemiologic Study

Temporal Sequence

Basic to all concepts of causality is the requirement that factors thought to be causal must precede the presumed effects in time. This requirement may be more difficult to document than one might expect for noise exposure and a health consequence such as elevated blood pressure; one is dealing with an ubiquitous exposure that is an essential part of life at desirable levels, as well as an effect (blood pressure regulation) which is a continuous, evolving process governed by several self-regulating systems with multiple feedback loops. Furthermore, a long latency or development period (at least 5-10 years) almost certainly would intervene between meaningful exposures and possible adverse health changes.

Strength

The stronger the association between the exposure factor of interest and the health outcome the more likely it is to be causal, because if a (spurious) association were due to confounding or some other bias, the biasing association would have to be even stronger. Weak associations are more likely than strong ones to be explained by undetected biases. However, the fact that an association is weak does not rule out a causal association, since the strength of an association is not a biologic feature. Strength of association is determined by the ratio of incidence rates in the variously exposed groups (relative risk). Knowledge of even weak causal relations is of value when the exposure affects large numbers of people, the disease occurs frequently and the exposure is readily susceptible to interventive change.

Dose-Response

More convincing evidence of a causal association is provided by a dose-response relationship, that is, an increase in frequency of an adverse health outcome with a corresponding increase in the level of (noise) exposure. Nonetheless, some causal associations may show no trend of effect with dose if all doses of exposure are sufficiently great to produce the effect, and/or the actual health change depends on other component causes or an interaction of causes.

Coherence

The observed association between the factor (noise) and the health problem should be biologically plausible and compatible with the existing knowledge.

Consistency

Consistent findings about types of associations and dose response relationships across many populations and across scientifically sound studies add credibility to the evidence of cause.

Interventions

Experimental evidence is seldom available for epidemiologic research with human populations. Therefore, the ultimate test of causality in observational research is the degree to which manipulations (usually reductions) of the risk factor influence the frequency of the health condition.

2.2.2 Epidemiologic Inference and Study Design

Epidemiologic studies may be classified as experimental or observational. In experimental studies, the investigator assigns individuals to exposure and nonexposure in an unbiased (often randomized) manner and observes health event outcomes. In observational studies, nature takes its course, and changes or differences in environmental, social, psychologic, biologic or other exposure characteristics are related to changes or differences in the health state, if any. Experimental epidemiologic studies of long-term effects of environmental exposures are not feasible for ethical and practical reasons. Intervention trials and adventitious experiments in which exposure changes for other than research purposes are the exception. As a result, the experimental approach is most likely to be associated with research on animals. Thus, a major strength of observational studies in relation to most experimental research is that they apply directly to human beings.

Observational studies may be categorized as etiologic (causal) or descriptive in nature. In etiologic studies, research is concerned with health states which may be caused or prevented when healthy persons are exposed to factors under study (e.g., aircraft noise). Presumed causal factors, usually referred to as risk factors, are characteristics that play an essential role in producing the *occurrence* of a given health state. Included in this concept of risk factors are those factors which serve primarily as precipitants of existing, but undetected or undetectable disease. For example, cigarette smoking is believed to act as a precipitant of sudden cardiac death.

Assessment of cause of disease expressed in terms of risk or probability of an event is best made in terms of the number of new cases of the disease that occur during a specific time period. The incidence rate is calculated as the ratio of the number of *new* cases occurring over the time period to the number initially at risk. Incidence rates are required to calculate relative risks. The relative risk expresses the probability of developing a disease for individuals exposed to some factor compared to the probability of unexposed individuals' developing the disease. Thus, the relative risk is a ratio of two incidence rates, a conditional probability. A relative risk of "1" indicates no association between the factor and the health event; any value higher than "1" means

there is a positive association or increased risk relative to the reference group of no exposure or low exposure; a value lower than "1", or a negative association, suggests that the factor may have a protective effect.

Because the relative risk indicates which factors may be causal, it specifies what needs to be changed to prevent or reduce disease incidence in populations. The direction and magnitude to the relative risk suggests the relative importance among all causes of the presumed etiologic agent to the health outcome. Since most diseases have many causes, a one-to-one correspondence between the exposure factor and disease would not be expected. One useful characteristic of the relative risk is that the greater the number of causal factors producing the disease state, the weaker the association will be between any one of them and the disease.

Prevalence is the only measure of disease frequency obtainable in cross-sectional studies. Prevalence refers to all cases of disease present at a given moment (or during a period of time) irrespective of the time of onset of disease. Thus prevalence depends on duration as well as incidence. Disease of long duration (such as heart disease and hypertension) tend to have higher prevalence than short term illnesses, even if the total numbers of newly affected individuals are about equal. A prevalence rate is the ratio of all existing cases to the number of persons in the target population at a point in time. Thus, it is not an estimate of the probability of occurrence of an event.

Cohort and case control designs, as well as hybrids of these designs, are forms of observational studies appropriate for etiologically-oriented research. Cross-sectional studies generate prevalence data and are basically descriptive. Fundamental to each of these three research designs is the use of the individual as the unit of analysis and the ability to assign exposure and health status to individual subjects. This allows adjustments for confounding variables and the assessment of interactions.

Ecologic studies, also descriptive, tend to be cross-sectional in design. In addition, the group rather than the individual becomes the unit of analysis. This makes it extremely difficult to adjust for confounding factors and impossible to consider potential interactions of putative risk factors.

The following subsections focus on selected issues as they apply to research on the effect of noise: (1) epidemiologic study designs, (2) the role of clinical and animal studies, (3) problems associated with latency and weak associations, (4) methodological issues of statistical power and sample size, and (5) various forms of bias.

2.2.2.1 Ecologic Designs

The term "ecological" has been applied to studies in which disease frequency is correlated with some measure of aggregate exposure to a supposed cause of disease in population strata defined by place, time or person variables.

Measures of association between noise exposure and disease frequency derived from ecologic studies may not adequately reflect the true strength of the association in individuals even when an association exists and is causal. As described by Morgenstern (1982), a fundamental limitation of ecologic data relative to cohort or case-control studies is the lack of information about the joint distribution of the study factor (noise exposure) and the disease within each group. The group becomes the unit of analysis: the numbers of cases of illness are known in the noise exposed and nonexposed groups, but the numbers of exposed cases are not knowable.

The feasibility of an ecologic study depends in part on the quality of morbidity and mortality data available over an extended period, preferably 10-20 years for diseases such as hypertension and heart disease. Use of incidence data gives greater validity to inferences drawn from ecologic analyses. Once collected, data can be evaluated through multiple time series analysis, a procedure which can be used to track changes in disease over time for exposed and comparison groups. Statistical tests are available to assess both overall differences in rates of disease (hypertension, myocardial infarct and arrhythmias) between the two groups as well as differences between the groups in the pattern of incidence over time.

The relationship between the change in the average exposure level determined from one data source and the change in the disease rate for a group determined from a separate source is examined in the time trend analysis of aggregate data. Adjustments for confounders are made from mean estimates of the level of the variables in the groups. When there is a gradual change in the average (noise) exposure levels, or when there is a lag for the development of disease, trends in both exposure and disease must be compared. Changes in levels of potential confounders over time should be taken into account as well.

Morgenstern (1982) describes a method for estimating the relative risk from ecologic data and suggests that ecologic data can be used in testing hypotheses in certain situations where aggregate measurements may be more accurate than individual measurements. He also enumerates the limitations of drawing causal inferences from ecologic data. These include the ecologic fallacy composed of bias due to grouping of individuals in addition to confounding bias; the sum of which may be called cross-level bias. According to Morgenstern, "ordinarily, cross-level bias exaggerates the magnitude of the true association." Other problems include reversal of the hypothesized cause and effect, measurement error, migration between groups and multicollinearity (extremely high intercorrelations.) Multicollinearity is especially problematic

for studies using geographically defined units of analysis that are large and/or few in number. The larger the population units under study, the cruder the data are likely to be, resulting in observed associations between the exposure and disease which can be explained by a relationship between other variables highly correlated with both exposure and disease.

Morgenstern suggests that these problems can be minimized by (1) using ecologic regression with as many risk factors as possible in the model, (2) making groups relatively homogeneous by using the smallest possible geographical units for analysis, (3) determining how groups were formed and analyzing accordingly, and (4) comparing ecologic results with findings of other observational studies designed to test etiologic hypotheses. Comparability of findings from studies of different design depends upon the quality of the data, the strength of the risk factor considered, the geographical distribution of confounding factors, and adjustment models used.

Ecologic comparisons are often referred to as geographic associations. In the past, they have been used primarily to suggest hypotheses that require individual-based data for further testing. One example is a study of changes in average annual age-sex specific cardiovascular disease mortality rates and change in water hardness (Crawford et al., 1981). Crawford found that the increase in cardiovascular mortality, particularly in middle-aged men, was less in towns that made their water harder than in towns that made their water softer. One inference that might be drawn from this finding is that water hardness protects against cardiovascular disease. Alternatively, the observed relationship between the two variables might be due to changes in diagnostic customs, or to trends in other cardiovascular risk factors, or to changes in the populations of compared areas over time. Interpretation of these results remain open to question until the association is or is not demonstrated in more etiologically-based studies.

Connor and Gillings (1984) have recently advised cautious use of aggregate level data when inferences needed about associations are concerned with individual-level relationships. Connor and Gillings compared aggregate level data from a Blue Cross-Blue Shield database to data collected from a random sample of 700 individuals in the health service area. Opposite conclusions about the impact of home health services to acute care utilization were supported by the two information sources. Richardson et al. (1987) have empirically demonstrated that in the case of complex multifactorial disease, ecologic analyses are likely to fail to identify etiologic factors. Thus, conclusions drawn from ecologic studies of airport environs are not likely to clarify existing understandings of the effects of aircraft noise on health.

2.2.2.2 Cross-sectional (Prevalence) Studies

Cross-sectional studies, although primarily descriptive, allow examination of the relationship between disease and factors thought to be causal as they exist in a defined population at a particular time. The basic issue addressed in a cross-sectional study is whether the exposure factors coexist with the health problem of interest. Cross-sectional studies provide prevalence data; therefore, they are important for identifying the extent of a problem in a given community so that health programs can be planned to prevent or treat that problem. They also serve a very useful purpose from an analytic standpoint. Associations observed in prevalence studies often suggest etiologic hypotheses that can be tested using more rigorous study methods. Frequently, a cross-sectional study is a preliminary step to a prospective cohort study. When a representative sample is employed with a cross-sectional study, those individuals defined as noncases become the cohort study population.

The cross-sectional study begins with a set of questions about the relationship between some possible risk factor and the disease of interest. Once the sample is defined, the necessary data are collected from each subject. Assessment of the study exposures and health outcome are made simultaneously in time.

Prevalence ratios are calculated by comparing the disease prevalence rates in the various exposure groups. If the group of individuals with the suspected factor is found to experience a higher rate of disease than those individuals without the factor, an association between the factor and the health state is said to exist. However, risk cannot be inferred from these prevalence ratios for three reasons. First, there is the antecedent-consequent problem. Because the information about the exposure characteristic and the health outcome are described simultaneously in time, one does not know which occurred first, the presumed cause or the disease (effect). Second, a cross-sectional study deals with survivors or all individuals (cases and noncases) who happen to be present in the target area at the time of the study. Individuals with the exposure characteristic may have died or migrated into or out of the study area at a different rate from those without the exposure characteristic. This is known as selective survival or selective migration and may lead to an unknown overestimate or an unknown underestimate of the association between the exposure and disease. Third, the disease process itself may change the biochemical, psychological, or physiological responses of those observed to have the disease and thus, lead to errors in inferring the correct relationships between exposures and health states. (For example, there is some evidence that cholesterol is lower after an acute myocardial infarction than before.)

2.2.2.3 Cohort Studies

A cohort study is one in which subjects are sampled on the basis of exposure to the etiologic factor of interest and followed for health outcome. Followup over time may be conducted in either of two ways. First, the cohort can be assembled in the present and the individuals followed prospectively into the future. Second, historical records can be used to identify the exposure characteristics of a defined and healthy group at a specific time in the past, then new cases of disease occurring in the cohort between the specified time in the past and the present may be determined from existing records. This design, variously termed a historical prospective design, a nonconcurrent prospective design, and a retrospective cohort study, is hereafter referred to as retrospective cohort. In both cohort designs, the individuals comprising the cohort are identified and information about their exposure is obtained before their disease experience is ascertained.

In a prospective cohort study, each cohort member is determined to be free of the specified diseases or health outcomes (e.g., elevated blood pressure) and classified as exposed or unexposed to the factor of interest (e.g., aircraft noise). Members of the cohort are then re-examined over a period of time during which new cases of disease or changes in the health state are identified. Exposure is quantified at study onset and reassessed at periodic intervals. Greater detail of exposure data (e.g., level of exposure, time of onset and duration of given levels of exposure, and whether exposure has stopped) leads to greater credibility of the causality of any observed association. The unexposed group may be defined as having no exposure to the presumed deleterious agent, or as experiencing lower doses than the exposed group. One difficulty with this approach is that an effect may not be detected if both lower and higher doses of exposure share a common effect.

By counting only new cases occurring during the followup period, the rate of disease is not biased upward by inclusion of long-term or recurrent chronic disease, nor is it decreased by early death or recovery. As previously indicated, the incidence rate (i.e., new cases) is particularly valuable for etiologic investigations because it can be interpreted as a probability and used to determine relative risk.

To avoid bias the exposed and nonexposed groups must be as similar as possible with respect to (1) the method of identification of subjects, (2) the method of followup, (3) the method of data collection on health outcomes and control variables, and (4) known risk factors for the health outcomes under study. To provide comparability of strong risk factors, researchers often collect data on potentially confounding variables and adjust for them in the analysis, a practice which introduces its own problems. It is important for a study to control all potentially confounding variables. Remaining uncontrolled differences between exposed and nonexposed groups must be considered as potential causes of the adverse health effect under study. Comparison of relative risks across studies should be based on estimates that have been adjusted for the same major confounding variables.

Research results can be difficult to interpret when data are adjusted for potentially confounding variables. The adjustment can be viewed as a device for statistically matching members of all groups. The analysis is performed on health outcome values that would have occurred had all participants been identical on the confounding variables, a supposition that may strain plausibility. A factor intervening in the causal pathway between exposure and disease which mediates or potentiates the effect of the exposure should not be treated as a confounder since its control can spuriously reduce any evidence in the data of a true association. Assessment of interactions of factors--that is, assessing whether the magnitude of the association between the exposure factor and a disease differs according to the level of a third variable--contributes to the understanding of disease causation. For instance, the relative risk between high noise and hypertension may be 2.0 when data for all men are considered; however, this overall risk may disguise the fact that men with a family history of cardiovascular disease (or men who are annoyed by noise) are at higher risk of hypertension when exposed to noise than men without this third characteristic.

Another potential source of bias in cohort studies is attrition. Followup of the individuals enrolled in a cohort study over time is an essential feature of the study design. If a substantial proportion of the cohort is lost to followup, the validity of the study conclusions is seriously compromised. Therefore, all subjects in the original cohort of exposed and nonexposed individuals must be accounted for whether they have developed the condition of interest or not.

Cohort studies in which individuals are followed forward over time are preferred because they offer the best opportunity for accurate measurement of exposures and outcomes and for identification of the cohort. However, the retrospective cohort study as described by Lilienfield and Lilienfield (1980) is frequently used in the study of workers or individuals enrolled in Health Maintenance Organizations (HMOs) for a long time. This design depends upon the availability of prior information on study factors for a well-defined population that has been followed for detection of new diseases or deaths. Attention to the fundamental issues of initial exposure characterization and health status, as well as followup status, are critical for such studies. A major limitation of the retrospective cohort study is that data relative to potential confounding and/or mediating variables are often missing, even when the exposure and outcome of interest are fairly well defined. When cohorts are available for study retrospectively, this design can be cost-effective for studying rare diseases or problems associated with long periods of latency.

2.2.2.4 Case-Control Designs

In contrast to cohort studies, the case-control or retrospective sampling approach begins with selection of cases and controls from the same referent population. Cases and controls are then assessed for exposure to the factor at an appropriate time in the past prior to the development of disease. The exposure odds ratio which can be derived under this sampling

frame provides an unbiased estimate of the incidence rate ratio, or relative risk (Miettinen, 1976). Estimation of relative risk follows from interpreting the case-control study as the result of sampling from a large, often fictive, cohort study from which incidence rates can hypothetically be estimated. Temporal bias is the greatest potential disadvantage. Selective survival and selective recall are more likely to present problems in the case-control than cohort designs. Although the case-control study is often criticized for its biases, it is increasingly recognized that it is not the analytic strategy itself but its misuse that is the problem (Ibrahim, 1979). Rothman (1986, p. 64) describes the strategy as follows:

> "The case-control design can be conceptualized as a followup design in which the person-time experience of the denominators of the incidence rates is sampled rather than measured outright. The sampling must be independent of exposure; by revealing the relative size of the person-time denominators for the exposed and unexposed incidence rates, the sampling process allows the calculation of the relative magnitude of incidence rates. Viewed in this way, the case-control study design can be considered a more efficient form of the followup study, in which the cases are the same as those that would be included in a followup study and the controls provide a fast and inexpensive means for inferring the distribution of person-time experience according to exposure in the population that gave risk to the cases."

In a case-control study it is strongly preferable to select incident cases (those who develop the illness during a specified time) rather than prevalent cases (those who are already ill). If exposure affects the duration of illness, as it often does, then a study based on prevalent cases will be unable to distinguish an etiologic role for the exposure from its effect on duration.

2.2.2.5 Hybrid Designs

A hybrid of the cohort and case-control strategies, referred to as an ambidirectional study by Kleinbaum et al. (1982) and nested-case-control by others (Lubin, 1986), combines some of the advantages of both cohort and case-control studies. In this design, a single population is defined at the onset without regard to exposure information and is followed for a defined period of time for the detection of all incident cases. The incident cases are then compared to a group of controls sampled from the same population with respect to previous exposure levels. The most appropriate situation for an ambidirectional study is one in which it is possible to identify most new cases of disease in a large population by using existing information systems such as insurance or employment records, disease registries or health system records (such as HMOs) or military health data systems which serve a defined area or defined group of people.

The advantage of this design over the case-control design is the assurance that cases and controls are identified from the same defined population. The ambidirectional study has the advantage over cohort studies of precluding the measurement of exposures on every subject in the study population, and is therefore more cost-effective.

2.2.2.6 Clinical Studies

A characteristic feature of clinical observations of health effects of aircraft noise exposure that are relevant to the general process model described in Chapter 4 is real-time monitoring of cardiac function in relatively small numbers of people exposed to aircraft or other noise with varying degrees of control. These groups could in principle range from patients in coronary care units in hospitals near flight paths (if such exist and are meaningfully exposed to aircraft noise), to ambulatory cardiac patients in health care institutions, to cardiac outpatients and groups of people with no overt cardiovascular disease in residential settings. Continuous monitoring of physiological parameters (e.g., arrhythmias, beat-to-beat heart rate variability, blood pressure, etc.) in such people, if coupled with simultaneous, source-specific measurement of noise exposure, could provide useful information about the range of physiological responses to acute and possibly even chronic noise exposure.

A number of practical difficulties limit the amount of data that can be collected in this fashion, however:

- The existence and accessibility of patient populations exposed in residential settings to the sorts of aircraft noise of greatest interest to the Air Force is unclear;
- Short term monitoring (on the order of weeks or even months) does not yield information optimally useful for assessing public health risks of chronic exposure to aircraft noise;
- New technology would have to be developed to permit cost-effective personal noise exposure monitoring and automated processing of very large quantities of temporally correlated physiological and acoustic recordings; and
- Difficulties related to obtaining informed consent for intentional exposure to aircraft noise of persons at relatively high risk of cardiovascular disease, as well as difficulties related to double blind study design, are likely to be encountered.

Clinical and laboratory studies of humans add to the weight of evidence for associations between exposures and health states and are especially helpful in indicating potential pathways through which proposed risk factors operate. Detailed descriptions of exposures and concomitant health parameters may suggest the range of noise levels and sources of noise as well as specific health states or biologic markers to be addressed in etiologic studies. These studies are not usually designed to support causal inference of long-term noise exposure effects on chronic diseases such as hypertension and myocardial infarction.

2.2.2.7 Animal Studies

Experimental research with animal models has certain advantages over observational research with human subjects. In general, epidemiology depends upon animal and laboratory studies to delineate specific mechanisms by which exposure actually produces observed health changes, and to test causal hypotheses under rigidly controlled conditions. The use of animals permits detailed specification of noise exposure, sophisticated continuous monitoring of physiologic activity and use of invasive measurement techniques if necessary, as well as control of extraneous variables.

The validity of this sort of research depends upon use of appropriate animal models; in this case, the selection of an animal with auditory and cardiovascular systems as similar to humans as possible. At present, the nonhuman primate, as used in the studies by Peterson et al. (1984) appears to be the animal of choice for research into chronic noise exposure. Questions as to whether long-term experiments in animals can yield information generalizable to the long-term course of noise effects in humans have not been adequately addressed to date. Peterson et al. (1983) describe their results as representing responses in "nonhuman species under a single set of conditions and for a necessarily limited period of time...". Although extrapolations from animals to humans can rarely be wholly appropriate no matter how carefully the animal model is selected and the study is designed, animal experiments complement observations on humans and often serve as an effective and cost-efficient prerequisite to time-consuming epidemiologic studies on humans (Hegsted, 1975).

2.2.3 Problems Associated with Lengthy Latent and Disease Induction Periods

Every epidemiologic study is based on either tacit or explicit assumptions about induction time and latent period of disease. Assumptions made about the induction period directly affect the credibility of study results, since inaccurate assumptions cause a special type of misclassification which results in an underestimation of effects (Rothman, 1986, p. 72). The induction period is usually defined as the time from causal action until disease is initiated. During this time many other causes are presumably operating as well. Therefore induction time can be conceptualized only in relation to a specific cause. The time interval between disease occurrence and detection has been called the latent period. Very little is known about the induction and latent periods for chronic diseases such as heart disease and hypertension.

In situations where the disease process may be reversed at any point, the latent period is encompassed by the induction period. Consequently, induction and latent periods are frequently used interchangeably to indicate the interval between first exposure and first appearance of symptoms (Armenian and Lilienfield, 1983). Conceptualizing the induction period from the time of first exposure involves the extreme assumption that the initial exposure amounted to a

biologically effective one. This assumption is highly questionable for sonic boom and other forms of aircraft noise exposure.

An advantage of an accurate estimate of the latent period (or induction-latent period) in a disease or health condition is the ability to determine the period after exposure for which individuals must be followed in prospective studies before expecting an effect. An accurate estimate of the latent period also permits determination of the period in the past that must elapse in case-control studies to allow identification of an etiologic agent.

When exposure itself is chronic it is necessary to conceptualize a period during which the exposure accumulates sufficiently to trigger a step in the causal process. This accumulation of exposure may be a function of time and intensity, implying that longer exposure times would be needed for etiologic success with smaller doses. In principle, the induction period begins only after the exposure has reached this triggering point (Rothman, 1986).

One approach to evaluating induction or latent periods consists of analyzing exposure history on an annual basis, and relating the information for each year with the specific endpoint under study. Two, 5, or 10 year windows might be preferred, varying roughly in proportion to the minimum time between exposure to the etiologic agent and disease detection. An open-ended period which allows for a minimum induction but no maximum period may fit some disease etiologies (Rothman, 1981).

The induction or latent period for the effect of aircraft noise exposure on elevated blood pressure or the cardiovascular system is essentially unknown. In general, studies indicate blood pressure increases with age when all etiologic factors are considered. Annual incidence rates ranging from 16.6 to 42.8 per 1,000 found in the 30 year Framingham followup study (Dannenberg et al., 1988) are noted in Table 2-1. Studies of high noise exposure in industry and prevalence of elevated blood pressure or hypertension suggest that continuous exposures over periods of 5-10 years are required to trigger adverse blood pressure changes (Verbeek et al., 1987; Kachnyi, 1977; Friedlander et al., undated; Meinhart & Renker, 1979; and Andriukin, 1961).

2.2.4 Epidemiologic Inference and Weak Associations

When cause and effect are strongly linked and when the effect follows soon after a cause, an association is easily discovered. Strong associations between high concentrations of chemicals and health outcomes and between potent viruses and disease have been observed for decades. However, it is rare to observe very strong associations between ubiquitous environmental exposures and disease, especially when the health outcome has multiple determinants. When an association between cause and effect is weak, and particularly if the

Table 2-1: Hypertension Incidence Rates per Year per 1,000 Persons.[1]

Incidence per Year per 1,000		
Age	*Men*	*Women*
40-49	21.9	18.0
50-59	23.6	24.9
60-69	28.0	34.7
70-79	31.1	42.8

[1]Based on Framingham study (Dannenberg et al., 1988).

effect occurs long after the cause, studies are likely either (1) to fail to show an association when in fact one exists, or (2) to demonstrate a spurious one when none in fact exists.

The usual measure of association in cohort and case-control studies is the relative risk or estimated relative risk. Generally the term relative risk is used without reference to whether an association is causal. Epidemiologists participating in workshops on "Guidelines to the Epidemiology of Weak Associations" considered weak associations as those with relative risks between 1.0 and 3.0 (or between .3 and 1.0 if a reduced risk) (American Health Foundation, 1982; Wynder, 1987). These risks represent situations in which exposure produces approximately a doubling or a halving of the risk of disease. Although a risk of 1.2 is taken to indicate essentially no association, this does not necessarily mean that the true association is 1.0. Rather, it means that when the exposure increases the adverse health effect by as little as 20% among the exposed, epidemiologic methods are unlikely to detect the association. A small effect or lack of evidence of a statistically significant effect is not evidence of a lack of a meaningful effect. An association of only 1.2 may be of public interest when the prevalence of both the risk factor (exposure) and the disease outcome is high.

In situations where the true association is weak, the range of variability in data is large relative to the true rate ratio and confounding bias may explain the observed risk. By contrast, the magnitude of the relative risk has little to do with the possibility that an observed association could be due to selection bias or observation bias (Monson, 1980). It is important to note that the closer the risk ratio approaches unity, the greater is the need for understanding of the underlying biologic mechanisms and the more likely it is that the observed association will be the result of bias, confounding, inappropriate analysis, poor choice of controls, or unexposed comparison groups (Wynder, 1987).

Problems in interpreting weak associations are especially relevant to the study of the effects

of aircraft noise on cardiovascular health since observed risk ratios in prevalence data range from about 1.3 to 3.5.

2.2.5 Statistical Power and Sample Size

The "power" of a study design is the probability that an adverse health effect of a specific magnitude will be detected when it is actually present in the population under investigation. It is a function of the following factors:

- The size of the groups being compared (exposed vs. nonexposed or cases vs. controls). In general, power increases as sample size increases.
- The strength of the expected association (relative risk) between exposure and outcome. Power is directly related to the size of the risk when all other parameters are fixed.
- The variability of the health outcome. In general, power is inversely related to the variability of the health measure.
- The precision in measurements of exposure and health outcome. In general, precision can be improved by increasing sample size, but refinement of exposure and response variables is generally necessary to avoid misclassification bias.
- The predetermined level of statistical significance (alpha error, or the probability of falsely detecting a nonexistent effect) that will be accepted as confirmation of an association between exposure and health outcome.

Tables 2-2 and 2-3, based on Schlesselman's (1974) work, show sample size requirements for cohort and case-control studies of the effect of noise exposure on hypertension, assuming no confounding. Few studies have been published on the incidence of hypertension. For sample size planning purposes, the average annual incidence rate among males in the 30 year Framingham study of 26 per 1,000 persons aged 40-79 years of age is used as a general estimate of incidence among controls or nonexposed (Dannenberg et al., 1988). Since the Paffenbarger et al. study (1983) of Harvard alumni is frequently cited, sample sizes are also shown based on estimates from this study. The annual incidence of hypertension derived from this study is 8 per 1,000 for males ages 35-74. For each sample size, Table 2-2 shows the magnitude of the effect that can be demonstrated at the two-tailed 5% significance level with a power of 90%.

For example, to detect a risk of 1.5 in a cohort study at these statistical error levels, a sample of about 3,904 exposed and 3,904 nonexposed (a total of 7,808 people) would be needed. Followup of this cohort for 5 years would generate only 723 hypertensives among the noise exposed and 482 among the nonexposed. If the true underlying rate were lower, a much larger sample size would be required to detect small differences between the exposed and nonexposed groups. If one is willing to settle for detecting only larger differences (for example, at least a

Table 2-2: Sample Size Requirements[1] for Cohort Studies of Noise Exposure and Hypertension.

Sample Size Requirements		
Relative Risk	*Cohort Sample Size (.026 Annual Incidence in Nonexposed)*[2]	*Cohort Sample Size (.008 Annual Incidence in Nonexposed)*[3]
1.5	3,904	12,989
2.0	1,162	3,887
2.5	597	2,011
3.0	381	1,289
3.5	272	926
4.0	208	712

$\alpha = .05$, 2-sided test; $\beta = .10$

Notes: Indicated sample sizes are for each (exposed and nonexposed) group.

Hypertension defined as SBP of at least 160 mm Hg and/or DBP of at least 95 mm on at least two BP readings.

[1]Calculated according to Schlesselman (1974.)

[2]Based on average annual incidence for men and women aged 40-79 years in Framingham Study (Dannenberg et al., 1988).

[3]Based on average annual incidence for males aged 35-74 years in Paffenbarger et al. (1983) study.

Table 2-3: Sample Size Requirements[1] for Case-Control Studies of Noise Exposure and Hypertension.

Case-Control Sample Size for Noise Exposure in Controls of:								
Relative Risk	*7%*	*15%*	*20%*	*30%*	*40%*	*50%*	*60%*	*70%*
1.5	1662	878	714	568	518	518	561	668
2.0	510	277	229	188	176	182	203	248
2.5	270	150	125	105	102	107	122	152
3.0	177	100	85	74	71	77	89	113
3.5	129	74	64	56	56	61	71	92
4.0	101	59	51	45	46	51	61	79

$\alpha = .05$, 2-sided test; $\beta = .10$

Notes: Sample sizes for each group (cases and controls).

Hypertension defined as SBP of at least 160 mm Hg and/or DBP of at least 95 mm on at least two BP readings.

Relative risk is estimated by the odds ratio.

[1] Calculated according to Schlesselman (1974).

two-fold difference between the exposed and nonexposed groups) smaller samples would suffice. To detect a risk of 2.0, only 1,162 in each group (2,324 people in all) would be needed in the cohort. It should be noted, however, that these sample sizes may be inadequate for detecting even a reasonably large risk if the population is very heterogeneous on factors which may confound or mediate the relationship between blood pressure and noise exposure.

For the case-control design, cases of hypertension and controls without hypertension would be selected from the same referent population. The columns of Table 2-3 display sample sizes as a function of the prevalence of the exposure (noise) among controls, based on Schlesselman's (1974) tables. The case-control study requires considerably fewer subjects in each group than a cohort study when the risk to be detected is small and 7% or more of the controls are exposed to noise.

However, it should be noted that large referent populations are also implied for case-control studies. For example, to detect a risk of 1.5 in a case-control study when the population disease rate is 26 per 1,000 and 50% of the controls are exposed, 518 cases and an equal number of controls are needed. To ascertain these 518 new cases within a year, a population of 19,923 would need to be screened. To detect a four-fold difference, 1,962 people would have to be screened to identify 51 cases.

These sample size estimates are optimistic ones, since they fail to account for the effects of potential confounding variables. They also do not address the ratio of controls to cases that may be advisable for the sake of efficiency in gaining statistical power.

One way to reduce the sample size for a study is to reduce the study power. However, prior studies which have failed to demonstrate associations between noise exposure and blood pressure changes have suffered from power too low to detect small differences. Therefore, reducing the power in sample size planning is undesirable for present purposes.

Failure to show associations imposes a burden of documenting sufficient power and rigor of research methodology to uncover a *bona fide* effect, were one to exist. It is thus virtually impossible to defend a finding of null results. Professional journals traditionally are loathe to publish reports of such studies for reasons related to the nature of statistical inference. The inferential model sets up a null hypothesis that no association exists to constrain researchers from asserting the existence of spurious relationships. Only in the face of highly convincing evidence, of the sort that may not be realistically obtainable in epidemiologic studies (Bross, 1985), can that hypothesis be rejected.

If the evidence is insufficiently convincing, the null hypothesis is retained, but never accepted. That is, a researcher can fail to reject the null hypothesis, but cannot accept it, because there are too many factors, other than a lack of association, that can produce null results. One

such factor just discussed is lack of power: small associations require large samples in order to be revealed. Other factors include imperfect measurement of variables--so that an association that exists is hidden--or insufficient control of potential confounding variables.

In short, nonoptimal research methods can easily produce null findings even when a true association exists. Researchers, therefore, do not generally conduct research in which they expect to find a null result.

2.2.6 Bias: Nonresponse, Confounding, and Misclassification Bias

Bias is any process at any stage of inference which tends to produce results or conclusions that differ systematically from the truth (Sackett, 1979). Avoidance of bias in epidemiologic studies is particularly important when there is reason to believe that the association under investigation is a weak one.

Major biases which may affect a cohort study are nonrepresentativeness of the range of exposures, differential distribution of potential confounders in the exposed and nonexposed groups, and the presence of incipient disease. In a case-control study, biases may result because of factors that affect the ascertainment of diseased cases, survival with the disease and the stage of disease (incident or prevalent cases). Potential biases in epidemiologic studies are extensively discussed in most textbooks (Schlesselman, 1982; Kleinbaum et al., 1982). Three sources of bias are of special concern in noise effect studies: nonresponse bias, confounding bias, and misclassification bias.

2.2.6.1 Nonresponse Bias

Two types of nonresponse typical in cohort studies are selection bias and attrition. Selection bias is produced by subjects' refusal or nonresponse to inquiries requesting participation in a study. Attrition is produced by individuals losing interest in continuing their participation in study monitoring procedures over time. The best study design can be compromised by high rates of refusal or high attrition rates. It is difficult for a researcher to assert that those who choose to cooperate and remain in the study represent the population of interest. Moreover, results become uninterpretable when response rates differ for exposed and nonexposed individuals. Efforts to assure that exposed and nonexposed are alike in all respects other than exposure are wasted if differential drop-out occurs, since one cannot readily refute the argument that those who choose to drop out differ in some systematic way from those who choose to remain under study.

The proportion of individuals declining to participate in studies in the United States has increased in the past 25 years, as evidenced by response rates in national health surveys. In the

1960-62 National Health Survey, 80-89% of adults 35-74 years of age who were contacted responded to the questionnaire, but by 1971-74 the response rate had dropped to 65-73% for individuals 35-74 years of age. In the 1976-80 National Health and Nutrition Examination Survey, the response rate was between 62 and 71% depending upon age group. Older individuals were the least likely to respond (NCHS, 1986). Likewise, in industrial populations where participation might be expected to be high, response rates of only 70%, such as those obtained by Talbott and colleagues (1985), are commonly observed. In a recently conducted case-control study of 4,730 women with breast cancer and 4,754 controls selected from the population, only 83.4% of the selected controls agreed to participate. Although only 7% of the women with breast cancer or their physicians refused to be enrolled, 12.6% were lost to the study because of death, debilitating illness or inability to locate individuals (Cancer and Steroid Hormore Study, 1986; Wingo et al., 1988).

Although Schlesselman (1982) suggests several ways to assess the impact of nonresponse, Feinleib (1987) maintains that "...ultimately, it is a judgment call as to whether nonresponse might bias the outcome. One method is to suppose a worst case situation in assigning the nonrespondents to different outcomes in the exposure and nonexposed groups. Unfortunately, this usually suggests, even with relatively small nonresponse rates, that the study will yield little information." (p.156).

2.2.6.2 Confounding

Confounding is the over- or underestimation of an association between the study exposure variable and the disease outcome it is thought to cause, due to the action of a third non-causal variable, the confounder. In epidemiology, a confounder is an extraneous variable that satisfies two conditions: (1) it is a risk factor for the disease under study, and (2) it is associated with the exposure under study but is not a consequence of that exposure; that is, it is not an intervening variable in the causal pathway between exposure and disease. Only risk factors should be candidates as possible confounders since unnecessary adjustment can lower precision and may even introduce bias into a study. The data-based criterion for establishing the presence or absence of confounding requires comparison of a crude measure of association between exposure and outcome with an adjusted measure that corrects for distortions due to extraneous variables. Confounding is said to be present when the crude and adjusted measures differ in value (Kleinbaum et al., 1982; Schlesselman, 1982). Much contemporary epidemiologic and biostatistical literature addresses stratification and design-analytic approaches for detecting and controlling confounding bias (Schlesselman, 1982; Breslow and Day, 1980 and 1987).

In noise effect investigations, control for confounding is limited by practical considerations rather than methodologic ones. A major limitation is the difficulty of collecting sufficiently detailed data on potential confounders (such as family history and extraneous noise exposures)

from the two study groups to statistically control for confounding effects. This bias is especially common in historical prospective and case-control studies in which the exposures from the confounding variables occurred years in the past.

2.2.6.3 Misclassification Bias and Risk Estimates

Misclassification bias, also known as information bias, refers to a distortion in the estimate of the relative risk or estimated relative risk (odds ratio) due to invalid measurement and incorrect labeling of study subjects on the exposure and/or disease variable. This bias is a function of sensitivity and specificity of the classification procedure.[5] The severity of misclassification errors depends upon whether the misclassification is the same (nondifferential) or different (differential) in the two compared groups (Kleinbaum et al., 1982).

In the present context, the two most likely sources of misclassification bias are uncertainty and error in information available about noise exposure and disease status. Under the rubric of "detection bias," Feinstein (1988) discusses an important source of such uncertainty that is especially applicable to cardiovascular pathology such as hypertension and arrhythmias:

> "In nonexperimental studies where the outcome event is the development of a disease, rather than a change in symptoms, a different challenge occurs in diagnostic detection. Many diseases, such as cancer, coronary disease, and other major ailments, are regularly first found at postmortem necropsy examination, having been undiagnosed while the patient was alive. The previously undiagnosed diseases were rarely fatal, and usually occurred as co-existing 'silent' phenomena that escaped detection during life because they had not produced the overt manifestations that might evoke the appropriate diagnostic procedures in clinical or technological examinations....
>
> The existence of these silent cases of disease constitutes a formidable difficulty in epidemiologic research because any therapeutic or etiologic agent that is associated with increased 'medicalization' and increased use of diagnostic technology will also be associated with an increased detection of silent cases. Since these cases will be overlooked in people who do not receive the same diagnostic attention, the apparent increase in occurrence of the diseases may then be erroneously attributed to the agents, rather than to the detection process."

Although error of measurement of disease status can be large enough to bias estimates of relative risk, misclassification of health outcomes is not addressed further in this section.[6] In the past, less attention has been paid to characterization of exposure status than to characterization of disease status. In studies of environmental exposures, it is becoming increasingly clear that

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[5]Sensitivity is the ability of a measure to detect true exposure, while specificity is the ability of a measurement to detect true nonexposure.

[6]Nor are the effects of simultaneous misclassifications of both disease status and exposure, for which commonly accepted estimation procedures are not yet in widespread use.

misclassification of the exposure status of individuals is likely to occur unless the exposure of interest can be assessed by a biologic marker. Few environmental contaminants meet this criterion and residential aircraft noise exposure is certainly not among them. Recent studies of Agent Orange exposures in Vietnam veterans illustrate the difficulty of correctly determining exposure status based on records, place measurements and self-report (CDC, July 24, 1987). Furthermore, since measurements of disease occurrence are costly to acquire, they are worth matching with as accurate a measurement of exposure as possible to secure a maximally sensitive analysis.

The likelihood of misclassifying individual exposure status in typical community-based noise effect studies is obviously great (as described in Section 2.1 and Appendix A). A stationary outdoor noise monitoring instrument can hardly reflect the exposure of a heterogeneous, mobile population. Individual exposures vary with time spent in proximity to multiple noise sources in residential, occupational, and recreational circumstances, as well as with personal habits. Indoor sources of noise also contribute significantly to personal noise exposures. Inevitably, then, some individuals will be incorrectly classified as exposed or nonexposed.

In general, nondifferential misclassification of exposure between the diseased and nondiseased in a study produces bias in the relative risk (RR) toward the null value, making true effects seem smaller (i.e., decreases the power). Table 2-4 illustrates this tendency using incidence of hypertension of 26 per 1,000 in the nonexposed group and 52 per 1,000 in the noise exposed group, a true relative risk of 2.0, no selection bias, and no misclassification of disease status. Examples 6 and 7 suggest that lower sensitivity produces a slightly greater bias than lower specificity of the same magnitude. Thus, a sensitivity of .9 indicates 10% of the exposed are incorrectly classified as nonexposed, while a specificity of .7 indicates 30% of the nonexposed are incorrectly classified as exposed. If the sensitivity and specificity add to 1.0, the risk ratio will be unity for both cohort and case-control studies (Copeland et al., 1977). It is generally agreed that the rarer the disease, the greater the bias toward the null produced by the same value for sensitivity and specificity (Kleinbaum et al., 1982).

The direction of bias under differential misclassification may be either toward the null hypothesis (deflating the risk ratio) or away from the null hypothesis (inflating the risk ratio), as shown in examples 8 through 10 of Table 2-4. Bias toward the null can occur when there is misclassification among the diseased who are truly exposed but are classified as nonexposed (i.e., low sensitivity for classification of exposure of the diseased), or among the nondiseased who are truly nonexposed but are labeled exposed (i.e., low specificity of exposure of nondiseased) (Shy et al., 1978). Bias tends to be away from the null if diseased individuals are classified as exposed (as by residence in a noisy neighborhood) but have truly had little exposure, or if the nondiseased, apparently nonexposed, have indeed experienced high exposures.

Table 2-4: Misclassification of Exposure Status: Effect of Varying Magnitudes of Sensitivity and Specificity on Risk Ratios for True Risk Ratio of 2.0.

Part A: Nondifferential Misclassification					
Case No.	***Diseased***		***Nondiseased***		***Risk Ratio Observed***
	Sens.	*Spec.*	*Sens.*	*Spec.*	
(1)	.9	.9	.9	.9	1.73
(2)	.8	.8	.8	.8	1.50
(3)	.7	.7	.7	.7	1.31
(4)	.7	.8	.7	.8	1.39
(5)	.8	.7	.8	.7	1.41
(6)	.7	.9	.7	.9	1.50
(7)	.9	.7	.9	.7	1.56

Part B: Differential Misclassification					
Case No.	***Diseased***		***Nondiseased***		***Risk Ratio Observed***
	Sens.	*Spec.*	*Sens.*	*Spec.*	
(8)	.9	.7	.9	.9	2.31
(9)	.9	.7	.7	.9	3.39
(10)	.7	.9	.9	.7	0.69

Notes:

Calculations assume no selection bias, no misclassification of disease status.

P(Exposure) = .5 .

P(Disease) = .039; .026 in nonexposed and .052 in exposed.

Misclassification of noise exposure that produces bias toward the null can occur when those who develop disease are labeled nonexposed to noise, but work or spend leisure time in a high noise environment; or when those who are classified as living in the exposed area and free of the disease, actually have not been exposed because of housing construction and location, or from spending time outside of the area. Misclassification that could inflate risk estimates can occur when individuals living in an area exposed to high outdoor noise levels who develop disease have been protected from adverse noise by staying indoors, or by other means. Misclassification that could inflate risk estimates can also occur when individuals living in areas with lower outdoor noise levels who remain free of disease work and play in areas of high noise exposure.

Some measure of the sensitivity and specificity of the noise metrics used is needed to estimate misclassification bias due to exposure. This measure requires assessment of personal exposures to noise in a representative sample of diseased and nondiseased persons included in various studies. Means of developing such estimates of exposure misclassification are not presently available. Nevertheless, this potential bias demands consideration, especially in studies where the true associations are anticipated to be weak. Since distortion occurs when only one form of misclassification bias is considered in isolation from other types of bias, it is likely that even greater distortion in the true risk ratio may occur if misclassification in exposure, in disease status and in the confounding variables are considered simultaneously. Little research has been conducted on the effects on epidemiologic inference of several biases acting simultaneously (Greenland, 1980).

2.2.7 Summary of Epidemiologic Design Issues for Studies of Aircraft Noise Exposure

Epidemiologic study designs and related issues pertinent to aircraft noise research may be summarized as follows:

- The ecologic design or aggregate study involves the group as the unit of analysis, often a geographically defined area such as a county or census tract. The primary analytic limitation of an ecologic study is the lack of information about the joint distribution of the study exposure and the disease within each group. Although this design is well suited to exploratory research, it is of limited usefulness in inferring adverse health effects from noise exposures where the distribution of relevant confounding factors (family history of hypertension, alcohol consumption, obesity, cigarette smoking, etc.) are not available and the risk to be detected is not believed to be strong. Geographic associations between aircraft noise exposure and cardiovascular health may serve as crude indicators of potential risks until more definitive results are available.
- The cross-sectional design generates prevalence of health outcome data rather than incidence data, and is plagued by many biases such as selective survival. It is most useful for studying diseases with very short latent periods and for generating hypotheses. It may serve a useful purpose as a descriptive study preliminary to a

cohort investigation of aircraft noise and health since few (only 17) studies have focused on aircraft noise *per se*. Since about 87% of the traffic and industrial noise studies reviewed in the annotated summary were cross-sectional in design, it is unlikely that prevalence data will contribute further to the understanding of the relationship of noise and cardiovascular health.

- Adverse health effects attributable to specific exposures are best determined in epidemiologic studies which permit the assessment of cause of disease or health changes expressed in terms of risk or probability of disease occurrence under varying degrees of noise exposure. Imputation of causality in epidemiology requires, at a minimum, designs based on the individual as the unit of analysis, adequate measurement of both exposure and health status, evidence that the presumed causal factor (noise) precedes the disease in time, evaluation of the interaction of risk factors and control for confounding variables. Case-control and cohort studies can be designed to meet these criteria.

- It has been postulated that chronic exposure must accumulate sufficiently, perhaps as a function of duration and intensity, to trigger a step in the causal process. Thus, the initial exposure may not be a biologically effective one. The induction/latency period for the effect of aircraft noise exposure on the cardiovascular system is essentially unknown. Studies of high industrial noise suggest that continuous exposures over 5-10 years are required to trigger adverse blood pressure changes. Any study design must encompass the inductive/latency period if an effect is to be attributed to the exposure under investigation.

- When an association between cause and effect is weak (relative risks between 1.0 and 3.0) studies are prone to confounding bias and require attention to determination of adequate sample size and statistical power for detecting such small differences between exposed and nonexposed groups. Knowledge of weak associations are valuable when the exposure affects large numbers of people, the disease occurs frequently and the exposure is amenable to change.

- Large sample sizes are necessary, especially in heterogeneous populations, for detecting aircraft noises effects on health. For example, without accounting for confounders, minimum estimates of samples required for a cohort study to detect a risk of 1.5 to 2.0 range from 2,324 to 7,808.

- Misclassification of exposure and/or health status can distort any estimate of risk, especially weak ones. The likelihood of misclassifying individual exposure status with stationary outdoor noise monitoring is great since individual exposures vary with time spent in proximity to multiple noise sources (see Section 2.1). Aircraft noise studies must include techniques for augmenting place measurements to reflect, to the extent possible, estimates of individual exposures.

- Statistical control for confounding bias may be limited in noise effect studies by difficulty in collecting sufficiently detailed data on potential confounders in the exposed and nonexposed groups. Care must be taken not to control for true intervening variables which are a part of the causal pathway.

- Specific advantages of employing cohort designs in noise effects studies include: (1) observation of the exposure level of (individual) subjects at the onset of the

followup period before the health outcome occurs; the relative risk is directly estimable as a measure of the strength of the association between exposure and health outcome; (2) unlike case-control designs, several health outcomes can be examined within a single study; and (3) compared to case-control strategies, it is possible to make more precise measures of exposure unless past exposures have been carefully documented.

- Major disadvantages of cohort studies are the cost and time required for followup between exposure and health outcome and the large sample sizes required for adequate statistical power to detect a risk of about 2.0 suggested by cross-sectional studies to exist between noise and cardiovascular outcomes.

- The stringent requirements of the prospective cohort and retrospective cohort designs preclude their ready application to investigations of subsonic aircraft noise and sonic boom. The long-term followup of a specific community-based or residential cohort is particularly difficult because of the absence of a common record linkage number (i.e., a method for identifying individuals) or system for tracking address changes.

- The retrospective cohort study design may not be feasible for health evaluations of noise exposure due to: (1) inability to precisely define and locate data for a residential cohort who lived in the vicinity of the greatest noise; (2) difficulties related to the small size of exposed populations; (3) difficulty in selection of appropriate historical comparisons; and (4) difficulties in ascertaining health outcome data between/during the time of exposure and the end of the followup period for individuals who do not share a common health or employment facility (Kleinbaum et al., 1982).

- The major advantage of a case control design for studying aircraft noise effects is that it allows estimation of the relative risk from an exposure odds ratio without a long followup period between exposure and outcome. A basic premise of the case-control study is that cases and controls derive from a hypothetical population from which incidence rates can be estimated. Sampling of cases and controls must be independent of exposure.

- A case-control design may not be feasible for aircraft noise studies due to the dependence on records from the past for noise exposure information and to the need for place measurements of noise exposure which makes sampling of cases and controls independent of aircraft noise exposure very difficult.

- Nonepidemiologic studies of humans such as case reports, clinical and laboratory studies help describe the nature of health parameters under varying noise conditions, usually high level noise with short exposures. Both laboratory and animal model studies are needed to elucidate the mechanisms (the how) by which aircraft noise may actually produce health changes.

2.2.8 Need For Etiologic Approach

As stated in Section 1.1, the Air Force needs to be able to make technically valid and defensible predictions of the potential impact of military aircraft noise on the health of civilian populations. In order to make such predictions, it is necessary to document that the observed health outcomes are attributable to noise exposure *per se* and not to other environmental exposures or personal characteristics. By definition, human health research which purports to demonstrate adverse health effects associated with a particular exposure should meet scientific standards for inferring cause; otherwise, willful interventions on the exposure variable (noise) will not alter the health outcome.

In epidemiology, inferences about the adverse nature of exposures on health states are based on estimates of risk (the probability that some harm will come to an individual or population as a result of a particular exposure.) The *sine qua non* criterion for attributing an adverse health outcome to an environmental factor is that the exposure (noise) must be shown to operate in time prior to the disease or health state of interest. The measure of disease which is most sensitive to this criterion is the incidence rate, especially for chronic diseases with long latency periods. Epidemiologic study designs which allow documentation of the temporal sequence of presumed risk factor and disease are cohort, case-control and intervention (experimental).

The accumulation of epidemiologic evidence of adverse effects of an agent usually begins with case studies or less formal observations, and continues with ecologic and cross-sectional investigations which describe what exists in populations and hypothesize about relationships of variables. Etiologic studies build upon this descriptive research. The need for an etiologic approach to the study of putative health effects of military aircraft noise exposure is underscored by the fact that the preponderance of the literature that has accumulated over the past several decades on effects of noise exposure on nonauditory health has been based on cross-sectional and ecologic studies (cf. Chapter 3).

Furthermore, environmental planners are beginning to demand epidemiologic data appropriate for making adequate risk assessments for the effects of environmental contaminants other than noise. Risk assessment may be considered to be the systematic process of estimating the probability that some harm will come to an individual or a population as a result of a particular exposure. It contains some or all of 4 steps: (1) hazard identification: determining whether an agent is causally linked to adverse health effects; (2) dose-response assessment: determining the relationship between magnitude of exposure and occurrence of effects; (3) exposure assessment: determining the level, amount, duration of the factor to which humans are exposed; and (4) risk characterization: describing the nature of the risk to humans, including attendant uncertainty (National Academy of Sciences, 1983). Step 1, hazard identification, is

virtually identical to what epidemiologists describe as "judging causality" and thus requires data from studies designed to study cause-effect relationships, whether they be experimental, laboratory, animal or observational epidemiologic investigations (Erdreich, 1985).

The first 2 steps in any feasibility study are generally a determination of (1) which study designs available are applicable to the hypotheses of interest, and (2) the degree of inferential capability that is desired. Given the need of the Air Force to predict the impact of military aircraft noise on the health of overflown populations, etiologically oriented studies such as cohort and case-control designs are of primary interest. This approach is consistent with the opinion of Peterson, Augenstein and Hazelton (1984) that "...there appears no satisfactory substitute for long-term prospective studies involving either human or animal populations" (p. 58). The requirement for studies which can produce data that most strongly support drawing of causal inferences is also supported by prior recommendations, including the following:

1. The CHABA (1981) Working Group 84 concluded that ..."Appropriate studies for analyzing a combination of exposure conditions for possible interactive effects are in order...The appropriate studies will be difficult and expensive both in cost and in commitment of time by research personnel for data acquisition and analysis. To demonstrate that long-term exposure to high level noise *per se* is a risk factor for cardiovascular or other disorders, it is necessary that highly sophisticated *epidemiological studies* be conducted with controls for other known risk factors..."(p.4).

2. In proposing both retrospective and small-scale prospective studies, the Office of Noise Abatement and Control of the U.S. Environmental Protection Agency (EPA 1981, p.4.4) described the priority:..."The most urgent health and welfare research need, in support of Federal, State and local noise control requirements, is to verify as soon as possible the extent to which a cause-effect relationship exists between noise and stress-related disease such as cardiovascular disease, on the grounds that the scope of the potential public health problem is great."

3. Hattis and colleagues (1980) in a report to EPA suggested that in addition to large-scale cross-sectional surveys of blood pressure in relation to workplace and community noise, retrospective cohort studies be conducted with such populations as Framingham and the Baughn/General Motors groups (pp. 213-214).

4. In an assessment of the literature on noise effects for the Motor Vehicle Manufacturers Association of the U.S., Taylor, Young, Bimie and Hall (1980, pp. 113-114) concluded that available data were inadequate to determine whether transportation noise had detrimental health effects and recommended retrospective cohort studies prior to mounting a true prospective cohort.

5. After extensive review of existing research on the nonauditory health effects of noise, an interdisciplinary review team recommended that "priority needs to be given epidemiologic studies employing designs which offer the strongest evidence for associations that may be causal in nature," and detailed scenarios for several cohort studies (Thompson, 1981, pp. 4-42).

6. A prospective epidemiologic study of traffic noise is currently being conducted as a part of the ongoing Caerphilly and Speedwell Collaborative Heart Disease Studies in the United Kingdom (Babisch and Gallacher, 1988).

3. Review of the Literature on Adverse Health Effects of Noise Exposure

A large body of research treating the effects of noise on physical and psychological health has accumulated over the last several decades. Much of this research has concentrated on the adverse effects of high level, continuous noise exposure in the workplace, rather than on residential exposure to transportation (aircraft and traffic) noise. Although a few studies have suggested that changes in fetal development (i.e., birth defects and low birthweight) (e.g., Jones and Tauscher, 1978; Knipschild et al., 1981; and Ando and Hattori, 1977) and mental health problems (e.g., Abbey-Wickrama et al., 1969 and Meecham and Smith, 1977) may be related to noise exposures, the weight of the evidence suggests that if there are indeed adverse effects, the cardiovascular system is the most likely physiological system in which they might manifest themselves. A brief literature review and justification for exclusion of fetal development and mental health outcomes from current consideration may be found in Appendix B.

Several recent and extensive reviews of the literature on potential adverse effects of noise on the cardiovascular system are available (Welch, 1979; Hattis et al., 1980; Taylor et al., 1980; Thompson, 1981; Rehm, 1983; DeJoy, 1984; and Kryter, 1985). This report focuses on those studies most appropriate for assessing potential cardiovascular effects of aircraft noise exposure. A tabular update of the research reviewed by Thompson (1981) may be found in Volume III of this report.

3.1 Industrial Noise Exposure

Many cross-sectional studies conducted since the early 1960s, particularly in Eastern Europe, have shown that blood pressures of workers with long-term exposure to continuous, high level noise are higher than blood pressures of individuals working under less noisy conditions. Major studies of the effects of noise and hypertension which suggest adverse effects include those of Andriukin (1961), Meinhart and Renker (1979), Shatalov and Murov (1970), Parvizpoor (1976), Friedlander et al. (undated), Britanov (1979), Belli et al. (1984), Verbeek et al. (1987), and Wu, Ko, and Chang (1987). Most of these studies must be interpreted cautiously because of selection and temporal biases associated with cross-sectional designs, lack of standardization of blood pressure measurements, inadequate documentation of noise exposures, wide variability in noise exposures, and insufficient control of potential confounding variables.

For example, Andriukin (1961) reported that hypertension was found more frequently in workshops with intense noise than in the less noisy toolmaking workshop. However, noise

levels in all work areas studied were high: e.g., 93 dBA in the toolmaking area compared with 103-120 dBA in the high noise areas. The data were presented by 10-year age groups, but there is no evidence in the analysis of controls for age, sex, socioeconomic class, medications, co-morbidities, or environmental conditions such as temperature, humidity, shift work, etc. The author reported that morbidity increased with duration of employment, particularly after 5 years of work in the noisiest workshop, but failed to provide supportive data.

Parvizpoor (1976) conducted a survey of 812 male weavers from 3 textile mills with noise levels of 96 dBA and 412 randomly selected controls of similar socioeconomic status without occupational noise exposure. He concluded that men employed in textile mills had a significantly greater risk of developing hypertension than other workers, and that this difference appeared relatively early in life (30-39 years of age) and increased with length of employment. He noted that textile workers were also exposed to high temperatures, high humidity and heavy dust concentrations for which no provision was made during data analyses. Although the crude prevalence ratio (of hypertension for high noise exposures compared to low) of 4.0 suggests a strong relationship, noise measurements were undocumented. If one can accept length of employment as a correlate of noise exposure and duration as an indicator of dose, the data showed an increase in prevalence of hypertension with an increase in exposure. To accept this conclusion, one must also rule out high temperature, high humidity, and heavy dust as agents of hypertension.

A frequently cited study of Friedlander et al. (undated) purports to be an historical prospective design, but there is no evidence of the identification of a specific cohort with appropriate followup. Most probably this study is a cross-sectional study of the medical records of 441 male civilian employees of the U.S. Curtis Bay Shipyard (personal communication with author). No specific noise measurements were cited, although a previous noise survey had been conducted. Low noise was defined as less than 70 dB, intermediate noise level as 70-79 dB, and high level as greater than 80 dB. The risks reported in this study would indeed be impressive but for its methodological problems. The only statistically significant differences observed were for 35-44 year olds: the high noise group was at 6.4 times the risk of having increased systolic blood pressure and 2.8 times the risk of having increased diastolic blood pressure as the low noise group. No data on mean blood pressures were provided.

More recently, Belli et al. (1984) observed a very weak association (prevalence ratio 1.34) between noise exposure and hypertension after controlling for blood concentrations of triglycerides and blood glucose using the Mantel-Haenszel procedure. Other potential confounders controlled by stratification in this study were age and smoking habits. No data were reported on family history or alcohol use. Hypertension was defined as diastolic blood pressure greater than 95 mm Hg. Belli and colleagues compared 480 workers of a textile plant exposed to high frequency noise greater than 85 dBA--frequently exceeding 100 dBA--to 450 white collar and office workers who were considered "not exposed."

Verbeek, van Dijk, and de Vries (1987) also provide suggestive evidence (prevalence ratio 3) that long-term exposure of workers to noise exceeding 80 dBA may lead to increased blood pressure. Age, non-noise induced ear pathology, past exposure to explosions and past exposure to noise outside of present employment were controlled statistically. A major difficulty with this study is that all subjects, 428 males, were exposed to noise (78 to 98 dBA) and duration of employment in the production plant was used to indicate exposure status with respect to a referent group of 0-9 years employment.

Wu, Ko, and Chang (1987) provide reasonably strong evidence that long-term exposure of shipyard workers to noise exceeding 85 dBA as compared with exposure to noise of 80 dBA or less may result in higher risk for developing hypertension. Sixty-three workers who developed hypertension during their employment (mean duration = 7.25 $\pm$2.4 years) were matched to normotensives on age, employment duration and body mass index. Individuals who had a family history of hypertension, suffered high blood pressure-related disease, had documented hypertension prior to employment or were working in the 80-85 dBA noise area were excluded prior to the computer matching procedure. They report a doubling of risk of hypertension (RR = 2.38, $p < .05$) among workers who were exposed to the higher noise levels. Although a limited number of potential confounding factors were examined, the fact that only incident cases of hypertension were included increases the validity of the findings from this study.

Studies by Malchaire and Mullier (1979), Brown et al. (1975), and Lees et al. (1980) have failed to show a relationship between noise exposure and elevated blood pressure. Other evidence which tends to refute the hypothesis of an association between noise and blood pressure is derived from studies of hearing loss as a surrogate measure of noise exposure. Drettner et al. (1975), Hedstrand et al. (1977), Takala et al. (1977), Demeter et al. (1979), Lees and Roberts (1979), A. Cohen et al. (1980) and Delin (1984) found no correlation between noise-induced hearing loss and hypertension.

Similarly, Kent et al. (1986), in a retrospective review of medical records of 2,250 Air Force aircrew members evaluated at the USAF School of Aerospace Medicine between 1957 and 1980, failed to show an association between noise-induced hearing loss and blood pressure, hypertension or coronary artery disease. On the other hand, Jonsson and Hansson (1977) reported that men with noise-induced hearing losses had significantly higher blood pressures and a higher prevalence of hypertension (160/100 mm Hg) than men testing normal on routine hearing examinations.

Two recent investigations have failed to show an association between increased incidence of hypertension and prolonged exposure to high levels of noise when other risk factors were controlled. Talbott and colleagues (1985) compared 197 randomly selected male workers in a noisy (> 89 dBA) metal fabrication plant to 169 randomly chosen men from a less noisy (< 81 dBA) plant. Variables considered included height, weight, pulse, blood pressure, alcohol

consumption, smoking habits, family history of hypertension, previous noise exposure and hearing level. No reliable differences were observed in mean systolic or diastolic blood pressure between workers in the two plants. The two groups were similar with regard to factors other than noise which can affect blood pressure.

The researchers reported a strong relationship between severe noise-induced hearing loss (> 65 dBA at 3, 4 or 6 kHz) and high diastolic blood pressure (> 90 mm Hg) even after adjustment for other risk factors in older workers (56 years or older) in both plants. Noise-induced hearing loss was high in both the noisy and less noisy plants, 65% and 47% respectively. Among older workers, the prevalence of hypertension was twice as great in those with severe noise-induced hearing loss versus those without such loss. This positive finding must be interpreted with caution since it was derived from the analysis of a subgroup of small sample size that was not specified in the original hypotheses. In both plants the prevalence of hypertension did not increase significantly with age in those without noise-induced hearing loss. In the noisier plant, severe noise-induced hearing loss, noisy hobbies and traditional risk factors (alcohol, body mass index, age, smoking, family history of hypertension) explained 18.6% of the variability in diastolic blood pressure, while none of the noise variables independently predicted diastolic pressure when controlling for these risk factors in the less noisy plant. The only significant predictors of high diastolic blood pressure among the traditional risk factors were body mass index for persons working in the noisier plant and alcohol for men in the less noisy plant.

Although this study used state-of-the-art statistical analyses and controlled for basic risk factors of hypertension, the nonresponse rate was 30%, and procedures for measuring sound levels differed for the two plants. Furthermore, blood pressure measurements were cross-sectional, since no documentation of workers' blood pressures early in their work experience was available. Finally, the study suffered from low power since the sample size was smaller than that needed to detect a 5 mm Hg difference in mean blood pressures.

Similarly, van Dijk, Souman and de Vries (1987) observed that hearing loss increased with total exposure to noise but blood pressure did not. They examined 552 workers employed in production departments of seven industries and constructed noise exposure indices from personal noise dosimetry and duration of exposure. After correcting for age, relative weight, use of hearing protection and working conditions, blood pressure was not significantly related to total noise exposure. An unexpected finding was a weak negative association between noise annoyance and a stress index with the corrected blood pressure. These investigators interpreted this observation to indicate nonsupport for the hypothesis of a positive association between stress and hypertension.

3.1.1 Summary of Selected Studies of Industrial Noise

A summary of selected studies from industrial populations shows lack of comparability of data on which researchers have based the inference of an association between continuous, high intensity noise and elevated blood pressure. This summary suggests that, to date, these investigations should not be viewed as conclusive studies. Although the prevalence ratios for hypertension between high and low noise groups may be as high as 6.4 for special groups, they are based on small, selective samples and do not represent incident cases. For the most part, studies suggesting the strongest associations have not taken into account potential confounders and covariates as did the Talbott et al. (1985) and van Dijk et al. (1987) studies. Those differences in mean blood pressure which have been detected have been relatively small--about 0-9 mm Hg for systolic and 0-8 mm Hg for diastolic blood pressure (see Table 3-1).

Hattis and Richardson (1980) addressed the question "If it exists, how important is the blood pressure raising effect of noise likely to be in the context of overall cardiovascular disease?" (p. 123). They then developed a method to tentatively place the results of 11 different studies of long term noise exposure and the prevalence of hypertension on a comparable basis. Their analysis, based on many assumptions, suggests that for populations with long-term noise exposures between 85-100 dB, shifts relative to controls for men under 40 years of age will be about 3 mm Hg for systolic blood pressure and 2.5 mm Hg for diastolic blood pressure, and shifts for men over 40 years will be about 6 mm Hg for systolic blood pressure and 4 mm Hg for diastolic blood pressure (p. 136). Using some very preliminary and assumption-laden calculations of the increase in cardiovascular risks which would be expected based on the multiple logistic risk model and risk coefficients derived from the Framingham study, Hattis and Richardson (p. 215) estimated risk of cardiovascular disease to be 10% higher in a population averaging 6 mm Hg increases in systolic blood pressure (absolute increase of about 200 cases per 100,000 at risk per year). While this increase in cardiovascular disease with increase in small population changes in blood pressure seems impressive, one must be cognizant of the inadequacy of the studies used in the statistical modeling and the multiple assumptions on which the method of estimation used by Hattis and Richardson is based.

As shown in Table 3-2, variation in noise levels defined as high and low is wide. Some of the high levels in U.S. studies are less than the low levels defined in Eastern European studies. For most of these studies, noise is poorly characterized as to type, source, frequency composition, and duration. Ambient noise levels and the locations of individuals relative to the noise source are only infrequently specified. There is also a great deal of variability in the precision of blood pressure measurements across studies, not to mention differences in the definitions of elevated blood pressure. Blood pressure measurements are sensitive to a variety of environmental stimuli such as temperature and humidity as well as to psychological states.

Table 3-1: Study of Differences in Blood Pressure from High to Low Noise.

Study	*Groups*	*Systolic BP Diff. in mm HG*	*Diastolic BP Diff. in mm Hg*
Takala et al., 1977	ages 40-59	4.0 (ns)	4.0 (ns)
Jonsson & Hansson, 1977	NIHL vs none	12.6	8.0
Ising et al., 1979	ear prot. vs none	6.6	2.3 (ns)
Singh et al., 1982	mean age 32	5.1	6.6
Talbott et al., 1985	ages 55-59	-3.0 (ns)	-0.6 (ns)
	ages 60-63	3.4(ns)	-2.2 (ns)
Kent et al., 1986	ages 31-40	1.3 (ns)	0.8 (ns)
	ages 41-50	-0.3 (ns)	-0.7 (ns)
	ages 51-60	0.4 (ns)	-1.7 (ns)
Verbeek et al., 1987	10 vs 20 yrs exp	16.0	7.0
Babisch, Gallacher, Bainton, et al., 1988	total group	2.4 (ns)	1.1 (ns)
	no family HX	4.0 (ns)	NR
Babisch, Gallacher, Elwood, & Ising, 1988	ages 45-59	5.9	-0.4 (ns)
Ising & Michalak, 1988	girls ages 9-13	9.0 (NR)	3.4 (NR)
van Dijk, Verbeek, & de Vries, 1987	mean age 37	-0.3 (ns)	-0.8 (ns)

Notes: HX = History
NR = Not reported
NIHL = Noise-induced hearing loss
ns = Not statistically significant
exp = Exposure

Table 3-2: Summary of Prevalence Ratios for Hypertension and Noise.

Study	*Hypertension mm Hg*	*Noise in dBA High*	*Noise in dBA Low*	*Groups*	*Ratio High to Low Noise*
Cieslewicz, 1971	150/95	96-116	80-90	women	2.2
				men	2.7
Parvizpoor, 1976	NR	> 96	NR	ages 19-59	4.1
Jonsson & Hansson, 1977	160/100	NIHL	No HL	men	2.8
Pilaswka, 1977	NR	> 85	< 75	all workers	2.0
Knipschild, 1977a	175/100	NNI > 37	20-37	ages 35-64	1.7
Britanov, 1979	160/95	90	50-90	men+women	2.8
Von Eiff & Neus, 1980	self report	66-73	< 50	men+women	1.6
Friedlander et al.,	140/90	> 80	< 70	ages 35-44	6.4 SBP
					2.8 DBP
Kent et al., 1986	140/90	max. NIHL	min. NIHL	ages 31-40	1.2 (ns)
				ages 41-50	1.1 (ns)
				ages 51-60	1.2 (ns)
Brini et al., 1983	160/95	> 80	NR	men	0.9 (ns)
Belli et al., 1984	> 95 DBP	86-108	< 85	all ages	1.3
Talbott et al., 1985	>90 DBP	89	< 81	men	NR
		NIHL	no HL	ages 40-55	1.1 (ns)
		NIHL	no HL	ages 56+	2.0
Wu, Ko, & Chang, 1987	160/95	> 85	< 80	men	2.4[1]
Idzior-Walus, 1987	160/95	105-116	NR	men	3.7 SBP
					1.8 DBP
Verbeek et al., 1987	160/95	exposed	exposed	all workers	3.1
		> 19 years	< 10 years		

[1]Odds ratio from case-control study

Notes: NIHL = Noise induced hearing loss; NR = Not reported;
ns = Not statistically significant, otherwise significant
SBP = Systolic blood pressure, DBP = Diastolic blood pressure

To date, dose-response relationships between noise intensity and cardiovascular function cannot be adequately evaluated because studies have, at best, measured high and low noise areas rather than varying levels of intensity of exposure. Virtually all researchers have used length of employment as a surrogate for level of exposure. Length of employment is highly correlated with age, making it impossible to distinguish between age effects and exposure in most of the studies.

3.1.2 Reported Effects of Hearing Protection on Nonauditory Health

A reduction of the increase in blood pressure with use of hearing protectors or noise abatement measures in high noise environments would offer strong support for the hypothesis that noise adversely affects health. The literature offers little but disparate evidence of the influence of hearing protection on the reduction of blood pressure. Two of the earlier studies (Cieslewicz, 1971; Paranko et al., 1974) suggested that even when workers wore anti-noise helmets in noise greater than 85 dB, there was an increase in blood pressure with increase in length of employment in high noise. Van Dijk, Verbeek, and de Vries (1987) compared workers exposed to average noise levels of 98 dBA in a shipbuilding shop to workers exposed to 85.5 dBA levels in a machine shop. Seventy-four percent of the workers in the high noise area used protection most of the time, but only 14% in the low noise area used ear protection regularly. Although the authors state the personal hearing protection may have been effective in reducing hearing loss, 39% of the high noise exposed workers had hearing loss in excess of 40 dB, as compared with 9% of new workers in the low noise area. No difference in blood pressure was observed between the two groups after adjustment for age and relative weight.

In a second study of workers from 7 industries, van Dijk, Souman, and de Vries (1987) report that hearing protectors were worn by 33% of the workers, more so in exposure to high noise levels. At all levels except L_{eq} greater than 95 dBA workers who felt themselves more annoyed by noise used hearing protection more often than those who experienced less annoyance. Blood pressure, adjusted for age and relative weight, was not significantly associated with total noise exposure, and the specific effect of hearing protection was not determined.

Talbott et al. (1985) observed that in a noisy work environment (> 89 dBA) men wearing hearing protectors all or most of the time had mean blood pressure levels nearly equal to those reporting little use of hearing protectors. An earlier finding in the Raytheon study (1975) of workers in a large boiler plant has been interpreted as supporting the view that hearing protection ameliorates nonauditory health effects. Four hundred thirty-four workers in the high noise (95 dBA +) group and 432 workers in the low noise (< 80 dBA) group were compared on 9 categories of medical problems and symptoms for 2 years prior to and after implementation of a

hearing conservation program. Workers judged to have always used hearing protectors showed the greatest reduction in total problems. Those judged as never using hearing protectors experienced the smallest relative change in health problems. Although these differences failed to appear in the cause-specific data, the authors concluded that the program was beneficial in reducing various medical conditions. Shortcomings in the determination of hearing protector use and specification of health parameters, as well as a small sample size and short study period, raise doubts as to the validity of this conclusion.

The best evidence of the effect of noise reduction is provided in a pilot study by Ising et al. (1979) in which 12 subjects wore hearing protectors during work for 1 week and worked without hearing protection for 1 week. Workers wore dosimeters so that the reduction in daily noise level provided by the hearing protection could be measured. The actual reduction was between 10-16 dBA when averaged over the entire day. Ising and colleagues reported that when working without ear protection at a mean exposure of 95 dBA the systolic blood pressure was higher by almost 7 mm Hg than when working with ear protection. Whether these short-term changes generalize to long-term exposures has yet to be determined.

Although most of the studies of the effects of noise on workers have been concerned with the development of high blood pressure or hypertension, a range of other cardiovascular conditions and risk factors has also been studied. No consistent pattern of findings has emerged from these studies, as indicated in Table 3-3.

3.2 Traffic Noise Exposure

Results of studies of exposures to traffic noise do not provide much insight into the relationship between community noise exposure and health of general populations. Knipschild and Salle (1979) showed no associations between traffic noise and hypertension, angina pectoris, or ischemia among housewives 40-49 years of age living in a community with noisy streets (L_{eq} = 65-70 dBA) relative to those on quiet streets (L_{eq} = 55-60 dBA). Data were obtained from a cardiovascular screening program with hypertension defined as Systolic Blood Pressure (SBP) greater than 160 and Diastolic Blood Pressure (DBP) greater than 105 mm Hg at rest. The authors suggest the failure to observe a relationship between noise and cardiovascular disease in this survey could be the result of unequal response rates for the two exposure areas and confounding factors such as social class, coupled with the limited range in level of noise exposure between the two groups.

Investigations carried out in Bonn, Germany by Von Eiff and Neus (1980) on people living for more than 3 years on streets with either high traffic noise (L_{eq} > 66-73 dBA) or low traffic

Table 3-3: Selected Studies of Industrial Noise and Health Parameters Other Than Blood Pressure.

Selected Studies	
Study Author & Year	*Findings*
Ising et al., Germany, 1979	Working without ear protectors increased noradrenaline by 16% and decreased magnesium concentration by 5%
Raytheon, USA, 1975	Workers using ear protectors experienced the smallest relative change in health problems; no differences in cause-specific data
Brown et al., USA, 1975	No changes in BP, heart rate, cholesterol or glucose due to noise
Knipschild, Netherlands, (1977a, 1977b)	Prevalence of CVD increased with increase in noise levels; contact rates for CVD increased from low to high noise areas
Suvorov et al., Russia, 1979	Neurocirculatory impairment of 0.5% with each 1 dB increase in noise
Rumiantsev et al., Russia, 1971; Khomulo et al., Russia, 1967	Increase in B-lipoproteins in noise exposed group vs. control group
Lees et al., USA, 1980	No significant difference between high and low noise groups for new ischemic heart disease, myocardial infarction
Frerichs et al., USA, 1980	No increase in age-race-sex cause-specific deaths in noise exposed areas
Idzior-Walus, Poland, 1987	Increase in triglycerides, but not in cholesterol in high noise.
Kent et al., USA, 1986	No association between degree of noise-induced hearing loss and cardiovascular disease in aircrew.
Babisch, Gallacher, Elwood, Ising, U.K., 1988	Higher levels of serum total cholesterol, HDL, LDL, blood glucose and plasma viscosity in highest noise group.
21 Research Teams	Electrocardiographic changes - no specific patterns due to noise

noise (maximum of L_{eq} = 50 dBA) suggest potential adverse effects of noise. A random sample of 458 men and 473 women between the ages of 20 and 59 years was interviewed in the home. Only 5 persons failed to respond. Families with apartments above the second floor in the noisier area were excluded. Age and sex were controlled by stratification, but the groups differed in social class and smoking rates. The authors argue against potential confounding on these variables since reported hypertension treatment was not associated with smoking, income, alcohol or coffee/tea consumption. No blood pressure measurements were taken. While there were no differences in the reports of treatment for diabetes, asthma, or gastric ulcers, significantly ($p < .05$) more persons (22.8%) in the noisy area were treated for hypertension than in the quiet area (14.6%). Reported treatment for hypertension was dependent upon length of residence in the high noise area but not in the control area.

In a 2-year followup of 36 normotensive individuals (14 from the noisy area and 22 from the control area), residents of the noisy area exhibited a 10.7 mm Hg greater increase in DBP than those in the control area (Neus et al., 1983). As a followup to this prospective pilot study, all inhabitants who had moved to the respective noise areas up to 6 months before the screening procedures were selected for a longitudinal study (Otten et al., 1988). Ninety-seven women and 95 men between the ages of 20 and 35 years met the study criteria of no disorders of the cardiovascular system, no more than 2 months' absence from the residence per year, and no planned changes of residence. In the noisy area the average noise exposure (a 6 to 22 h equivalent level measured in front of buildings) exceeded 63 dB (range = 63 dBA < L_{eq} < 78 dBA). In the control area the average exposure level was less than L_{eq} = 55 dBA. Blood pressure, clinical examinations, electrocardiograms, family history and health behaviors, noise sensitivity and noise annoyance were assessed at 6, 12, 18, 24, and 36 months after the start of the study.

Mean blood pressure of males in the noisy area and males in the control area did not differ after four years. However, for males who did not move, both SBP and DBP increased throughout the followup period in both noise areas. Subjects who moved out of the noisy areas showed no increases in blood pressure but apparently tended to be younger. The authors interpreted the finding that an increase in noise sensitivity and annoyance in men was significantly correlated with the increase of blood pressure as well as the level of blood pressure as supporting the notion that blood pressure is influenced by psychological processes. No differences were observed in blood pressure changes for women, but women living in the noisy area described the noise as more intolerable and moved out of the area earlier than women living in the control area. This study clearly demonstrates the difficulty of conducting longitudinal studies of traffic noise effects, since 75% of the subjects moved out of the residential area within four years, and moving was correlated with noise sensitivity.

In yet another study of 56 men chosen at random from the two noise areas, Von Eiff,

Friedrich and Neus (1982) concluded that the extent of blood pressure reactions to noise seems to be determined by genetic rather than subjective factors of habituation. Blood pressure was measured while the men were submitted to 5-min stressor of mental arithmetic under "emotional" noise (90 dB) and to 30 min of experimental traffic noise (72 dB). The DBP rose beyond baseline values under noise but SBP rose and then dropped below baseline. During exposure to arithmetic problems and noise, men with a positive family history of hypertension showed higher increases in SBP as well as DBP than men without such family history ($p < .05$).

The data from the original investigation of 931 individuals were reanalyzed to examine the possible moderating effect of subjective reactions and attitudes toward noise on hypertension (Neus et al., 1983). An index of tolerability of noise or "subjective reaction" based on 4 items and 3 measures of attitudes--health, noise sensitivity and noise adaptability--was constructed by factor analysis.

Attitudes toward noise did not differ significantly between the high and low noise groups, but subjects living in the noisy area rated the traffic noise to be less tolerable than the control group ($p < .001$). In addition, residents not annoyed by traffic noise and living in the control area had a lower rate of antihypertensive treatment than in all other groups: 50.2% of the people living in the control area were in this nonannoyance group, whereas only 9.6% of those in the noisy area had similar scores for noise tolerability ($p = .007$). No differences between subjects with and without family histories of hypertension could be detected on noise attitudes or subjective reactions.

Pulles, Biesiot, and Stewart (1988) observed no significant relationship between traffic and aircraft noise level and blood pressure in a survey of 830 healthy Dutch individuals aged 20-55 years after multiple confounders were controlled. In support of their argument that psychological concepts of stress are relevant to the understanding of environmental noise and health, they noted that the data indicated that individuals exhibiting a coping style based on avoidance showed a higher noise sensitivity than those using other coping styles. The authors hypothesized that noise had to be appraised as unwanted, threatening, or annoying to induce adverse health effects; the appraisal may be dependent upon perceived loss of control and available coping strategies of the individual.

Neus et al. (1983) suggest that while experimental studies are insufficient for providing comprehensive information about the influence of noise on humans, they may be useful in beginning to investigate the effect of noise on blood pressure in interaction with other stressors. Such a study by Ising et al. (1980) indicated a significant increase of systolic and diastolic blood pressure under exposure to traffic noise (L_{eq} = 85 dBA) for 7 h when learning new work. A group of 50 men, 18- 34 years of age, when working under noise as compared with working without noise, had significant increases in total cholesterol, epinephrine, serum and urine magnesium but a decrease in erythrocyte-magnesium. According to Ising and colleagues, this

slight decrease in erythrocyte magnesium level, accompanied by an increase in serum magnesium (with a 15% increase in magnesium excretion in the urine), shows that cellular magnesium is decreased in the presence of noise. These researchers postulate that continued "stressful" noise exposure might lead to a greater magnesium decrease, with an attendant calcium increase in the myocardium, and an increased risk of sudden death ischemic heart disease.

Findings from two large prospective collaborative studies of heart disease may eventually contribute to understanding of the effects of traffic noise on cardiovascular risk. At present, cross-sectional data from these cohorts offer contradictory results which are difficult to interpret (Babisch and Gallacher, 1988; Babisch, Gallacher, Elwood, and Ising, 1988).

One cohort studied is composed of 2,512 men between the ages of 45 and 59 years who are on the electoral rolls of the small town of Caerphilly, Wales. Another cohort of 2,030 studied consists of a sample of men 45-59 years of age identified from the registers of 16 general practitioners working in the two health centers in the vicinity of Speedwell, a suburb of Bristol. Standardized methods were used for data collection. Health indices considered in both studies include an array of cardiovascular events such as myocardial infarction, ECG abnormalities, blood pressure and multiple risk factors.

Traffic noise levels have been carefully assessed by short- and long-term measurements in Speedwell and by short term measurements in Caerphilly to determine 6 to 22 h L_{eq} levels 10 meters from streets at each subject's home. The authors believe this measure to be a good general descriptor of the noise load of the subjects. Subjects were grouped in 5 dB categories of traffic noise level, ranging from 51 to 70 dBA.

A response rate of about 90% was obtained in both samples. The cross-sectional data from Caerphilly showed a slight increase in SBP (2.4 mm Hg) in the noisiest area as compared to the quietest area. These cross-sectional data also show the increase in systolic blood pressure on the Caerphilly Cohort to be more pronounced (4 mm Hg) if men with a history of hypertension and heart troubles were excluded. In Speedwell, the lowest group means for SBP were found for men in the noisiest area. The DBP, very low density lipoproteins, cholesterol, plasma fibrinogen and prevalent ischemic heart disease showed no association with noise. The HDL cholesterol was highest in the highest noise category (66-70 dBA), a finding inconsistent with the hypothesis that noise exposure increases the risk of heart disease. Glucose levels were shown to be higher in the noisiest category of exposure (Babisch and Gallacher, 1988; Babisch, Gallacher, Elwood, and Ising, 1988).

A quasi-random subsample of men in the Caerphilly cohort plus 84 men who lived on the noisy streets was selected to study the combined effects of traffic and occupational noise on cardiovascular health (Babisch et al., 1988). Noise exposure at work was determined by personal noise dosimeters worn for 2 days by each of 255 men in the study. Data were available

on potential risk factors including age, social class, relative body weight, smoking, alcohol, physical activity, and family history. The data showed that effects of traffic noise on blood pressure and blood lipids were more pronounced in subjects who were exposed to higher levels of noise at work (> 90 dBA) than in subjects who were exposed to lower levels of noise at work (< 90 dBA).

In summary, it appears that street traffic noise is generally not associated with increased blood pressure or other cardiovascular changes. Perhaps traffic noise levels are too low or too variable to have a measurable effect on physiological health. To date it has been impossible to adequately assess the level, frequency and duration of exposures and the many personal, social, and lifestyle conditions which determine the sounds that are actually heard by free-living populations.

3.3 Aircraft Noise Exposure

An association between exposure to infrequent sonic booms, aircraft flyover noise and health seems unlikely in view of the inconsistent and weak associations observed in studies of long-term exposure to industrial noise and traffic noise. However, a few studies of exposure to general aircraft noise in neighborhoods near airports have been conducted. Additionally, two studies have explored effects of workplace noise exposure on aircrew members.

3.3.1 Studies of Residential Aircraft Noise Exposure Effects

The most substantial research showing a positive association between exposure to aircraft noise and adverse health effects is that of Knipschild (1977a & b) and Knipschild and Oudshoorn (1977). These researchers conducted a general practice survey, a community cardiovascular screening survey and a drug survey in the vicinity of Amsterdam Schiphol Airport. In the cardiovascular survey, 5,828 people were given medical examinations of heart x-rays, ECG, height, weight and blood pressure by the same medical staff. Data were obtained as to angina pectoris (WHO standard questionnaire), smoking habits, medical treatment for heart trouble and hypertension and the taking of drugs. After the examinations, data were classified with regard to noise exposure in units of the Dutch measure B (Knipschild, 1977a). Corresponding values for DNL were later provided by Knipschild (1980) to indicate that noise exposure in the study area with "little" aircraft noise was 50-65 dBA, "much" aircraft noise was 60-70 dBA, and "very much" was 65-75 dBA.

Knipschild found that sex- and age-adjusted prevalence rates of medical treatment for heart

disease, use of cardiovascular drugs, pathological heart shape and hypertension (175/100 mm Hg and/or use of anti-hypertensive drugs) were significantly higher in people from the high aircraft noise area than in the lower aircraft noise area. Rates of angina pectoris and pathological ECG were slightly higher, but not significantly so, in the higher noise area. The most striking difference observed was for hypertension; the adjusted rate in the high exposure area was 15.2% compared to 10.1% in the low exposure area ($p < .001$). Particularly impressive is the evidence of a dose-response relationship indicating that for each increase in B of 3 units there was a 1% increase in hypertension rate. The weighted regression indicated that in an area of $L_{dn} = 68$ dBA the hypertension prevalence rate was twice as high as in an area of $L_{dn} = 55$ dBA.

There are several possible confounding factors in the cardiovascular survey. Although a stratified analysis showed the differences to be small, more participants in the high noise area smoked and were overweight than in the low noise area. Knipschild (1977a) believed that although there were indications that socioeconomic status in the high aircraft noise area was lower than in the less noisy area, the difference was not sufficient to explain the differences in health effects of the two groups. He also argued that the groups were comparable even though only 42% of those invited to participate took part in the study. There were no indications that aircraft noise was a reason for nonresponse. The noise survey was undertaken after the Central Bureau for Medical Examinations started a cardiovascular screening program and so was "double-blind" (Knipschild, 1980).

In the general practice survey in March 1974, 19 family physicians working in three areas near the airport were asked to register each of their patient contacts for a week. The population sizes in the three areas were known: 17,500 in the "little noise" area; 5,650 in the "much noise" area and 12,000 in the "very much noise" area. The contact rate was defined as number of patient contacts in a week divided by the population at risk. Contact rates adjusted for age and sex in Table 3-4 were reported by Knipschild (1980) for persons 15-64 years of age. Results for aged persons were excluded because one doctor in the low noise area visited a small clinic for aged persons and registered most of them for cardiovascular disease.

As Table 3-4 indicates, the findings of the general practice survey confirmed those of the cardiovascular survey in that the higher the noise in the area, the higher the contact rates for hypertension, cardiovascular disease, psychosomatic problems (low back pain, spastic colon, stomach complaints) and psychological problems. Knipschild considered the contact rate to be a combined measure for the prevalence rate and the consultation rate. The extent to which the contact rate accurately assesses total prevalence is unknown. As in the cardiovascular survey, the socioeconomic status of the low noise area was higher than in the other areas, and was uncontrolled.

Knipschild and Oudshoorn (1977) conducted a time-related study of the consumption of drugs (tranquilizers, sleeping pills, antacids and cardiovascular medications including

Table 3-4: Summary of Results of Knipschild's General Practice Survey.

Survey Results				
Aircraft Noise, L_{dn} in dBA	< 60	60-65	> 65	χ^2 *test for linear trend*
Size of Population at Risk	14,625	4,050	3,650	
Total Contacts	57.1%	79.7%	93.4%	$p < .001$
Contacts for:				
Psychological Problems	6.5%	11.3%	17.5%	$p < .001$
Psychosomatic Problems*	11.2%	15.4%	16.9%	$p < .001$
Cardiovascular Disease	4.6%	6.0%	8.2%	$p < .004$
With Hypertension	2.5%	3.1%	4.3%	$p < .030$

*low back pain, spastic colon, stomach complaints, allergic diseases, tinnitus, dizziness, headache.

Source: Knipschild (1980), p. 284.

antihypertensive agents) in the neighborhood of Schiphol Airport, to supplement the evidence from aircraft noise over the study period of 1967-1974, while a control village experienced no aircraft noise until 1969 when a new runway introduced noise on the order of $L_{dn} > 64$ dBA (B = 35-55). In 1973 all night operations ceased. Number of drugs purchased in a year divided by the adult population was used as an index of prevalence of disorders. The inhabitants in the noise area were a little older than those in the control village, but there were no changes in the age or socioeconomic distribution during the 8-year period. The data revealed that in the control area, the purchase of antihypertensive drugs and antacids remained at a constant level over the study period; in the exposed area, the purchase of antihypertensive drugs and antacids increased gradually up to twice the initial quantity (13 to 28 drug index). The authors state that no statistical testing was done because of the exploratory character of this drug survey.

Earlier work by Karagodina et al. (1969) of the effects of aircraft noise on the health of adults living near nine Soviet airports suggested that morbidity due to hypertension and hypotension, nervous disorders and gastrointestinal disorders was from two to four times higher in areas located near the airport (1-6 km) than in areas located away from the airport (40 km). No explicit noise measurements were reported in conjunction with this study. Likewise, Koszarny et al. (1976) reported significantly more complaints related to heart pain and a greater frequency of medications for heart problems, nervousness and headaches among women living in an area with noise greater than 100 dBA compared with a group exposed to noise of 80-90 dBA. Men who experienced unfavorable noise exposures in the work environment did not show similar effects. Mosskov and Ettema (1977a & b) claim that data from an experimental study of exposure of 12 subjects to 3 h of aircraft noise (peak 89-100 dBA) which showed an increase in diastolic blood pressure of 6 mm Hg supports Knipschild's findings.

Several studies do not support the hypothesis that aircraft noise exposure results in adverse cardiovascular effects. In 1974 Graeven reported that the correlation between level of aircraft noise exposures in five areas and number of health problems was in the hypothesized direction, but not statistically significant. This cross-sectional survey of 552 women reporting cardiovascular and other symptoms contributes little to the understanding of aircraft noise and cardiovascular health because of problems in self-selection, inadequate noise measurements, and use of reported symptoms without confirmatory measures.

Cohen et al. (1980) reported that children from noisy schools had slightly higher blood pressures than children from quiet schools. The mean peak sound level (an idiosyncratic measure of noise exposure that defies comparison with more conventional measures used in most other studies) for the noisy classrooms located in schools in the air corridor of the Los Angeles Airport was 74 dBA, compared with 56 dBA in the quiet school. Standardized blood pressure measurements were taken and controls were instituted for ethnicity, social class, race, weight and mobility, but not age. In a 1-year year followup study, Cohen et al. (1981) reanalyzed the cross-

sectional data for changes in blood pressure of children who moved from noisy classrooms to noise-abated rooms in contrast to changes in blood pressure of children who remained in noise-impacted rooms. On reanalysis, the association between noise exposure and increase in blood pressure could be accounted for by attrition and age differences. There were no significant differences between blood pressure observed for the 39 noise-exposed children who had experienced a year of noise-abatement and the continuously exposed children. A relatively high proportion of the noisy-school children had been lost to attrition.

The findings reported by Meecham and Shaw (1979) of higher death rates for stroke and cirrhosis of the liver due to noise exposure from the Los Angeles Airport have been refuted by Frerichs and colleagues (1980). Unlike Meecham and Shaw, Frerichs et al. compared age-race-sex specific death occurrences in the noise exposed and control areas and found no appreciable differences. Both studies must be interpreted with caution since they share problems associated with diagnosis and reporting of deaths and the ecologic nature of the data. However, the vital records mortality analysis of Frerichs et al. (1980) is more methodologically sound than the crude analysis by Meecham and Shaw (1979).

More recently, Meecham and Shaw (1988) reported an 18% higher cardiovascular disease death rate (Standard Mortality Rate 1.18) among individuals 75 years of age or older living in the 90 dBA noise contour around the Los Angeles airport compared to a similar age group living in nearby areas. No significant differences in cardiovascular mortality rates for 35-74 year-olds were found between the two noise areas. Although the groups were selected to be similar in race and income, and the data were age specific, there was evidence of rapid turnover of residence in the high noise area. Thus, selective migration as well as lack of adjustment for other risk factors may account for the observed differences between the noise areas. The study also suffers from inflated Type I error rate (cf. Section 5.2) due to multiple statistical tests. Since 5 separate tests of cardiovascular effects were done, 1 for each of 5 age groups, an appropriate level of confidence would have been 99%. By this criterion, there is no statistical reliable effect.

Anton-Guirgis and colleagues (1986) were unable to produce convincing evidence of a relationship between sonic boom exposure and adverse health effects in residents of the State of Nevada, where supersonic flight operations have been carried out since 1969. Data on morbidity by 5 categories (cardiovascular, hypertensive, cerebrovascular accident, cancer and other causes) were collected from annual hospital discharges from 20 of the 33 licensed hospitals in the state. Of the 13 hospitals declining to participate, 10 were in the high noise area, 2 in the moderate noise area and 1 in the low noise area. It is unclear whether agreement to participate was associated with degree of noise exposure. Mortality data were obtained from Nevada vital record tapes and population files.

Estimates of the sonic boom environments in Nevada from 1969 to 1983 were based on analysis of historical records of supersonic operations of both fighter and SR-71 aircraft,

including information about the total sorties flown per year per aircraft type and estimates of numbers of supersonic events per sortie. The estimated annual C-weighted DNL due only to sonic booms was spatially averaged over each township in the state (Kamerman et al., 1986, p. 62). The space-averaged L_{Cdn} values ranged from 0 to 56 dB in the townships. The average sound level for each township across the period of 1969-1983 was calculated by dividing the sum of L_{Cdn} values across years by 15. Townships with an average L_{Cdn} greater than 36 dB (upper one-third) were classified in the high area[7]; those with average sound levels less than 31 dB (lower one-third) were classified in the low noise area; and the remaining townships were grouped in an intermediate exposure area. For county level estimates, data for townships were weighted by two methods: by event (sonic boom) and by population. Measures of peak pressure, carpet area and L_{Cdn} generated by these two methods were very similar for each county (Anton-Guirgis et al., 1986, p. 11).

Since township populations could not be partitioned by race, sex and age, adjusted morbidity/mortality rates could not be estimated. County level data showed that crude rates of mortality increased systematically from low exposure to high exposure areas. However, no consistent relation was observed for overall mortality or for cause-specific mortality when the rates were stratified by sex and adjusted for age. A trend analysis over time showed that noise exposure as measured by L_{Cdn} increased over the period. Over the same period there was a decline in all-cause age-adjusted mortality, inconsistent changes in age- adjusted cardiovascular, hypertension and cerebrovascular accidents and an increase in cancer mortality. Bivariate linear regression between sonic boom exposures and mortality revealed no consistent pattern of relationships for any cause.

Several problems are apparent in the Anton-Guirgis et al. (1986) study, including:

- difficulties in relating mortality and morbidity data to small geographic areas such as townships;
- absence of empirical measurement of noisc exposure;
- difficulty in obtaining and relating population data by age, race and sex to the desired township exposure unit leading to use of a larger unit, the county (which may have diluted any effect); and
- the usual difficulties in deriving meaningful inferences from ecologic analyses of vast geographic areas where the population at risk is relatively small.

The first report of effects of rapid onset time noise exposure from high speed, low-altitude flight is that of Ising and Michalak (1988). Ising and Michalak studied effects in the elderly and in school children of increases in noise levels of 30 dB in less than 0.5 s and L_{max} values above

[7]Wind noise alone is capable of producing L_{Cdn} values of comparable magnitude.

110-115 dBA. In the first study, 24 volunteers between 70 and 88 years of age were exposed via earphones to two types of recorded flight noise: noise with level increases of 30 dB within 4 s and noise with level increases of 30 dB within 0.4 s. Persons with conductive hearing losses greater than 10 dB at one or more frequencies between 0.25 and 4 kHz, or with an acute disease or unstable blood pressure were ineligible for the study. Significantly higher blood pressure increases were observed in response to earphone presentation of the rapid onset time flight noise than to the more gradual onset time noise.

In a second study, cross-sectional data were obtained from 430 nine to thirteen year olds in villages within a 75 m low level flight area and in a neighborhood in a 150 m low level flight area. Maximum noise levels reached 125 dBA in the two communities. The girls' mean SBP, but not the boys', was 9 mm Hg higher in the area in which lower altitude flights were permitted. Since few details of the study are reported, it is difficult to evaluate these findings in the context of chronic exposure to low level flights.

3.3.2 Workplace Studies of Aircraft Noise Exposure Effects

Two studies of aircrew workers, primarily pilots, have not greatly clarified the nature of the relationship between aircraft noise exposure and cardiovascular disease. No changes in blood pressure, heart rate, cholesterol or glucose levels were found in a historical study, utilizing cross-sectional data from annual examinations of 29 pilots and 29 non-flying controls (Brown et al., 1975). Although pilots and controls had both had at least 8 years in the executive physical examination program, no measurements or estimates of actual noise exposure were available for the two groups.

Kent and colleagues (1986) recently used hearing threshold levels as an indirect measure of past noise exposure to examine the effects of noise on blood pressure and clinically diagnosed cardiovascular disease in 2,250 Air Force aircrew members. Subjects were referred from 1957 to 1980 to the USAF School of Aerospace Medicine for evaluation of borderline conditions potentially disqualifying them from flying duty. The actual noise levels for classifying individual exposures were not available but cockpit noise levels ranged from 89 to 102 dBA suggesting that the total cohort had relatively high exposure over a period of time. Since these subjects were referred for borderline conditions, it is not clear that they represent a total cohort of individuals exposed to various levels of aircraft noise, nor that they represent all aircrew personnel who develop cardiovascular disease. The extent to which hearing protection was used was not known. Analyses that included multiple regression, t-tests and analyses of variance showed no significant differences in blood pressure or cardiovascular disease between those with minimum hearing loss and those with maximum hearing loss when the data were stratified by 10-year age groups.

A major factor detracting from these two studies is that of inadequate sample size, especially after stratification, which increases the probability of a Type II error (failing to declare two population means significantly different when in fact they are different). In studies of special groups such as pilots, proportionately more healthy than unhealthy men remain in the program, making it especially important to follow the original cohort to account for all adverse health outcomes.

3.4 Implications of Reviewed Literature

Only tentative and speculative conclusions can be drawn from the existing research about the effect of aircraft noise on human health. Even the best of this work, such as the studies conducted by Knipschild (1977 a & b), fails to provide clear evidence of an adverse effect of aircraft noise on humans. These two studies suffer from temporal bias, or lack of data to show that the noise exposures preceded the health events, and from inadequate data to control for potential confounding variables such as socioeconomic class, which could have accounted for the differences observed between the high and low noise exposed groups. The drug study (Knipschild and Oudshoorn, 1977), although a time dependent investigation, utilizes a surrogate measure (use of drugs) for the health outcome rather than new hypertensive or cardiovascular events. The extent to which drug usage actually represents evidence of new disease in a community is unknown.

Studies of mortality data and noise exposure for large geographical areas such as those of Frerichs et al. (1980) and Anton-Guirgis (1986) are of limited utility in making causal inferences for several reasons:

- the larger the population units under study, the less complete and accurate the data generally are;
- since the quality of data is not under the investigator's control, data are often incomplete or inaccurate; and
- it is virtually never possible to exclude with confidence the possibility that the observed relationship between noise exposure and disease is explained by unmeasured agents or variables highly correlated with noise. This fundamental uncertainty makes it difficult to isolate the effects of noise on the health outcome.

4. General Process Model

This chapter develops a general process model that identifies independent, intervening, and dependent variables that can arguably be linked to form hypothetical causal chains between aircraft noise exposure and adverse effects on human health. Two requirements must be met for Air Force environmental impact analyses to present convincing predictions of health consequences based on causal chains of reasoning. First, a cause-effect relationship must be shown. Second, a quantitative dosage-effect relationship must be established that shows amount of change in health outcomes as a result of amount of change in exposure.

A simplified version of the model is presented schematically in Table 4-1. As suggested in the table, one or more aspects of aircraft noise exposure, acting in conjunction with the influences of some number of confounding variables, may affect or be modified by one or more intervening psychological or physiological processes in a manner that eventually produces one or more adverse health consequences.

A useful general process model requires specification of the aspects of noise exposure, the confounding variables, the intervening and mediating psychological and physiological processes, and the health consequences in sufficient detail that testable hypotheses can be stated. Constructing a detailed process model is difficult because of (1) the diffuse nature of the inferences that can be drawn from the literature on health effects of noise exposure (cf. Chapter 3); and (2) the paucity of evidence that noise exposure exerts a measurable influence on extra-auditory health. As an initial step in this direction, however, it is possible to list variables in each of the just mentioned categories as in Table 4-1 so that their relationships may be explicitly discussed.

A simplified schematic version of the model is presented in Figure 4-1. Arrows between boxes represent hypothesized direct effects. Arrows that intersect other arrows represent influences that moderate or interact with such effects. The model depicts a number of intervening variables between exposure to aircraft noise and disease states. It also depicts a number of points in the chain where the process can be altered by moderating variables. Each link in the process model and each moderating point is predicated on published findings, some of which are described briefly in the following pages. Studies concerned with the effects of stress on health generally study links between one of two of the boxes in the diagram. The process model in Figure 4-1 is accordingly based on assumptions about the most likely ways in which all of the parameters might be connected.

Starting at the left, the model depicts noise exposure as leading to and individual's subjective appraisal of the meaning of the noise intrusion (arrow 1). Exposure to aircraft noise is

Table 4-1: Examples of Potential Variables in Causal Chain Linking Noise Exposure and Health Consequences.

Examples of Variables		
Independent Variables	*Confounding and/or Mediating Variables*	*Health Consequences*
total noise exposure number of aircraft noise intrusions onset time of aircraft noise intrusions noise exposure above threshold distribution-sensitive measures (peak, centiles)	genetic predisposition (race) (family history) behavior (type A behavior) annoyance disease states (diabetes) (hypertension) (hypertensive H.D.) occupational and recreational noise exposure conventional cardiovascular risk factors (cigarette smoking) (cholesterol, LDL, HDL) (alcohol) (sedentariness) (diet: high sodium) (obesity) (age) (sex) enhancers (excessive autonomic activation) (psychological stress) (annoyance)(low control over stressor) anticipated threat negative affectivity neuroticism buffers (social support) (high control over stressor) (type B behavior) (background noise) (sex)	arrhythmias hypertension sudden cardiac death myocardial infarction mental illness acute depression reproductive outcomes (low birthweight) (teratism)

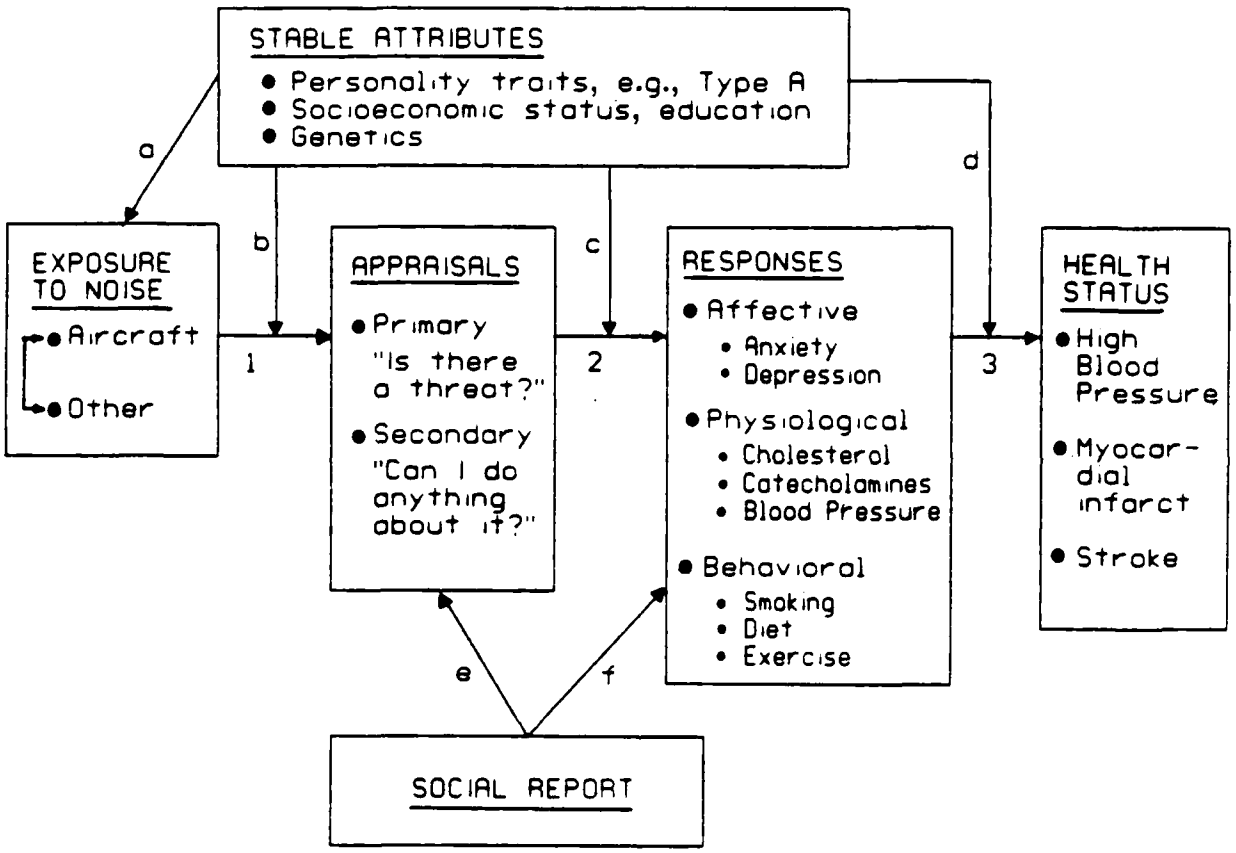

Figure 4-1: Model of Potential Chain Between Noise Exposure and Hypertension.

distinguished from exposure to other forms of noise, and the model allows for the possibility that the two forms are correlated (double arrow between "aircraft" and "other" in the leftmost box.) For example, the two forms of noise exposure could be correlated if low education and income increased the likelihood that a person would live and work in environments characterized by exposure to aircraft and non-aircraft forms of noise (cf. Fidell, 1978.) Statistical controls for the confounding effects of exposure to other forms of noise and self-selection into noise environments would be necessary in cases where low socioeconomic status was associated with elevated risk of cardiovascular disease, as is the situation in North America (Marmot, 1982). Allowing for exposure to noises other than those from aircraft or to noise at a prior time opens up the possibility that such exposure may condition individuals' appraisals of aircraft noise and vice-versa. Such relationships are omitted, for ease of presentation, from Figure 4-1.

Following Lazarus' theory of stress appraisal (Lazarus and Folkman, 1984), stressors such as noise are subject to two types of appraisal. Primary appraisal considers whether the stressor is a threat to a person's needs, whereas secondary appraisal considers whether the person is able to do anything about the noise. Under the rubric of primary appraisal one might further evaluate whether the noise is perceived as expected or unexpected (and hence, potentially startling). Secondary appraisal combines two types of assessments.

One type of secondary appraisal is the individual's assessment of the values of potential outcomes that might result by taking specific actions to limit or terminate the effects of the noise. For example, the actions of (1) wearing protective hearing devices, (2) insulating living and working space from sound, (3) finding employment in a quieter environment, and (4) telling oneself to ignore the noise could all be evaluated by the person against outcomes such as: (1) the benefits of quiet, (2) the disruption of social activities, and (3) the costs of resettlement. The other type of secondary appraisal is the perceived likelihood that the person is able to successfully take the considered action.

These two elements are usually multiplied together (intersection of arrows 1 and c) in most theories of motivation aimed at predicting coping and other forms of behavior (e.g., Fishbein and Ajzen, 1975; Atkinson and Feather, 1966). The effects of stressors on both primary and secondary appraisal may vary due to stable predispositions to view stimuli in negative or positive terms (e.g., Eysenck and Mathews, 1987).

Appraising noise exposure as a threat to either current or future well-being (e.g., a threat to the future ability of the person to sell a home at a reasonable profit) and feeling helpless to respond, according to the model, is hypothesized to lead to negative affective responses such as fear, anxiety, depression, and anger (arrow 2). Other potential sequelae include physiological reactions such as elevated catecholamines (Frankenhaueser, 1980), biochemicals thought to play a role in the production of atherosclerosis, and changes in behaviors which have cardiovascular consequences--for example, changes in cigarette smoking (Nesbitt, 1973). Studies of repeated

exposure to harmful situations from which individual cannot escape and in which the individual's sense of efficacy is undermined suggest that depression may be generated (Abramson et al., 1978; Seligman, 1975) and that the immune system may be weakened (Laudenslager et al., 1983).

These affective, physiological and behavioral responses may vary as a function of personality characteristics associated with physiological reactivity (such as Type A or hardiness) (Friedman et al., 1970; Kobasa and Puccetti, 1983), and the prior physiological condition of the person (intersection of arrows 2 and c). The effect of this constellation of responses on disease states (arrow 3) may also depend on genetic and other preexisting conditions of the person (arrow d). Such responses may also vary as a function of social support, which as noted below, may buffer the effects of objective stressors by helping people reassess the meaning of such stressors and search for meaningful ways of coping with them (arrow c; Thoits, 1986), and provide palliative comfort during times of emotional upset (arrow f).

If the person is startled, affective sequelae (fear), physiological sequelae (elevated heart rate, for example), and behavioral sequelae (startle response) may occur without or prior to conscious appraisal (Kryter, 1985). Further specification would be required to determine the rate at which adaptation to originally-startling stimuli occurs and to map the relationship between adaptation and indicators of physical and emotional well-being.

In sum, a complex set of hypothesized linkages between exposure to aircraft noise and disease states may exist . Assuming (1) that such complexity exists, (2) that the effects expected at each link are weak to moderate (e.g., accounting for 4 to 20% of the variance in each variable at the end of an arrow), and (3) that scientific procedures of acceptable reliability and moderate validity are available to assess such complexity, it would be very unlikely that one could detect any direct, simple link between exposure to aircraft noise and disease.

The implications of this complexity for developing an ideal study are discussed in Chapter 5. The remainder of this chapter presents a discussion of the components of the model and the complexity of the relationships among the variables within the model. The discussion also considers to a limited degree the biological plausibility and existing evidence or the hypothesis that noise can act as a stressor mediated by psychological and physiological processes to produce adverse cardiovascular health effects. The individual components of the model are first discussed separately in the next sections of this chapter. The reader should not lose sight of the relationships among the model elements during the initial discussion, since these relationships are essential for deriving hypotheses about the association between aircraft noise and health outcomes.

4.1 Aircraft Noise Exposure as an Independent Variable

As suggested by the examples of the attributes of noise (the independent variables) in Table 4-1 and the discussion of Section 2.1, noise is a complex environmental stimulus that defies satisfactory quantification for present purposes in a single number index. The reasons for this difficulty and the implications of these reasons are most simply demonstrated with respect to one of the most common measures of aircraft noise, an integrated measure of exposure.

Sound exposure is a measure of the accumulation of the sound intensity of a series of noise events occurring within a specified period. In community noise analyses, it is common practice to specify average sound levels (often called "equivalent" sound levels) instead of sound exposure. The average sound level is the sound level in decibels obtained by dividing sound exposure by a specified time interval. The average sound level is sometimes modified by application of "corrections" (either penalties or licenses depending on one's viewpoint) to exposures occurring during more-or-less arbitrary times of day (Fidell and Schultz, 1980), or by limitation of energy integration to time periods during which absolute levels exceed a threshold (Gjestland, 1988; Gjestland and Oftedal, 1980).

It is implicit in such representations that noise exposure *per se*, or at least some quantity highly correlated with noise exposure, is the agent responsible for creating an effect. For example, the use of DNL to predict prevalence of annoyance in a community is based on the assumption that annoyance due to aircraft noise exposure is related to the total amount of noise energy produced in a community by aircraft operations (rather than to peak levels or numbers of operations).

The reasonableness of characterizing noise exposure by an energy-related measure thus depends on the nature of the effect of interest and the mechanisms believed to create the effect. In the case of an adverse health effect, characterization of aircraft noise exposure in energy-related terms requires an assumption that adverse health effects of exposure are mediated by a psychological or physiological process for which there is some evidence of a dependence on integrated noise energy.

So little is known about mechanisms which might convert aircraft noise into adverse health effects, however, that it is not clear what assumptions are reasonable. Might teratism or myocardial infarcts, for example, be products of individual, startlingly high noise events rather than of cumulative, long-term exposure? Are there bases for believing that some other adverse health effects are created not by the total or average energy produced by aircraft operations, but rather by specific numbers of occurrences, or peak levels, or duration alone, or onset times of individual flyovers? If so, would it not be more reasonable to represent aircraft noise by measures more sensitive to these parameters?

Since the literature reviewed in the preceding chapter provides so little guidance about an optimal measure of noise for purposes of predicting adverse health consequences, it is important to make explicit the assumptions needed to establish a possible causal chain between a measure of aircraft noise and a measure of an adverse health effect. Only after these assumptions are stated can the difficulties attending quantification of personal noise exposure be addressed (cf. Chapter 2).

4.2 Short Term vs. Long Term Responses as Outcomes

Noise exposure could conceivably activate mechanisms in individuals which produce biologic responses resulting in physiological adaptations within a short time. Noise exposure could also activate mechanisms that produce more lasting responses which progress to a variety of pathological states. The occurrence of either of these outcomes could depend upon the combinations and/or levels of situationally-specific mediating variables.

Only defined disease states are considered as outcomes in Table 4-1 for several reasons[8]. First, measurable disease states are more amenable to study using epidemiologic strategies in large populations than are reversible short-term reactions; ultimately, disease end points are of interest to the general public for intervention purposes. Secondly, even though it is now possible to simultaneously measure biochemical and hormonal responses (such as changes in blood cholesterol, triglycerides and catecholamines), patterns of response to specific stressor events are poorly understood. It is not yet possible to establish a threshold or critical level separating those changes which have significance for health from those which do not, since it is not known at what point reversible physiological changes begin to make a significant contribution to an eventual disease process (Herd, 1983; Julius and Cottier, 1983).

Although genetic influences may contribute to blood pressure regulation under varying noise levels, the extent of the genetic influence may depend on the behaviorally-induced psychophysiologic state of the individual. Such a mechanism would be consistent with the notion postulated by Herd et al. (1987) and Krantz et al. (1987) based on animal and clinical studies that genetic predisposition, diet, and emotional behavior may act synergistically to produce a sustained increase in blood pressure. A synergistic mechanism would mean that noise as a stressor must be studied simultaneously with psychological and physiologic factors and specific environmental circumstances to differentiate effects of confounding and intervening variables.

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[8]Although mental illness and reproductive outcomes are listed in Table 4-1 as examples of potential consequences of noise exposure, they are not considered in further description of the general process model for reasons presented in Appendix B.

4.3 Intervening, Mediating and Confounding Variables

Difficulties encountered in quantifying the action of noise as a potential stressor are compounded by the multiplicity of ways that people can react to noise. Because the effects of noise on people are not mediated exclusively by physiological processes, psychological processes may also in principle contribute to pathogenesis.

Thus, the effects of noise as a stressor on bodily systems could vary in ways that reflect interactions of intervening and mediating variables that could either accentuate or minimize ("buffer") health outcomes. To adequately judge the contribution of noise to clinical disease, the effect of noise on each he alth event must be viewed in relationship to known strong risk factors for that particular outcome and for other potential confounding variables. A confounding variable is an extraneous one that wholly or partially accounts for the apparent effect of an agent under study. For example, any association between living in a noisy environment and cardiovascular illness could occur because both outcomes may be coincidental results of low income and education which are associated with elevated risk of cardiovascular disease in North America (Marmot, 1982).

The action of confounding variables makes it difficult or impossible to accurately determine the effect of independent variables of interest. Since confounding requires consideration of both causal associations believed to be operative in a study population and data-based associations, it is difficult to determine *a priori* all possible confounders of a given hypothesis. Factors intervening in the causal pathway between exposure and disease are not necessarily confounders, since their control can eliminate or reduce any manifestation in the data of a true association between noise exposure and disease.

Practically speaking, it is difficult to distinguish intervening and mediating variables from risk factors which may confound a relationship of interest, especially when there are complex interactions among the factors. Kleinbaum et al. (1982) suggest a cautious approach of retaining as potential confounders all risk factors that are not obviously intervening variables. There is considerable overlap between intervening and confounding variables in Table 4-1 since the literature provides so little guidance for making this differentiation. It should also be noted that a variable confounding one hypothesized relationship may be a strong mediator for another relationship. For example, genetic predisposition may in itself increase risk for hypertension (e.g., Blacks show a predisposition toward hypertension). That predisposition may also dictate a stronger hypertensive reaction in the presence of noise. Noise exposure levels that cause no harm in the absence of the predisposition are accentuated in those with the predisposition, elevating blood pressure even more. When hypertension is found in an ecologic study, it is difficult to determine whether the outcome has occurred as a result of the genetics-hypertension path or as a result of the noise-genetics- hypertension path, or a combination of both.

Accordingly, in an association between noise exposure and hypertension, family history may be an important link in the causal pathway; that is, noise exposure may be important only in persons of certain genotypes. Therefore, rather than statistically controlling for family history of hypertension, stratifying on family history and examining the relationship within each stratum may be more appropriate in identifying a true association.

4.3.1 Potential Effects of Psychosocial Variables

A number of psychosocial variables have been identified as potential moderators which reduce the effects of environmental stressors on human health outcomes such as coronary heart disease, anxiety and depression. They include social support (Thoits, 1982; House, 1981), sense of internal control (Kobasa and Puccetti, 1983), and actual control over the onset, duration, and termination of the stressor (Cohen, 1980; Glass and Singer, 1972; and Seligman, 1975). Other potential moderators include: Type A behavior pattern (Friedman and Rosenman, 1974), neuroticism (Zyzanski et al., 1976; Haynes and Feinleib, 1982), cognitive blunting of potentially stressful inputs (Miller and Birnbaum, 1988), and negative affectivity (a tendency to view all phenomena in negative terms) (Watson and Clark, 1984).

Social support is of potential interest because longitudinal large-sample panel surveys of communities have suggested that support reduces the risk of mortality from all diseases (House et al., 1982; Berkman and Syme, 1979). This finding remains after controlling for numerous potentially confounding variables including family history of cardiovascular disease, blood pressure, serum cholesterol, and cigarette smoking. In addition to direct effects on health, social support buffers the effects of life events on mental health (e.g., Cohen and Wills, 1985; House, 1981). Research on the beneficial roles of social support has been examined with regard to a number of potential stressors including occupational work load, loss of employment, and illness. There has been little research, however, on the interplay between noise exposure, social support, and health outcomes. Herridge and Chir (1972) found that psychiatric hospital admission rates were higher in noisy areas of a community for women who were single, widowed, or separated, suggesting that loss of social support might exacerbate noise-related illness (or, that noise might exacerbate illness due to lack of social support). The study failed to control for demographic characteristics of the noisy and quiet neighborhoods and used inappropriate indices of noise. Replication of the study with better measures and controls failed to produce significant results (Gattoni and Tarnopolsky, 1973).

Research on neuroticism, Type A, negative affectivity, and blunting is similarly deficient. Like Type A (Rosenman et al., 1975), neuroticism is associated with elevated risk of cardiovascular disorders (Haynes and Feinleib, 1982; Zyzanski et al., 1976). Neuroticism is also associated with the inability to elicit social support (e.g., Coyne, 1976; Coyne et al., 1987). No

research has been conducted, however, to determine if neuroticism has effects on health which are independent of those for social support or if neuroticism might mediate any relation between exposure to noise and coronary heart disease and its risk factors. Low blunters/high monitors are defined as persons predisposed to monitor external cues of environmental threat and internal physical symptoms. In response to experimentally induced negative and uncontrollable situations, these persons show greater and more sustained physiological, subjective, and behavioral arousal than high blunters/low monitors (e.g., Phipps and Zinn, 1986). These cognitive styles have not been examined in studies of the effects of noise on well-being.

The mediating effects of background noise have been explored in several studies. Neus, Ruddel and Schulte (1983) found that self reports of intolerability to noise were correlated with treatment for high blood pressure within control areas, but not within residential areas with relatively high levels of traffic noise. Changes in blood pressure over a 2-year period were positively associated with ratings of noise annoyance only within the lesser impacted areas. This study suffers in that it compares large areas which differ in ways other than noise exposure. The comparison by Cohen et al. (1981) of the effect of aircraft noise at school on systolic and diastolic blood pressures of children from quieter and noisier homes failed to demonstrate moderating effects of background noise.

In addition to the parameters just discussed, moderate alcohol consumption appears to exert a protective effect on coronary heart disease (but not hypertension), possibly by lessening the atheroma, increasing HDL cholesterol or decreasing platelet aggregation (Marmot, 1984). Several studies have been unable to confirm such findings. Fraser and Upsdell (1981), using a community-based register of acute coronary events in New Zealand, found high alcohol consumption associated with a higher risk of sudden death, especially in the presence of myocardial infarction. They suggest that adverse effects attributable to alcohol are often manifested by death before hospitalization so that survivors would tend to have a lower consumption pattern. Thus, in studies of hospitalized and/or community based patients with heart disease, moderate consumption may spuriously appear protective.

Both epidemiologic and applied physiology studies provide evidence that exercise favorably alters coronary heart disease and some of its risk factors (Leon, 1985). Present evidence also indicates an inverse relationship between exercise and levels of blood pressure (Blair et al., 1984; McMahon and Palmer, 1985). There are no studies on whether exercise and noise exposure are correlated. Consequently, there is no empirical basis for knowing whether exercise might confound the relationship between noise and illness.

4.3.2 Effects of Noise on Sudden Cardiac Death and Myocardial Infarction

Two major types of coronary heart disease (CHD) in the United States are myocardial infarctions and sudden cardiac deaths (deaths occurring within 24 h of onset of cardiac signs and symptoms). Plausible physiological mechanisms by which life stressors such as noise exposure might induce sudden cardiac death remain to be established. However, many investigators believe that cardiac arrhythmias are involved. Eliot and Buell (1983) describe the contributions of (1) coronary artery disease, (2) electrical instability of the heart, and (3) myocardial disorders to the occurrence of sudden cardiac death. These three factors develop over time and ultimately are brought together by behavioral influences mediated through the central nervous system. Herd (1983, p. 251) summarizes the links between risk factor behavior and underlying mechanisms described by Eliot and Buell as follows:

> "...(1) hypertension increases local vascular turbulence with special hydrodynamic impact at vascular branching sites and thus tends to induce mechanical trauma; (2) serum lipids, both from diet and mobilized from body stores by neuroendocrine processes, foster smooth muscle cell proliferation, and become incorporated in the proliferative and necrotic lesions; (3) smoking behavior, through its nicotine effects on catecholamines and also the effects of carbon monoxide upon vascular permeability, tends to promote processes contributing to atherogenesis; (4) platelet mobilization and aggregation in areas of endothelial trauma release a platelet-derived growth factor and the vasospastic agent, thromboxane A2, contributing to smooth muscle cell proliferation in combination with vasoactive constriction. Catecholamines appear to be significantly involved at several points in these pathophysiologic processes. Their secretion is heavily influenced by the central nervous system and linked to behavior. .....there is a role for highly stressful experience in the precipitation of acute clinical events, including fatal arrhythmias. It is hypothesized that such events as the unexpected death of a loved person can cause such a massive outpouring of catecholamines that coagulative myocytolysis occurs, with concomitant vasospasm or myocardial infarction (MI)."

Eliot and Buell (1983) describe this response among those who experience sudden death as literally an overdosing on one's own catecholamines. In general, the evidence suggests that these reactions to stressors must interact with pre-existing cardiac pathology to produce disease, although some studies show that acute massive psychosocial stress can apparently precipitate sudden death or ultimately fatal arrhythmias in the absence of significant atherosclerosis (Lown et al., 1978).

Lown et al. (1977) demonstrated that an emotionally disturbing interview can evoke arrhythmias from patients with a recent myocardial infarction, even though no arrhythmias were present prior to the interview. Studies of patients who had previous ambulatory electrocardiographic monitoring for the detection of arrhythmias have concluded that ventricular arrhythmias are correlated with cardiac death and sudden death but are not nearly as important a correlate as congestive heart failure (Rocco et al., 1987). Thus underlying congestive heart failure may confound relationships between noise and arrhythmias and sudden cardiac death.

Other studies suggest a potential relationship between stressors, arrhythmias and sudden death. It has been demonstrated that a constant slow heart rate in middle-aged men increases the risk for sudden death (Hinkle et al., 1972); that post-myocardial infarction patients with the greatest tendency to bradycardia in response to a startle have the poorest prognosis (Schneider, 1957) and that ventricular tachycardia can be elicited by vagal stimulation in the ischemic heart (Kerzner et al., 1973). Ventricular arrhythmias in otherwise healthy individuals without evidence of cardiovascular disease have not been associated with poor prognosis (Crow et al., 1981).

As noted in Chapter 3, previous studies of noise exposure and cardiovascular disease other than hypertension have failed to show meaningful associations, possibly due to poor study methodology or small sample sizes (e.g., Capellini and Maroni, 1974; Meinhart and Renker, 1979; Raytheon Report, 1975). Studies of immediate cardiovascular responses to noise in humans have produced somewhat vague and conflicting results, but many have been conducted by Eastern European investigators who have not described their methodology adequately for evaluation.

Bradycardia and other cardiac rhythm irregularities in response to noise have been noted by Jansen (1961), Cuesdean, (1977), Lanzetta et al., (1979), Singh et al., (1982) and many others, but no consistent patterns of response have been identified. Virtually none of these studies considered potential influences of confounding factors. Field investigations of noise have shown increases in both epinephrine and norepinephrine (Andren, 1982; Cesana, 1982; Ising and Melchert, 1980). No epidemiologic studies of noise effects have differentiated between sudden cardiac deaths and myocardial infarctions.

In summary, results from both field experiments and epidemiologic studies clearly show that the simple stimulus-response stress concept cannot sufficiently explain the complex biochemical, physiological and disease state relationships observed in these investigations. By extension, it is most unlikely that useful hypotheses can be generated that directly link aircraft noise exposure to adverse health consequences without consideration of complex roles for an indeterminate number of several types of intervening variables considered to be risk factors.

4.3.3 Intervening Risk Factors

The strongest risk factors for coronary heart disease are known to be hypercholesterolemia, obesity, hypertension, Type A behavior, sedentariness or lack of exercise, positive family history, diabetes, hypertensive heart disease, and cigarette smoking (Eliot and Buell, 1983; Kuller et al., 1986; Kannel and Thomas, 1982). The positive association between blood cholesterol levels and cardiovascular incidence and mortality is one of the most consistent and well established of all areas of epidemiologic inquiry.

Even though there are probably many important lipid-related risk factors (such as the protective effect of the omega 3 family of polyunsaturated fatty acids in fish oil) yet to be discovered, most epidemiologic studies of large populations could at best consider only the lipoproteins--high density (HDL), low density (LDL) and very low density (VLDL)--and total cholesterol. While HDL shows an inverse dose-response relationship, VLDL, LDL and total cholesterol increase the risk of coronary heart disease, exerting a stronger influence on myocardial infarction than on sudden cardiac death when other factors are controlled (Pearson, 1984; Wallace and Anderson, 1987; Stamler, 1979; and the American Health Foundation, 1979). There is limited evidence that stressful situations which have been related to coronary risk may operate by altering blood cholesterol levels (Van Doorman and Orlebeke, 1982).

Obesity seems to contribute independently to sudden cardiac death but only contributes to coronary artery disease when some other risk factor is present (Eliot and Buell, 1983). Although cigarette smoking has been identified as a strong risk factor for both myocardial infarctions and sudden cardiac deaths, its action may occur through means other than atherogenesis. Kuller and colleagues (1986) interpret data which show little increased risk of heart attack among smokers living in countries with low intakes of saturated fat and cholesterol and relatively low serum cholesterol levels as suggestive that cigarette smoking acts primarily as a precipitant of heart attack rather than acting on the progression of atherosclerosis. According to Kuller, only in the presence of a critical degree of coronary artery disease does smoking become a major precipitant of heart attacks, exerting its action by increasing thrombosis or precipitating ventricular fibrillation.

Some question the definition and significance of Type A behavior pattern in coronary heart disease (cf. Matthews, 1988 and Friedman and Booth-Kewley, 1988), but a panel of scientists convened by the National Heart, Lung, and Blood Institute has agreed that it confers a risk of developing clinically apparent CHD over and above that imposed by age, systolic blood pressure, serum cholesterol, and smoking. This risk appears to be of the same order of magnitude as the relative risk associated with any of these other risk factors (Weiss, 1981).

4.3.4 Effects on Blood Pressure

It is generally accepted that emotional stimuli and stressors can raise blood pressure. Experience of stressful conditions such as high level noise exposure or challenges to perform a variety of behavioral tasks have been shown to elevate blood pressure for short periods (Julius and Cottier, 1983). From such experiments evolved the notion that repeated emotional elevations of blood pressure may eventually lead to sustained hypertension. In the aggregate, available data on prevalence of elevated blood pressure in noise exposed populations suggest that long term, high exposure to occupational noise may be associated with an increase in

hypertension in later decades of life (cf. Table 3-2). The evidence of adverse health effects from traffic and aircraft noise is even more tenuous (cf. Chapter 3).

Although mechanisms whereby stressors produce hypertension remain unclear, several have been proposed. One of the more plausible mechanisms is that stimulation of the central nervous system induces changes in cardiac function and peripheral vascular resistance, which in turn results in a rise in blood pressure and gradual resetting of the baro-receptor control system. The development of hypertension may also result from sympathetic nervous system stimulation of the renin-angiotension system with modification of the responses by the prostaglandins and kallikrein-kinin systems (Hunt et al., 1980; Hattis and Richardson, 1980).

Factors found to be associated with hypertension in epidemiologic studies include obesity (Chiang et al., 1969), high sodium diet (Walker, 1980), family history of hypertension (Feinleib and Garrison, 1979), exaggerated cardiovascular response to psychological stimuli (Herd, 1984), diabetes (Barrett-Connor et al., 1981) and alcohol consumption (Klatsky et al., 1979; MacMahon, 1987). Type A behavior does not appear to be associated with essential hypertension (Shekelle et al., 1976). Although several studies have revealed significantly lower blood pressure in smokers or ex-smokers than nonsmokers (Seltzer, 1974), the effects of smoking on blood pressure remain controversial due to its confounding with weight (Friedman et al., 1982). While smoking may be a confounding factor because of its inverse association with obesity, it is not likely to be a significant intervening variable for the study of noise exposure and blood pressure since changes in blood pressure have not been found to be associated with quitting cigarette smoking (Gordon et al., 1975). Adjusting for the effects of obesity should minimize much of the potential effect of smoking on blood pressure.

4.4 Cardiovascular Parameters as Dependent Variables

Coronary heart disease and hypertension are insidious processes requiring many years or even decades to develop to the point of manifest symptoms. In researching effects of noise on health, noise has been examined primarily as an isolated stress component even though it is generally acknowledged that an individual continually experiences a large number of stressors. The use of a stress index score in a study of noise in industry and blood pressure by van Dijk, Souman and de Vries (1987) is an exception.

The amount of noise stress which will result in a rise in blood pressure or a worsening of ischemia apparently differs for individuals. Such stress depends, inter alia, on the physiological and psychosocial state of the individual and the distribution of many behavioral characteristics which become known as risk factors because of their association with the disease endpoints of

the process. Noise appears to elicit many of the same biochemical and physiological reactions as do other stressors. The long latency period for manifestation of symptoms makes it very difficult to delineate the general role of stress in cardiovascular and hypertensive diseases.

Epidemiologic studies of disease endpoints are based on the premise that all individuals can indeed be accurately classified as to clinical disease. However, the study of clinical disease is beset with multiple and complex problems. Clinical cardiovascular disease is almost always associated with severe coronary arterial stenosis, but many individuals have severe coronary artery stenosis without obvious clinical disease (Kuller, 1976). Kuller goes on to make the following observations about the inability to accurately define heart disease, a problem that seriously impedes epidemiologic inference.

Because of the difficulty in explaining and identifying the relationship between clinical and subclinical disease, many individuals with extensive coronary artery stenosis but no clinical disease may be (and frequently are) classified as so-called controls in studies. If the ratio of clinical to subclinical disease is low, as in the United States, it is very difficult to separate cases and noncases solely on the basis of clinical criteria.

The problem of appropriately classifying cases and controls is made more difficult by the following set of circumstances:

- diagnostic tests for coronary artery disease are usually referenced to coronary angiography (a highly invasive procedure);
- only a small fraction of individuals suspected of having heart disease are actually referred for angiography; and
- those who are referred often are not typical of all others with underlying disease nor of the general population.

Accepting the position of Kuller and others that clinical heart disease is almost always associated with atherosclerosis or stenosis, then it follows that in the absence of coronary atherosclerosis those factors related primarily to the development of overt disease are of minimal importance. Then, according to Kuller, the maximum difference in risk factors (such as noise) or highest relative risks will be found when the frequency of atherosclerosis is low among noncases (those not diagnosed as diseased).

The degree of coronary atherosclerosis in the general population is unknown and unmeasurable, but possibly high at present. The comparison is therefore between individuals who have both coronary atherosclerosis and clinical disease (the study outcome of present interest), and controls who have varying amounts of atherosclerosis and no recognizable disease. Because of the unclear relationship between subclinical stenosis and clinical disease, a control can become a case any time after classification without any change in underlying disease.

Under the same premises, factors that are primarily related to the onset of clinical disease rather than to the underlying atherosclerosis would not be associated with increased risk of disease in population groups with little atherosclerosis. For these and other reasons, accurate classification of disease states becomes critical in the study of potential risk factors (such as noise) which are probably associated with relatively low risks, if any at all. When relatively low risks are further masked by misclassification of the exposure status, the probability of detecting any differences between noise exposed and nonexposed populations becomes minuscule.

Since the causal mechanism for potential cardiovascular effects of noise exposure is unclear, and since the intervening pathways are as yet unidentified, one might hypothesize that noise exerts an effect only after a certain degree of atherosclerosis has developed or when there is a predisposition to atherosclerosis due to a combination of strong known risk factors. Likewise, it could be hypothesized that noise leads to an increase in atherosclerosis due to some stress-related causal mechanism.

A similar argument could be made for hypertension. Hypertension has been defined by statistical deviations in the population based on an indirect blood pressure measurement. Even though no other clinical method has been used as long or as frequently as the auscultatory method of blood pressure measurement, and even though standardized measurement procedures are well documented, interpretation of results is more difficult than appears at first sight. Many studies in which results of the noninvasive method have been compared with invasive determination of blood pressure have concluded that the auscultatory method can show large errors, particularly in obese persons (Labarthe et al., 1973).

Other problems in defining hypertension for study purposes stem from the fact that blood pressure varies considerably over time, and any study obtains a momentary value only. Averaging repeated measures of blood pressure might improve accuracy of classification, but averaging is often difficult to accomplish, since repeated measurements on separate days are usually prohibitively expensive in large population based studies.

Perhaps of greater importance in identifying cases and non-cases in population based studies is the fact that so little is known about the disease labeled hypertension and its presumed risk factors. Patients with different types of hypertension may present symptoms with different etiologies and pathogenesis. Few studies have examined the underlying dynamics for the associations found with such factors as obesity and high sodium diets. Just how noise as a stressor might relate to different hypertension types is unclear.

As with atherosclerosis and heart disease, it may be that noise is only a significant factor in the development of hypertension for what has been called a "noise sensitive" group of individuals. Whether this hypothetical sensitivity is related to a genetic predisposition, to a high sympathetic system reactivity, to proneness to increased alertness, or to some other factors, is

unknown and difficult to study. A few investigators (e.g., Talbott, 1985) have suggested that noise sensitivity may explain why some people exposed to high noise levels develop no apparent consequences, while others react markedly. This suggestion is consistent with the work of Feinleib and Garrison (1979), which suggests that 50-82% of the total variance for blood pressure may be genetically determined. Efforts to associate noise sensitivity and personality types (e.g., Shigehisa and Gunn, 1979) have been uniformly unpersuasive.

It has been demonstrated that of the 60-70% of coronary heart disease deaths that occur outside of a hospital, two-thirds are classified as sudden, having occurred within 24 h of onset of symptoms (Kuller et al., 1986). One could argue that noise eliciting a startle response may act primarily as a precipitant of clinical events--that is, noise may only bring to light a disorder previously subclinical or previously known, but tolerated or controlled. If noise were a strong precipitant of cardiovascular events, particularly sudden cardiac deaths, one would expect to see an increase in such events with an increase in noise exposure over time. However, evidence is accumulating suggesting a decline in sudden coronary heart disease deaths, especially in persons without preexisting heart disease, may account for much of the decline in CHD mortality. A decline in sudden cardiac deaths but not in myocardial infarctions has been documented over the past 10-15 years (Kuller et al., 1986).

As previously described, arrhythmias have been correlated with cardiac death but have been highly dependent upon the presence of existing disease. According to Rocco and associates (1987), it is not clear why ventricular arrhythmias appear in persons who die suddenly; is it because of electrical instability regardless of the degree of myocardial dysfunction, or is it because arrhythmias are correlated with prognosis merely because they are markers of the extent of underlying damage? The slowly accumulating evidence of arrhythmias in apparently healthy people which do not result in myocardial dysfunction makes it difficult to judge the significance of arrhythmias in the general population as a health outcome of noise exposure.

Many stressors and factors including exercise have been shown to trigger the so-called benign arrhythmias. The argument against using arrhythmias as a long-term outcome of noise exposure in the general population is supported by the fact that the specific mechanism through which noise may be linked to arrhythmias is yet unknown. In the presence of other strong risk factors (such as cigarette smoking) which probably serve as precipitants of ventricular fibrillation, it is not likely that weak associations can be observed in epidemiologic data.

On the other hand, any stressor (including noise) which stimulates episodes of arrhythmias in a poorly functioning heart may be worthy of further investigation. Although ambulatory monitoring methods are currently available for studying arrhythmias in individuals outside of hospitals, several problems may make their study impractical. These include:

- problems in selecting the types of underlying disease to include in the study population and finding such populations;

- difficulties in determining the appropriate time relative to the disease process for monitoring;
- the duration and repetitions of the monitoring episodes required for adequate measurement;
- problems in monitoring the timing and duration of arrhythmias in relation to the noise event; and
- following individuals long enough to detect meaningful outcomes. Followup time may not need to be long in selected populations if the Rocco et al. (1987) data (showing an average of only 38 months' followup) is representative of other patient populations.

In addition to the problems inherent in defining and reliably measuring health outcomes, difficulties in measuring potential confounding variables plague epidemiologic studies. As confounding factors are added to the model to allow detection of a noise effect, the probability of misclassification biasing the risk ratio rises precipitously. Serious measurement problems exist for virtually all of the "known" risk factors (e.g., cholesterol level) described previously. For example, variation among laboratories in cholesterol determinations has been shown to result in important variation in risk estimates even though a standardization program is feasible. A related issue noted by Wallace and Anderson (1987) is the accuracy of procedures for estimation of lipoprotein levels not directly determined, such as LDL, which are estimated by means of a formula based on total cholesterol, triglyceride and HDL values.

Assessments of smoking exposure (Sepkovic and Haley, 1985), obesity (Lee et al., 1981), and diet (Willett, 1987) all suffer from measurement problems which may lead to spurious study conclusions. For example there is no way that is both reliable and practical to measure an individual's sodium intake. Intra- and interindividual differences in the rate of sodium excretion as well as day-to-day fluctuations in sodium intake further confound the data analysis.

4.5 Potential Mechanisms for Noise-Induced Stress

The current status of theories suggesting potential mechanisms whereby noise may influence the cardiovascular system has been reviewed in detail by Hattis and Richardson (1980). Cardiovascular and hypertensive disease processes are believed to be the result of chronic cumulative pathological processes in response to adverse conditions. These processes appear to consist of chronic accumulations of incompletely repaired or misrepaired small-scale damage. The rate at which these processes occur depends in some way on the amount of time over which the individual experiences elevated levels of risk factors.

As the cumulative pathological processes progress, a vicious circle may ensue if a control mechanism is impaired or if the system is made more responsive to perturbations in some way, as by genetic predisposition. Homeostatic processes clearly play a prominent role in the day-to-day and year-to-year regulation of cardiovascular functioning. Overt clinical disease is manifest only when major departures from homeostasis occur. This sort of chronic accumulation of individually insignificant damage does not fit within the framework of massive short-term breakdown of adaptive mechanisms.

This theory of pathogenesis is consistent with the notion that cumulative noise exposure may produce a large number of "stress events," some of which may exceed the adaptive threshold and over time produce physiological changes in the cardiovascular system and blood pressure regulation.

According to Hattis and Richardson, sympathetic nervous stimulation in general, and emotional responses to stressful stimuli and responses to brief exposures to loud noises in particular, can trigger dangerous types of ventricular arrhythmias in hearts which have been rendered electrically unstable by a variety of conditions (p.166). In these situations, noise onset time, peak levels and number of startle episodes may be more important than total noise energy in hypothesizing relationships between noise and cardiovascular disease.

After a review of literature on nonauditory-system response to noise and effects on health, Kryter (1985, p. 506) argues for an indirect pathway through psychosocial factors:

> "Many sounds (or noises) can indirectly (nonreflexively) cause autonomic- system reactions that are deemed physiologically stressful. These are sounds which create feelings of emotion (startle, fear, anger, frustration, etc.) in the listener because of the unexpectedness or other meanings the sounds convey or because of the annoyance caused by interference with sleep, with rest, or with the hearing of wanted sounds, or both.
>
> Experimental evidence demonstrates that autonomic-system responses that are probably stressful occur only after conscious or unconscious cognitive processes are completed. That is to say, sound or noises are not inherently aversive or a cause of physiological stress except to the ear. The findings indicate that autonomic-system responses that might be physiologically stressful and appear to be associated with noise are due to psychological factors related to the situation or to the experiment."

Such a conclusion implies that noise is not inherently harmful, but only potentially harmful if it is found to be annoying. Such a conclusion increases the complexity of evaluations of the health consequences of aircraft noise by adding the challenges of measuring potential mediating and intervening psychological processes to the complexities of assessing exposure to noise and links between physiological responses and disease. The process model implies that an adequate evaluation of the link between aircraft noise and physical health will need to include the assessment of cognitions regarding threat, control, and helplessness; health-related coping; and

traits such as neuroticism and Type A. Such an evaluation will require a sample large enough to allow for the possibility that noise exposure that is potentially harmful to one person is merely noise to another.

4.6 Statement of Specific Hypotheses

Specific hypotheses are required before the start of a well designed epidemiologic study to avoid the consequences of a practice termed "data dredging" by Feinstein (1988). Data dredging is the generation of hypotheses by hindsight. As Feinstein notes,

> "In many epidemiologic studies...vast amounts of information can be assembled. It can include demographic data (age, race, sex, socioeconomic status), data about individual agents (diet, smoking, alcohol, environmental exposures, pharmaceutical substances, other treatments), and data about individual outcomes (birth defects, stroke, heart disease, cancer, death).
>
> "With modern electronic computation, all this information is readily explored....A large number of statistical associations are explored in an automated manner for diverse individual groups, agents, and outcomes. The groups can consist of all the people under study, or demographic divisions having one, two, or more than two separating characteristics (such as men and women, old men and young women, or old poor black men and young rich white women). Within each group or subgroup, each of the multiple individual agents is statistically associated with each of the multiple individual outcomes. Whenever a 'statistically significant' result emerges during the myriads of computation, the event may be proposed as a cause-effect relationship." (p.1259)

Many such results are, of course, spurious artifacts that are likely to arise by chance alone in the course of conducting multiple simultaneous tests of association.

The selection of viable hypotheses to test by epidemiologic strategies should be governed by several criteria in addition to general scientific principles and the desire to focus on noise exposure and health outcomes of public health concern. More specifically, the disease state or health condition must be measurable for large populations and must be measurable in a relatively noninvasive manner. If the health outcomes of interest are subject to confounding by variables other than noise exposure, these other variables should be known and measurable. Plausible intervening pathways should have been identified through clinical and/or laboratory studies. Other practical considerations include the accessibility of population health information, the need for samples of adequate size, and the availability of population health information on a time scale appropriate to the study of either acute or chronic disease.

Furthermore, since the goal is to draw causal inferences, only the most biologically plausible hypotheses should be investigated. In the case of environmental exposures, causal

inference is greatly enhanced not only by accurate place measurements of exposure, but also by relating such exposures directly to the exposed individuals over an extended period.

The following hypotheses, rank ordered on the bases of both plausibility and feasibility of investigation, are derived from the foregoing discussion. The hypothesized associations can be expected to be stronger among individuals not experiencing a modifying or buffering effect by such factors as social support, perception of control over stressors and other buffers.

Exposure to sonic boom and low level aircraft flyover noise is hypothesized to be positively associated with an increase in:

1. incidence rates of hypertension after taking into account age, race, sex, obesity, sodium in diet, diabetes, alcohol use, psychological stress, avocational and occupational noise exposures and background noise, and family history of hypertension. When stratified by family history this association will be much stronger for the subgroup with a family history than for those without such a history.
2. incidence rates of myocardial infarction after taking into account the effects of age, race, sex, cholesterol, obesity, hypertension, diabetes, cigarette smoking, sedentariness, type A behavior, psychological stress, avocational and occupational noise exposures.
3. incidence rates of myocardial infarction among those with preexisting heart disease but not among those without known preexisting conditions after adjusting for risk factors and other noise exposures.
4. rate of occurrence of sudden cardiac death among a general population of individuals with no known cardiovascular disease after taking into account age, race, sex, hypertension, cholesterol, diabetes, cigarette smoking, psychological stress, avocational and occupational noise exposures.
5. rate of occurrence of sudden cardiac death among individuals with preexisting arrhythmias and/or heart disease after adjustment for risk factors and current and past avocational and occupational noise exposure.
6. rate of occurrence of sudden cardiac death among individuals with congestive heart failure after taking into account risk factors and current and past avocational and occupational noise exposures.
7. the incidence of arrhythmias among individuals without known underlying cardiac disease after adjustment for risk factors and other noise exposures.
8. the rate, length and number of episodes of arrhythmias with onset time concurrent with noise exposures for persons with diagnosed non-life threatening arrhythmias after adjustment for major risk factors and other concurrent noise exposures.

No quantitative estimates of the magnitudes of any of the hypothesized effects are justified by the existing technical literature reviewed in Chapter 3 and in Volume III of this report.

5. Examination of Research Options for Community-Based Tests of Hypotheses Derived from General Process Model

The preceding chapters have discussed generalities of research designs, assumptions, and uncertainties of various types that constrain epidemiologic study of nonauditory health effects of noise exposure. For purposes of exposition, this chapter begins with a description of an ideal (experimental rather than observational) but unachievable study design. Subsequent sections of the chapter evaluate designs for particular tests of hypotheses about health consequences of exposure to sonic booms and subsonic aircraft noise that are consistent with the hypotheses stated at the end of Chapter 4.

The feasibility of an ideal study is not seriously considered in this chapter for reasons noted below. The chapter does, however, contain detailed examinations of issues pertinent to the design and conduct of more plausible studies. A comparison of the advantages and disadvantages of the various study designs is contained in Chapter 8.

5.1 Design Characteristics of an "Ideal" (Experimental) Study

This section describes design features of an unreasonably ideal experimental study of the effects of MOA and MTR noise on health: one that could resolve essentially all major questions about the effects of residential aircraft noise exposure on human health. A design of this sort is worth consideration only for the sake of exposition, since such a study is unattainable for pragmatic and ethical reasons (cf. Section 2.2.2.7). An experimental study is also beyond the scope of the present evaluation of epidemiologic study designs. The nature of epidemiologic study of community noise effects is characteristically observational (relying on adventitious distributions of exposure over existing populations) rather than interventionist (experimentally manipulating exposure conditions and study populations).

It is nonetheless instructive to describe key aspects of an experimental study design, if only to bring into focus for the reader the contrast between an ideally definitive study and epidemiologic studies that are within the realm of the possible. The major features of an experimental research design include the following:

- A fundamental feature of an experimental study design is explicit manipulation of the independent variable, aircraft noise, through intentional creation of exposure.
- An equally fundamental feature of an experimental study is random assignment of individuals to exposed and nonexposed conditions, so that self-selection bias may be

ruled out as a confounding variable.[9]

- Communities chosen for an experimental study must be representative of the universe of all communities exposed to noise of the sort audible in the vicinity of MOAs and MTRs.
- Random assignment of test participants to exposure conditions prevents potential confounders from contributing systematic bias to a study. However, such confounders can still contribute error and thereby reduce power. To reduce this error, statistical control is required for effects of such confounders in addition to empirical control. Perhaps the most effective technique for achieving this kind of control is matching of pairs of participants. Within each pair, random assignment is made to exposed and control conditions. Since genetic factors play such an important role in the health outcomes most likely to be affected by noise exposure, pairs composed of identical twins, preferably reared together, would be the ideal test participants. Use of an identical twin design would leave only lifestyle factors to contribute error.
- To adjust data analyses for the influences of such lifestyle factors (smoking, exercise, diet, and the like) complete information must be available about them so that adjustment may be made for them. Since these factors are likely to change over the duration of the study, frequent reassessment is necessary. Furthermore, since self-reports of these factors are likely to reflect response bias, particularly in terms of social desirability, some form of objective monitoring would also be required.
- Additionally, statistical adjustment should be made for the dependent variables--health outcomes most likely to be affected by exposure--by measuring them before the study so that baseline data are available.
- The independent variable, aircraft noise exposure, would have to be measured accurately so that its contributions to total noise exposure of individual study participants could be rigorously quantified. Only through long term, continuous monitoring of individual noise exposure, preferably through some form of personal, source-specific dosimetry, can such accuracy be assured.
- The duration of an ideal study would have to be greater than the longest plausible induction and latent periods for diseases of interest. A minimum of ten years for each study phase is advisable.
- The health outcomes of interest must be monitored frequently throughout the study, and followed up for a period of at least 10 years following termination of exposure.
- Finally, a double-blind design is required to control for bias on the part of the researchers as well as the exposed and nonexposed study participants.

The number of participants in a study with all of the design features just described could be as small as a few hundred. It would, however, be possible to compensate for the loss of power

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[9]Since exposure is confounded with location, individuals would need to be assigned to a variety of communities, with half the communities randomly chosen to be noisy and the other half to be free of noise.

suffered in a suboptimal study design (e.g., one in which identical twins cannot be randomly assigned to aircraft noise exposure conditions) by using much larger samples. The most critical element of the design, however, is the one that is least feasible. Neither ethical considerations nor the cooperation of potential participants allows random assignment of individuals to geographic areas for long periods.

Although an experimental study of the sort just described is not even remotely feasible, the remaining sections of this chapter describe and evaluate the designs of more realistic potential studies. A brief discussion that is applicable to all study designs of tolerable error rates precedes the detailed consideration of individual studies.

5.2 Selection of Levels of Type I and Type II Error Rates Appropriate for Feasibility Analyses

The discussion of basics of sample size calculation in Section 2.2.7 concentrated on factors that determine the power of an epidemiologic study. Two other issues of statistical inference must also be considered, however, in this chapter's evaluation of research designs: tolerable error rates and directionality of inferences.

Acceptable rates of Type I and Type II errors are set not by immutable statistical principles, but rather by the real-world costs associated with their occurrence. (Recall that a Type I error is a "false alarm"--an erroneous conclusion that an effect exists when in fact it does not; whereas a Type II error is a "miss"--a failure to detect the existence of a *bona fide* effect.) In an extreme case, the costs associated with a miss may be prohibitive, while the costs associated with a false alarm may be negligible. In other cases, the costs may be reversed, or intermediate in magnitude.

Error rates conventionally adopted in epidemiologic study are $\alpha = .05$ for Type I and $\beta = 0.2$ for Type II error. In other words, 1 chance in 20 of a false alarm and 1 chance in 5 of a miss are generally considered acceptable for inferences drawn from observational epidemiologic research. All other things being equal, studies supporting inferences at these error rates are regarded as publishable in professional journals, whereas studies that can support only inferences with higher error rates are viewed with some skepticism. Different standards of proof may be justified for purposes other than publication of findings in a journal, however, particularly when the costs of erroneous conclusions are unaffordable.

Occurrence of a Type I error in a study of the effects of aircraft noise on cardiovascular morbidity would spuriously indicate that noise exposure adversely affected health, when in fact

it did not. At $\alpha = .05$, there is 1 chance in 20 of drawing an erroneous conclusion of this sort from a study of the effects of aircraft noise exposure on cardiovascular morbidity. It is therefore prudent to consider limiting this risk to one chance in 100 ($\alpha = .01$) rather than to 1 chance in 20.

It is also important to guard against inflation of Type I error when multiple statistical tests are applied within a single study. Using a Type I error rate of .05 in a study in which 20 statistical tests are done makes it highly likely that at least 1 will show statistical significance spuriously. If one is willing to accept a 5% chance of a false alarm in such a study, an appropriate error rate for each individual statistical test would be .05/20 = .0025 (25 chances in 10,000).

Occurrence of a Type II error in a study of the effects of aircraft noise on cardiovascular morbidity would spuriously indicate that noise exposure did not affect health, when in fact it did. Two types of costs can be identified with an error of this type. First, resources spent on the study, which could well be considerable, would be wasted. Second, in the inherently adversarial environmental review process, a study with too high a rate of Type II error could easily be criticized as inadequate: that is, too likely *a priori* to miss an important effect. Since power is the complement of the Type II error rate (power = 1.0-β), the motivations for sponsoring an insufficiently powerful study could well be questioned. For these reasons, it is also prudent to consider adopting a Type II error rate of $\beta = 0.1$, half of the rate usually considered adequate for publication.

All of these considerations (and others as well) have a direct influence on sample size calculations, which can play a key role in decisions about feasibility of epidemiologic research.

5.3 Episodic Exposure to Sonic Booms: Oklahoma City

Residents of the Oklahoma City metropolitan area were intentionally exposed to sonic booms from February to July of 1964 as part of a cooperative project carried out by the Federal Aviation Administration and the Air Force. To date, this Oklahoma City study has produced the most prolonged, predictable, and well-documented exposure to moderate level (1-2 psf) impulsive aircraft noise in a large residential population in the United States. Barring repeal of the National Environmental Policy Act of 1969, it is also the largest scale study of its type that is ever likely to be conducted in the United States. It is regrettable from the current perspective that no provisions were made at the time for study of potential health consequences of sonic boom exposure.

The following subsections examine the feasibility of studying potential sonic boom effects

on health a quarter of a century later. The feasibility analysis starts by identifying certain unavoidable assumptions of any studies that might be conducted in Oklahoma City. The analysis then continues with examinations of what is known about sonic boom exposure of individuals, of the sort of health information available today, of the size and accessibility of the population at risk, and of the adequacy of potential sampling plans. These discussions are applied first to a retrospective morbidity or mortality study, and then to a case-control design.

5.3.1 General Assumptions Required for Studies in Oklahoma City

What sort of epidemiologic study can be designed a quarter of a century after the fact to determine whether sonic boom exposure in Oklahoma City produced an increase in cardiovascular disease consistent with the hypotheses listed in Section 4.6? An answer to this question requires an examination of (1) the nature of the noise exposure itself, (2) the size of the effect that can be reasonably expected, (3) the nature of the dependent variables available to characterize health effects, (4) the size and composition of cohorts residing in exposed and nonexposed areas, (5) the ability to estimate and control for effects of confounding variables, and (6) the types of statistical analyses that can be performed on data produced by such a study.

A fundamental assumption of a study intended to take advantage of the sonic boom exposure in Oklahoma City is that 6 months' exposure to a maximum of 8 sonic booms per day suffices to produce observable symptoms of cardiovascular disease. If the total duration of exposure to sonic booms necessary to produce cardiovascular symptoms exceeds 6 months, it is not possible to document the production of disease from the 1964 Oklahoma City exposure.

Common experience indicates that few (if any) individuals manifest overt symptoms of cardiovascular disease immediately following exposure to loud impulsive noises. What then is the period that must elapse before symptoms of disease are apparent? Although there can be little certainty about the matter, the latent period is probably on the order of several years to a decade or more. A study of the effects of sonic boom exposure in Oklahoma City must therefore be designed to provide an opportunity to study morbidity rates over a comparable period.

Several other general assumptions implicit in the design of such a study may also be identified. For reasons described in Chapter 4, disease produced by half a year's exposure to a maximum of 8 booms per day would be most likely to manifest itself in cardiovascular symptoms. Such symptoms could in principle be detected in morbidity statistics, or perhaps in mortality rates. A retrospective morbidity or mortality study would therefore have to discern a greater frequency of such symptoms in a noise exposed population than in an otherwise similar ("matched") unexposed population. It follows from the discussion of Section 2.2 that the more defensible studies of disease associated with the half-year long episode of exposure in the Oklahoma City area are (1) one in which rates of cardiovascular morbidity could be compared in

cohorts of people residing in exposed and nonexposed areas, and (2) a case-control design. Assumptions must also be made in the design of such studies about (1) the proportion of the population susceptible to the disease; and (2) the identity of the subpopulation at greatest risk of developing the disease.

The availability of information to support these assumptions is examined in the following subsections.

5.3.2 Noise Exposure in Oklahoma City

The aircraft that produced the sonic booms in the Oklahoma City study flew a straight line course 100 nm long as illustrated in Figure 5-1 (adapted from Borsky, 1965). At different times during the 6 month period of exposure sonic booms generated along this flight track varied in intensity from 1 to 2 psf, in number from 1 to 8 per day, and at times of day ranging from 7:00 a.m. to 1:20 p.m. No further published documentation of the day-to-day variability in this schedule is available. The only documentation available of acoustic measurements made to quantify the resulting noise exposure is reproduced in Figures 5-2 through 5-4. No original information is available about C-weighted Day-Night Average levels produced by these booms. Estimates made after the fact are described in Section 5.3.4.

Although residential addresses in Oklahoma City and its suburbs can be classified with respect to proximity to the flight track, it is doubtful that such classification could provide a reliable basis for estimating the sonic boom exposure of any individuals living in the environs of Oklahoma City in 1964 Sonic booms were produced in daytime hours during which large portions of the residential population are commonly away from home. An unknown number of sonic booms was almost certainly audible to virtually the entire population living or working in the environs of Oklahoma City during the test period. Since no provisions were made in 1964 to quantify anything other than place exposure, the numbers of booms heard by different individuals or classes of individuals is essentially unknowable today.

5.3.3 Nature of Dependent Variables Available

Given that a study must be conducted retrospectively, the number of dependent variables that can be scrutinized for evidence of cardiovascular disease is rather limited. The more obvious metrics of the cardiovascular morbidity data include numbers of visits to physicians and numbers of hospitalizations. Statistics from death records are the obvious source of mortality information.

The former information is very difficult to obtain retrospectively from individual private

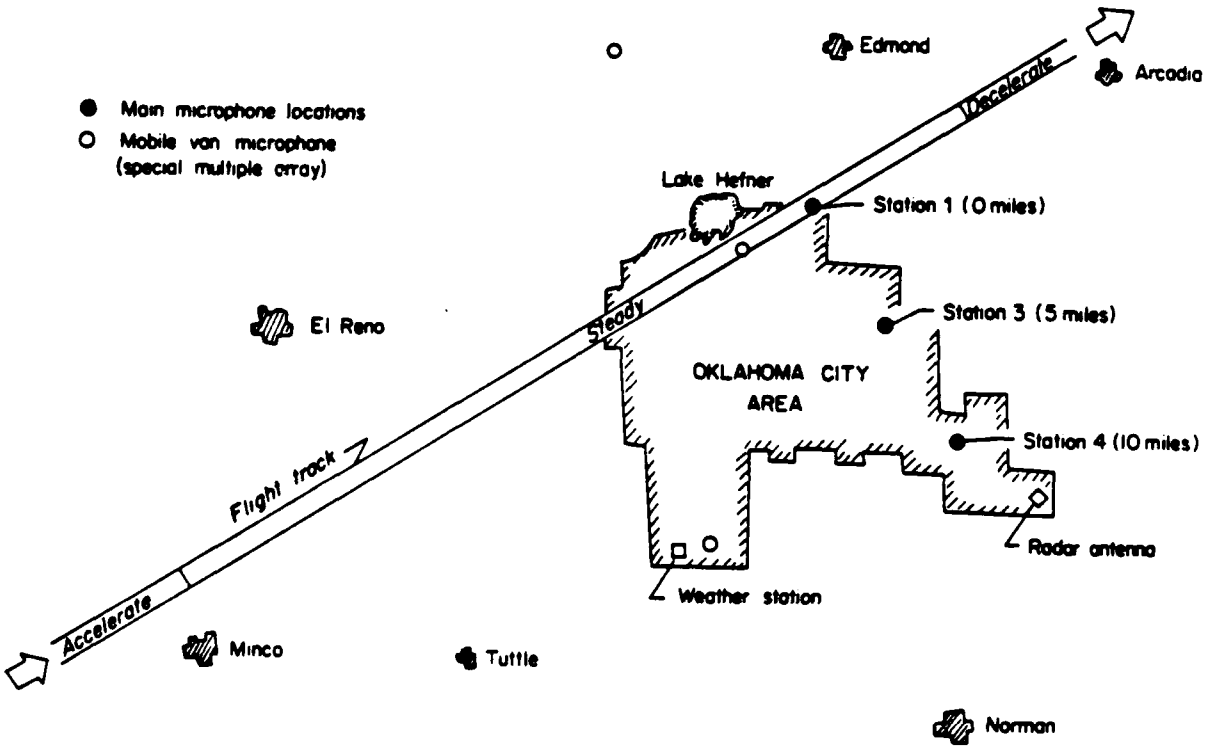


Figure 5-1: Planview Sketch of Sonic Boom Test Area Showing Flight Track, Measuring Stations, and Facilities (from Borsky, 1965).

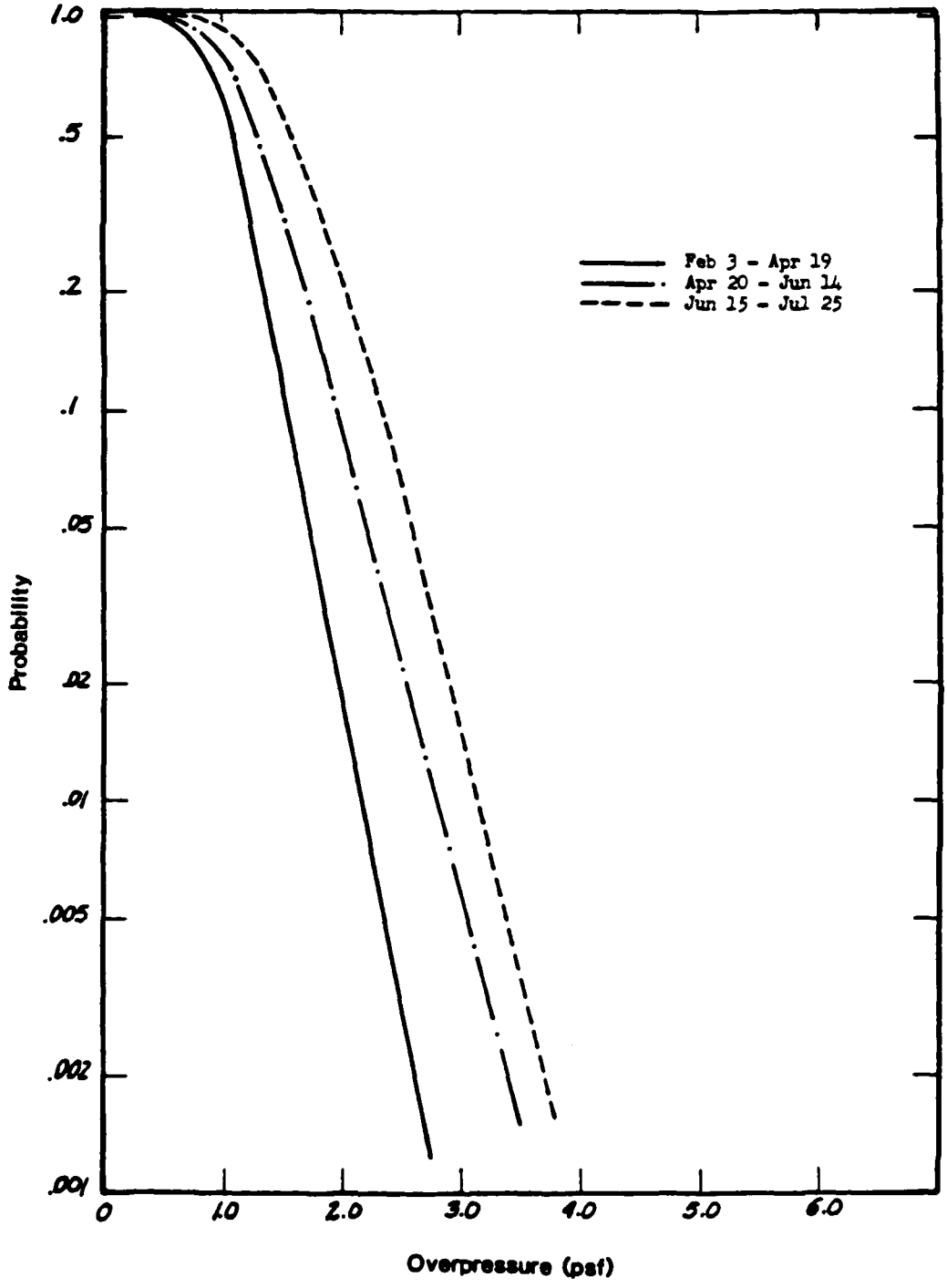


Figure 5-2: Cumulative Probability of Experiencing Sonic Boom Overpressures 0-8 Miles from Ground Track (from Borsky, 1965).

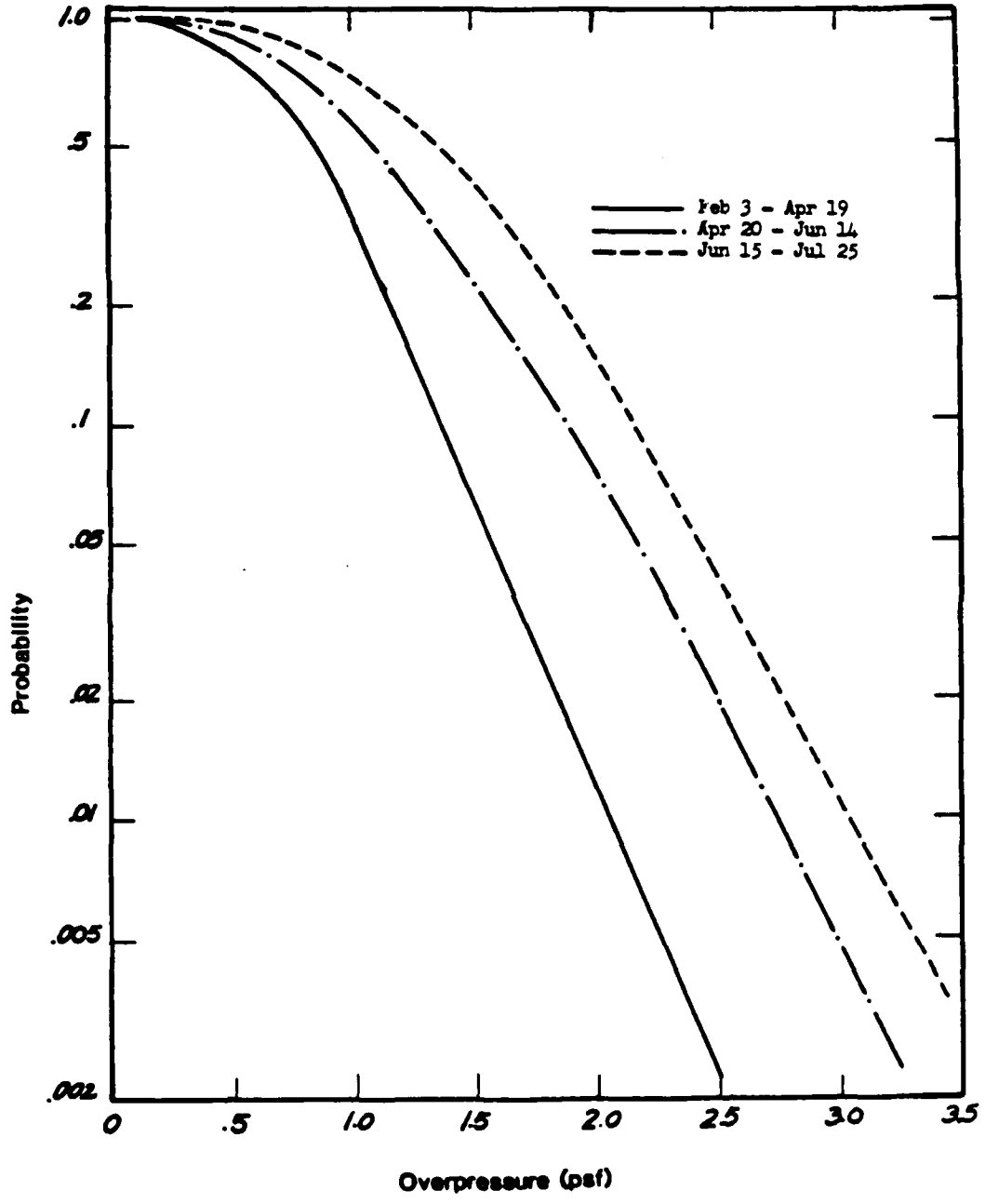


Figure 5-3: Cumulative Probability of Experiencing Sonic Boom Overpressures 8-12 Miles from Ground Track (from Borsky, 1965).

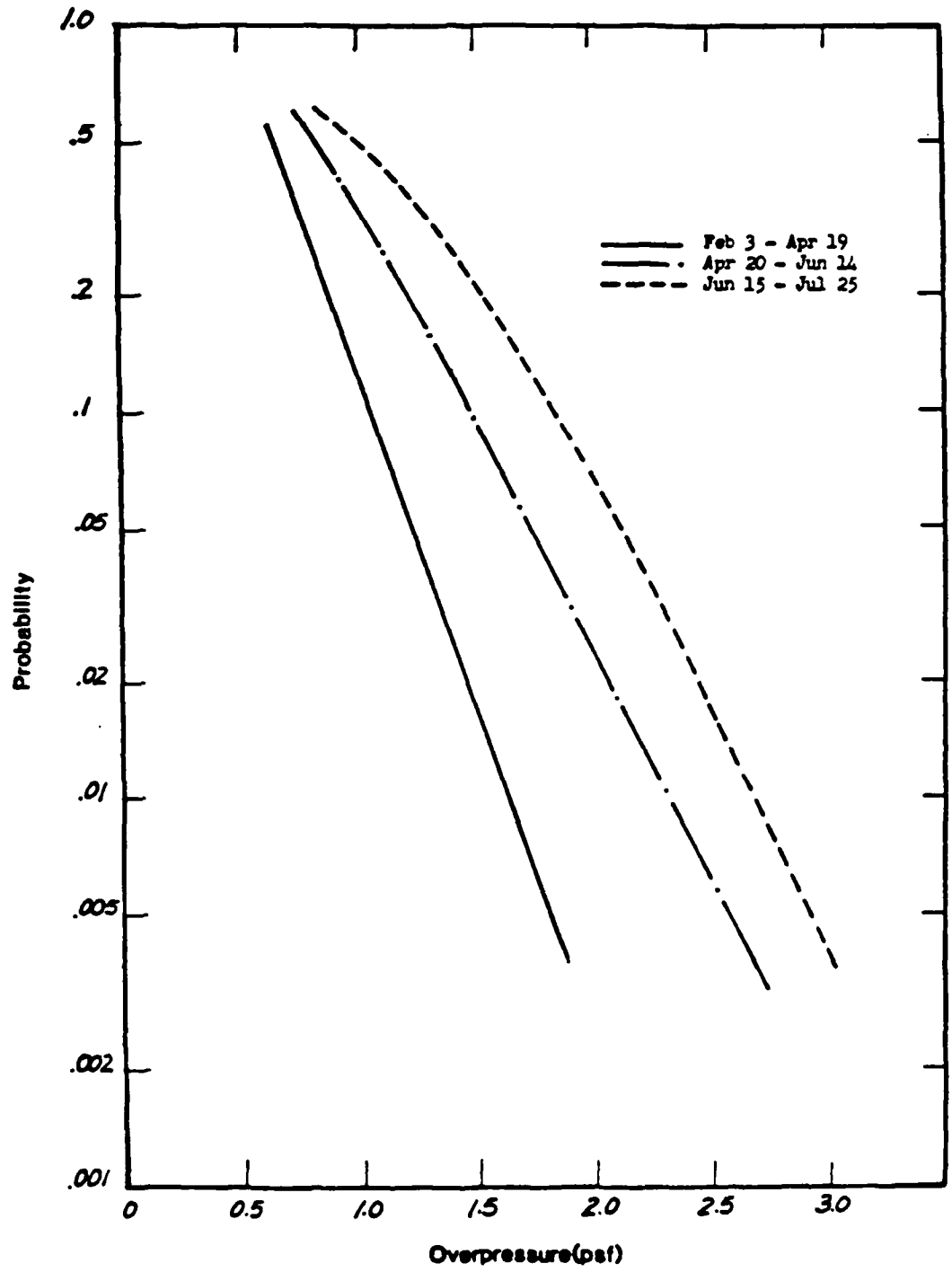


Figure 5-4: Cumulative Probability of Experiencing Sonic Boom Overpressures 12-16 Miles from Ground Track (from Borsky, 1965).

practitioners. Physicians are in general reluctant to release records of their patients' office visits, both on grounds of confidentiality and out of concerns about litigation. Most, if not all, physicians practicing today (who may include relatively few of those who were practicing in the area a quarter of a century ago) would require informed consent for participation in such a study from their patients. Obtaining such consent from survivors of the 1964 exposure period or, if deceased, from their next of kin, is not a trivial matter. Furthermore, physicians cannot be counted on to cooperate in a study with little apparent prospect for producing definitive outcomes or immediate benefits. Even if adequate numbers of physicians were willing to cooperate in an epidemiologic study, individual physicians' record keeping practices may limit the amount of useful information about the periods of interest.

Records of outpatient services provided by HMOs or other large health care organizations are potentially less difficult to obtain, but are not public records that may be examined as a matter of law. Access to such information is obtainable only through the cooperation of officials of health care organizations. No guarantees of such cooperation are possible.

This limitation leaves hospital admission and discharge information as the primary indices of cardiovascular morbidity. In some areas of the United States, information of this sort is routinely collected by vital statistics agencies. In some cases, it may even be possible to gain access to length of stay and discharge diagnoses coded by the *International Classification of Disease* (ICD). However, hospital admission and discharge data are records of events; an individual may be represented in the data as many times as he/she is admitted to any hospital in the reporting system. Personal identifiers (such as name, address, hospital number, or social security number) that would be required to identify the number of individuals admitted and to link these records to other data sources are not necessarily available. Historical information about residence addresses of individuals at times of sonic boom exposure is not recorded in admission and discharge records. Without historical and current identifiers for individuals, it becomes virtually impossible to conduct a study that can control for effects of confounding risk factors such as smoking, diet, and others discussed in Chapter 4.

Great difficulties are also encountered when efforts are made to assess cardiovascular mortality through death records. Historical residence addresses of individuals are not identifiable on death records. Although readily obtained, death records are often unreliable. Feinstein (1988) cites several studies demonstrating the inaccuracy of death certificates due to (1) incorrect or inadequate identification of individual diseases, (2) unrealistically low rates for individual diseases because only one of many diagnoses is cited on the death certificate, and (3) records that do not include the many silent diseases that are first detected (if at all) at necropsy. To be of value, death records would have to be supplemented with morbidity information subject to all of the difficulties just noted.

There is also an important logical difficulty in retrospective mortality analyses. Meaningful

analyses of a given cause of death require control for all other causes of death. Random variation in causes of death makes it highly likely that any one mortality study will find that the death rate for some cause is higher in one location and/or time than another. At the same time, the death rate for another cause will be lower, unless there are unusually large differences in gross mortality between locations or times. Since the total number of deaths from year to year is roughly constant in an area of stable population, a potential finding of greater mortality from cardiovascular disease in Oklahoma City during a particular time would almost certainly be accompanied by a complementary finding of reduced mortality for another cause, say cancer. It would make as little sense to argue from such findings for the adverse effect of sonic booms on cardiovascular health as for the protective effect of sonic booms against cancer.

5.3.4 Identification of Population at Risk

Identifying a population at risk of cardiovascular disease is among the first problems that must be addressed in designing a study. This population must be defined to permit construction of a defensible sampling plan. Sampling, in turn, is needed to avoid the prohibitive costs of an exhaustive study through identification of a representative subset of the population.

Sonic booms produced during the test period were audible in varying degrees at varying distances from the flight track. Since Figure 5-5 depicts areas with boundaries as far as 16 miles from the centerline of the flight track, residence within a 32 mile wide corridor centered on the flight track is at least a starting point for a criterion of exposure. More refined criteria would seek to distinguish subpopulations exposed in categories of sonic boom exposure within yet narrower boundaries. (Recall, however, that place of residence is a poor criterion in any event, as discussed in Section 5.3.2.)

One potential source of information for an attempt to define the population at risk is a sampling frame for a social survey of community response to the 1964 sonic boom exposure. The National Opinion Research Center of the University of Chicago has indicated that block-level information about areas in which interviewing was conducted might be available. Since interviews were conducted in only a small portion of the total exposed area, however, even this information would provide only a start at a definition of the exposed population. Thus, the potential for errors of misclassification of exposed and unexposed populations would not be usefully reduced by the availability of the sampling frame for the 1964 social survey. As no personal identifiers were retained for individual survey respondents, no followup would be possible in any event.

Figure 5-5 shows that the 32 mile wide corridor encompasses portions of 7 Oklahoma

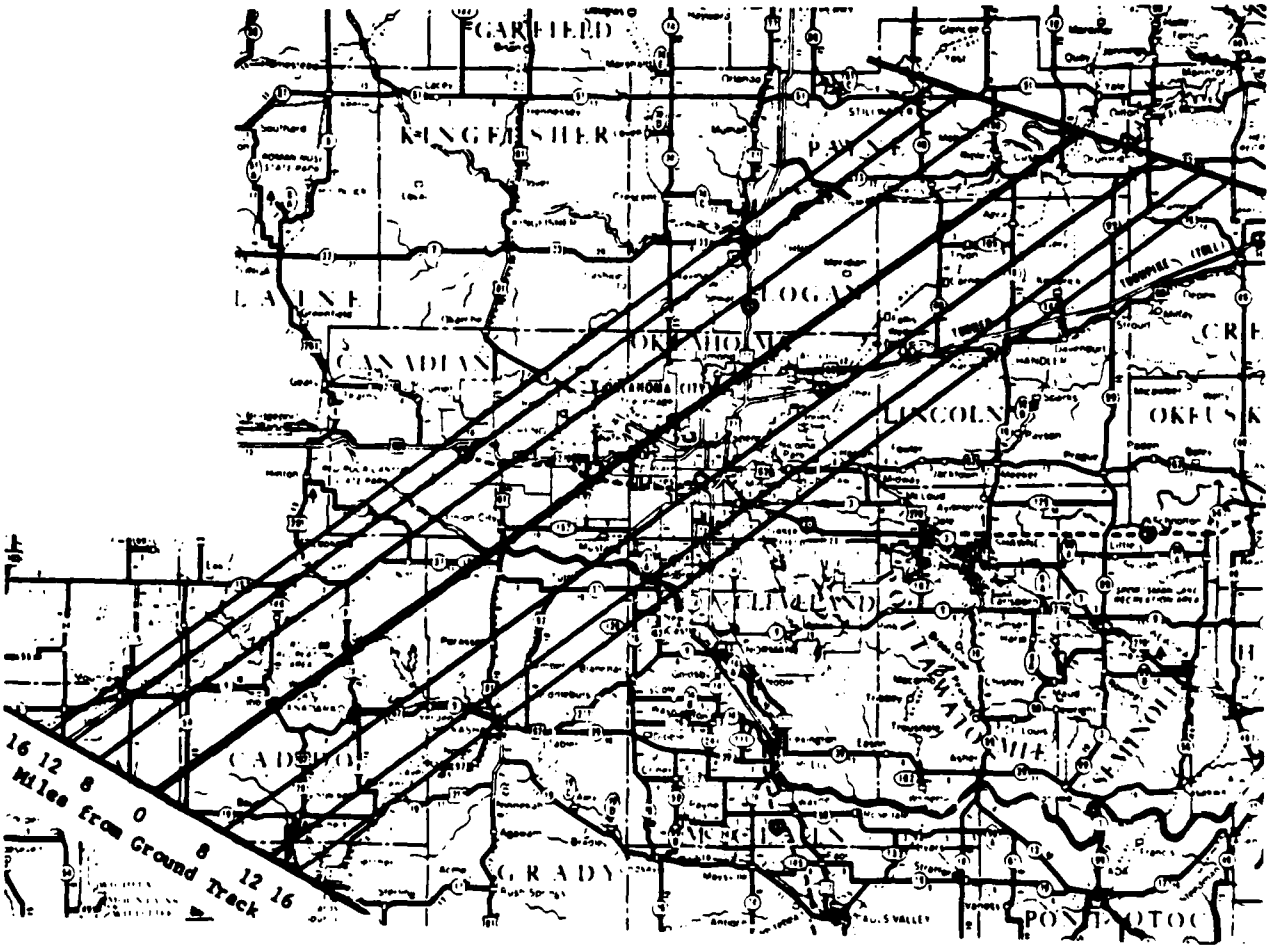


Figure 5-5: Map of Flight Track Showing Sonic Boom Exposure Zones in Oklahoma City in 1964 (from Borsky, 1965).

counties. Figure 5-6 displays estimates of the total populations aggregated over these counties[10], both for the year in which exposure occurred and for subsequent years through 1986, the most recent year for which census estimates are available. The figures on which these and subsequent population estimates are based are derived from a set of publications by the Oklahoma Employment Security Commission (1981, 1987). As may be seen in Figure 5-6, the populations of the counties overflown supersonically in 1964 have increased markedly over the past two decades, from about 650,000 to nearly a million persons. Large portions of the overflown counties were semi-rural in the mid-1960s. The population in one county (Oklahoma County, containing the only large metropolitan area) exceeded that in all other counties by an order of magnitude.

The population in a 32 mile wide rectangle bisected by the supersonic flight track is considerably smaller than that in the entirety of the 7 counties. Figure 5-7 plots estimates of the populations of the overflown areas of the 7 counties by year. The 1964 approximations are probably overestimates of the size of the sonic boom exposed population, since they are proportionate area estimates, based on an assumption of constant population density throughout entire counties. Note that the populations of the overflown areas of the 7 counties increase with time (even though the size of the sonic boom-exposed cohort of 1964 residents of course does not) due both to net immigration and indigenous population increase.

Likewise, not all persons exposed to sonic booms will necessarily develop cardiovascular disease. If the proportion of people who developed cardiovascular disease following the exposure in Oklahoma City were very large (say, a tenth or more of the exposed population), an anomalous increase of such proportions would long since have attracted extensive attention, even if its etiology were unknown. Since such is not the case, any study of sonic boom influences on the cardiovascular system must be designed to detect small increases in disease incidence and/or mortality.

Given that sonic boom effects are most plausibly manifested in cardiovascular symptoms, the subpopulation at greatest risk would clearly be those already at greatest risk of cardiovascular disease. At highest risk for hypertension are elderly women, followed by elderly men, as seen in Table 2-1. Rates drop off steadily for younger persons. As discussed in Chapter 4, psychosocial factors (e.g., annoyance and other noise-related stressors) are hypothesized to mediate effects of noise on cardiovascular function. Therefore, elderly women and men annoyed to a consequential degree by sonic boom exposure can be expected to be those most susceptible to cardiovascular disease attributable to sonic boom exposure, followed by middle aged men and women.

The general process model (Section 4.5) emphasizes the importance of intervening

[10]These estimates should not be viewed as precise, since few are based on an actual census.

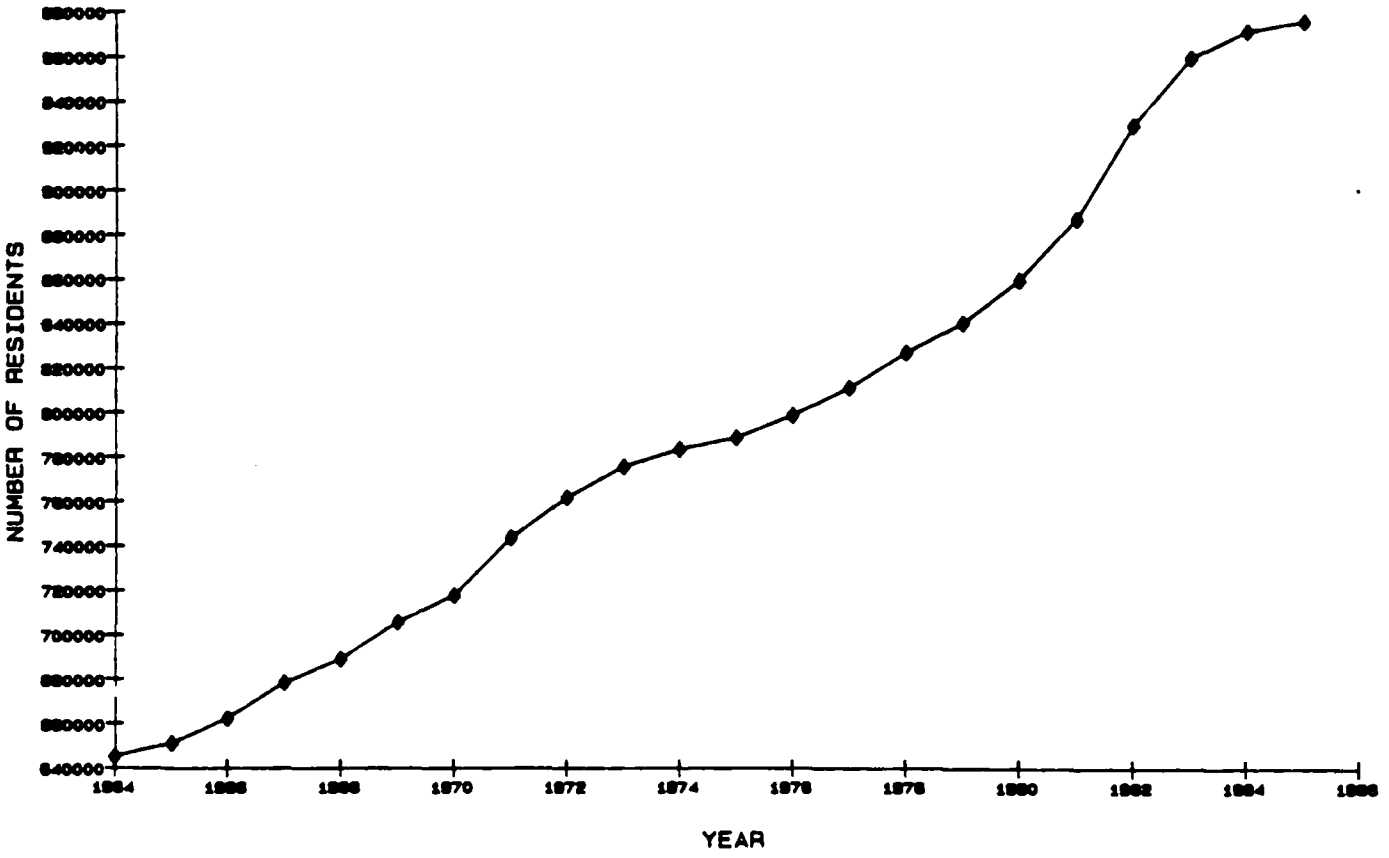
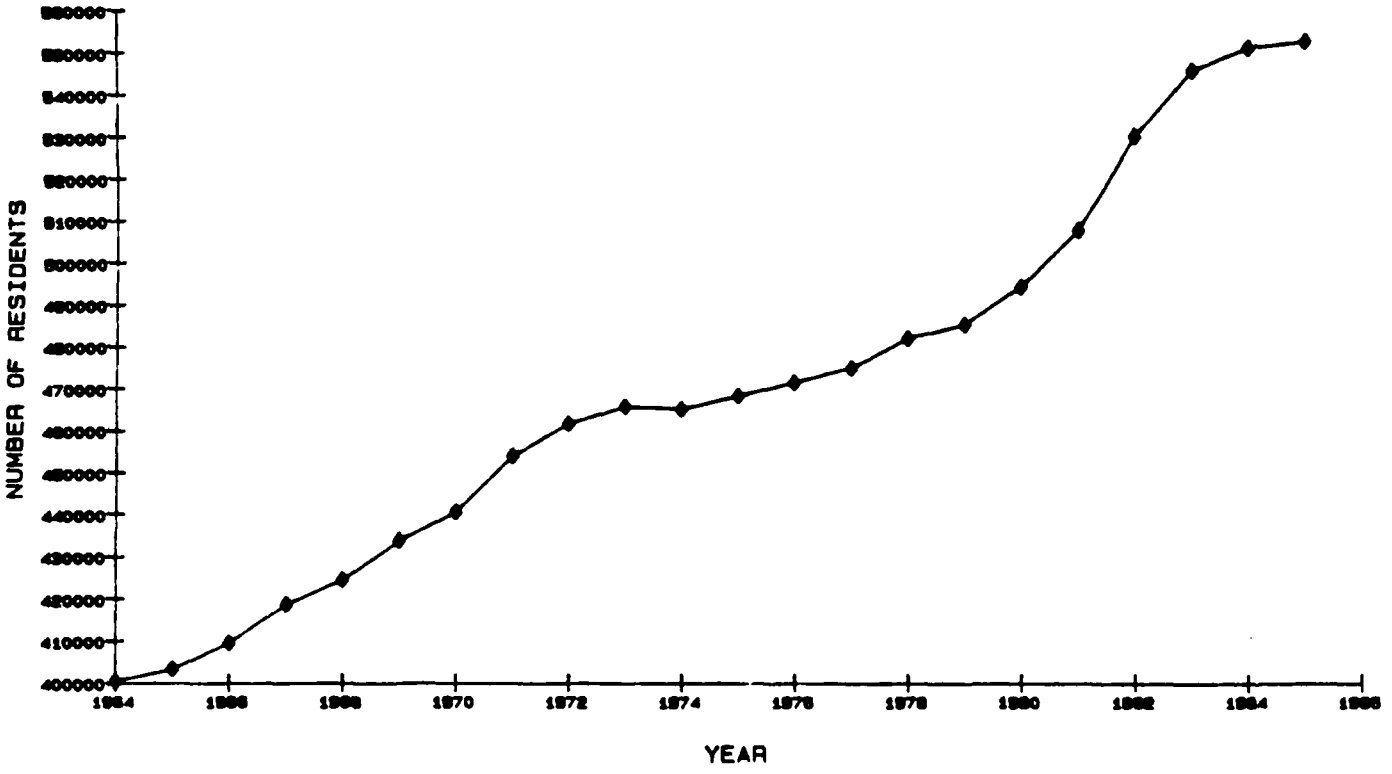


Figure 5-6: Approximate Total Population of 7 Oklahoma Counties Overflown Supersonically in 1964.



Note:
Assuming constant population density throughout county.

Figure 5-7: Approximate Population Living Within 16 Miles of 1964 Supersonic Flight Track.

variables such as annoyance as potential for noise-induced stress in the linkage between noise and possible health effects. Galloway's (1981) adaptation of data from Borsky's (1965) social survey of Oklahoma City residents overflown in 1964 permits estimation of the percentage of population consequently annoyed by sonic booms. As seen in Table 5-1, the percentage of those annoyed varied from about 3% to 22%, depending on location within the flight path and peak overpressure during three successive time intervals. Since there is little evidence of associations among age, sex, and prevalence of noise-induced annoyance (cf. Fidell, 1978), these rates can be reasonably applied to the entire middle aged and elderly subpopulation.

5.3.5 Estimation of Size of Population at Risk

Tables 5-2 and 5-3 estimate the size of the middle aged and elderly population of the overflown portions of the 7 counties still alive in 1964 and subsequent years. The estimates of each age and gender cohort were constructed on a county-by-county basis. The figures have also been adjusted for age-specific mortality rates for each age and gender cohort (ranging from 2 per thousand per annum for women 40-44 to 55 per thousand per annum for men aged 65-74) as well as for out-migration from the exposed area.

Figure 5-8 continues this line of reasoning by taking the estimated total numbers of middle aged and elderly men and women in the potentially exposed 1964 cohort still living and reducing those numbers to those most likely to manifest the expected cardiovascular symptoms of sonic boom exposure: those who were seriously annoyed. The lower two curves in Figure 5-8 reduce the susceptible population as suggested by the estimates in Table 5-1 to those most likely to manifest sonic boom-related cardiovascular symptoms. About 43,000 of the exposed population over 40 remained alive in 1986. Approximately 1,300 to 9,500 of them may have been sufficiently annoyed to be at risk for developing cardiovascular disease.

Figure 5-9 expresses the same information in the form of proportions; that is, as proportions of the population of men and women who were 40-74 years of age during 1964, and hence, could potentially have been exposed to sonic booms and been sufficiently annoyed by them to develop or exacerbate cardiovascular disease. Note the rapid reduction in numbers of such individuals over time. Even though these figures are somewhat uncertain estimates, it is nonetheless clear that most members (over 70%) of the subpopulations with the greatest vulnerability to cardiovascular disease arguably attributable to sonic boom exposure were no longer alive in 1986. Fewer would be alive were a study to commence after 1989.

Table 5-1: Estimated Day-Night Average Sound Levels and Percent of Total Population Expressing "Serious" Annoyance due to Sonic Boom Exposure in Oklahoma City in 1964.

Conversions from Borsky (1965) Oklahoma City Study				
Location 1	*Nominal* Δp	L_{dn}	L_{Cdn}	*Percent Annoyed*
1st period	1.13	52.6	62.3	10.5
2nd period	1.23	53.6	63.0	16.1
3rd period	1.60	56.1	65.3	21.7

Location 2	*Nominal* Δp	L_{dn}	L_{Cdn}	*Percent Annoyed*
1st period	0.8	47.6	59.3	7.9
2nd period	1.1	52.1	62.0	12.2
3rd period	1.3	54.1	63.5	15.2

Location 3	*Nominal* Δp	L_{dn}	L_{Cdn}	*Percent Annoyed*
1st period	0.65	44.1	57.7	3.0
2nd period	0.85	48.6	59.8	6.5
3rd period	1.0	50.6	61.2	10.1

Table 5-2: Estimated Number of Women Aged 40-74 in Sonic Boom Exposed Areas in 1964 Still Alive in Subsequent 1-Year Periods.

Year	*40-44 years old*	*45-54 years old*	*55-64 years old*	*65-74 years old*
1964	9736	21550	19415	14705
1965	9717	21442	19220	14338
1966	9692	21324	18997	13919
1967	9660	21195	18745	13448
1968	9623	21055	18463	12926
1969	9581	20904	18153	12353
1970	9532	20742	17813	11728
1971	9478	20570	17444	11051
1972	9420	20386	17046	10323
1973	9357	20193	16619	9544
1974	9288	19988	16163	8713
1975	9215	19772	15677	7831
1976	9138	19524	15124	6801
1977	9055	19244	14503	5625
1978	8967	18932	13813	4301
1979	8875	18587	13056	2831
1980	8777	18210	12231	1213
1981	8665	17800	11338	0
1982	8539	17359	10377	0
1983	8398	16885	9348	0
1984	8242	16378	8251	0
1985	8071	15839	7086	0
1986	7886	15225	5727	0

Table 5-3: Estimated Number of Men Aged 40-74 in Sonic Boom Exposed Areas in 1964 Still Alive in Subsequent 1-Year Periods.

Year	*40-44 years old*	*45-54 years old*	*55-64 years old*	*65-74 years old*
1964	9252	19762	17135	10392
1965	9215	19584	16792	9873
1966	9169	19385	16398	9301
1967	9113	19163	15953	8678
1968	9048	18920	15456	8002
1969	8974	18655	14908	7275
1970	8891	18369	14308	6495
1971	8798	18060	13657	5664
1972	8694	17730	12954	4780
1973	8580	17379	12200	3845
1974	8456	17005	11395	2858
1975	8322	16610	10538	1819
1976	8178	16155	9596	779
1977	8023	15642	8568	0
1978	7859	15069	7454	0
1979	7684	14436	6254	0
1980	7499	13744	4969	0
1981	7286	12993	3598	0
1982	7045	12183	2142	0
1983	6777	11314	600	0
1984	6481	10385	0	0
1985	6157	9397	0	0
1986	5806	8310	0	0

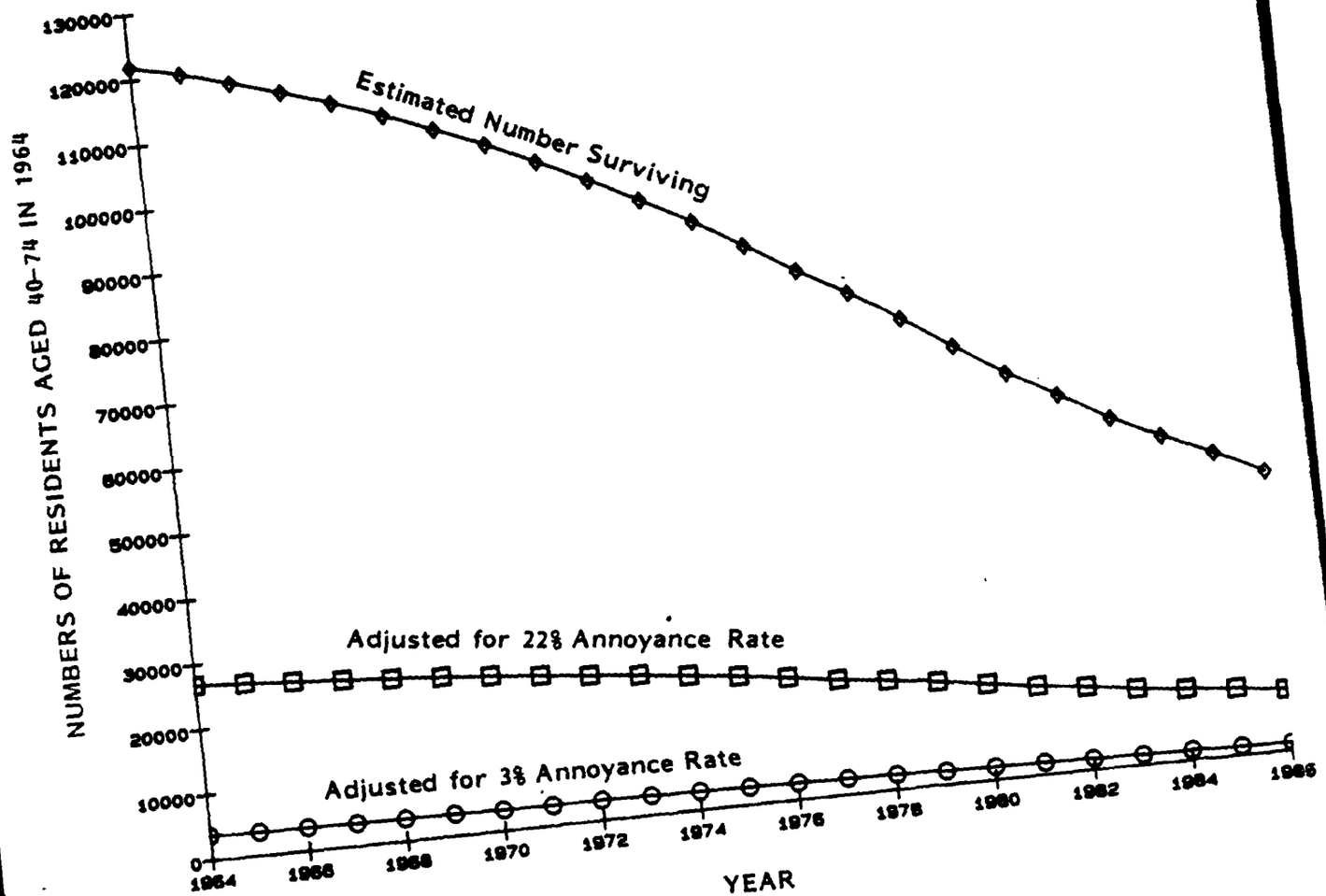


Figure 5-8: Estimated Numbers of Surviving Residents in Overflown Areas Aged 40-75 Years in 1964.

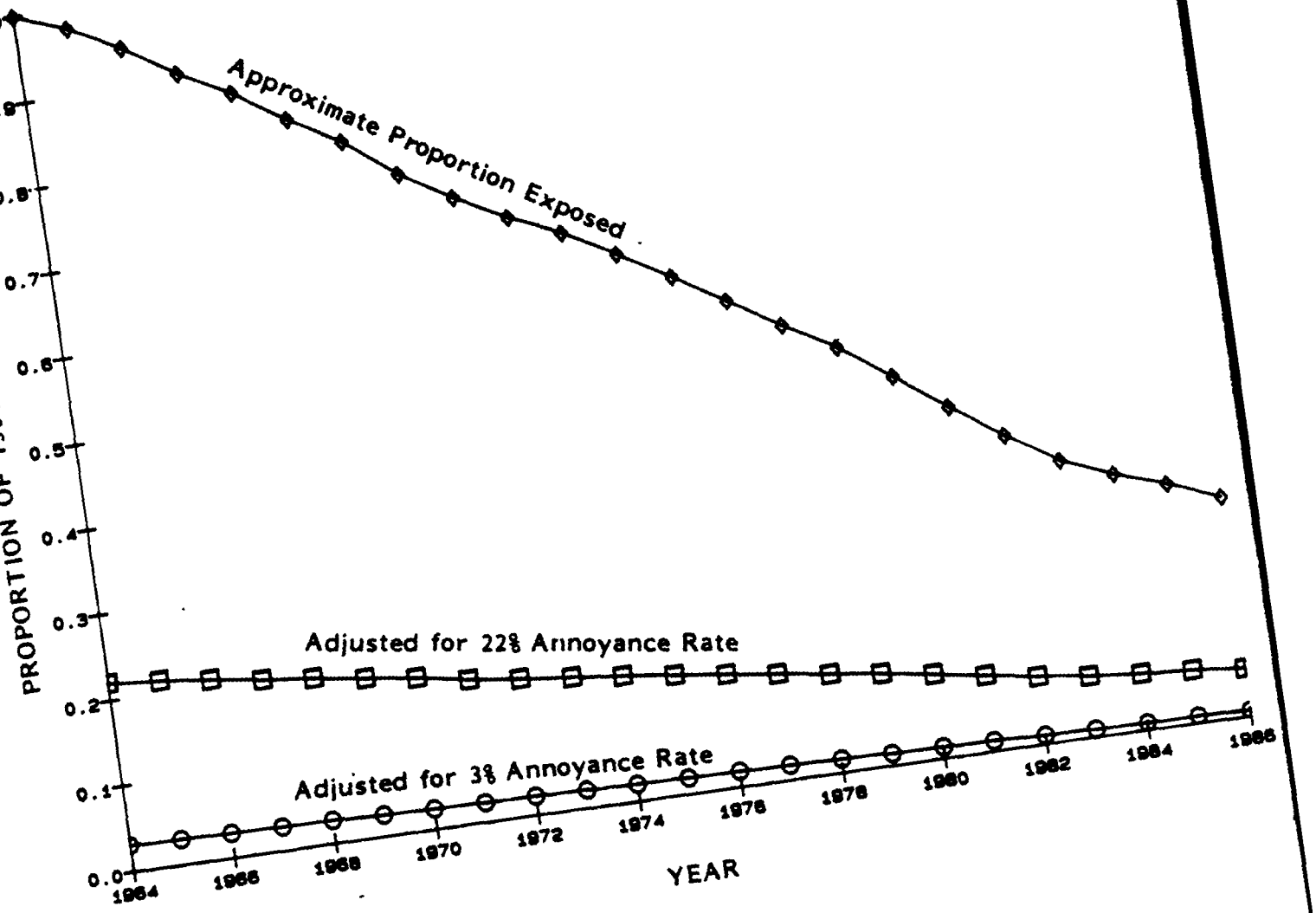


Figure 5-9: Approximate Proportion of 1964 Exposed Cohort of Residents Aged 40-74 Years, Adjusted for Age Specific Mortality and Population Growth as well as Out-Migration.

5.3.6 Examination of Feasibility of a Retrospective Cohort Study

It is clear from the foregoing section that for all practical purposes, few of the older 1964 residents of Oklahoma City most susceptible to symptoms of sonic boom-related cardiovascular disease are available for interview and examination today. Nonetheless, this section explores the feasibility of studies based on secondary as well as primary sources of information about these residents, and studies of younger 1964 residents who may have been less susceptible to sonic boom related cardiovascular disease but might be available for interview and medical examination today.

5.3.6.1 Cohort of 1964 Oklahoma City Residents at Greatest Risk

The Framingham 30-year followup study for hypertension (Dannenberg et al., 1988) shows that women over 55 and men over 65 are most susceptible to hypertension, with incidence rates at about 30 per thousand and higher. Among these residents, Tables 5-2 and 5-3 show that only 5,727 older women would be expected to be alive in 1986. Extrapolating to 1990, the survival rate of this cohort would be expected to be about 2,010. At annoyance rates of 3% and 22%, this suggests about 60 to 442 surviving women (or 251, averaging of the two estimates of annoyance rate) at high risk for cardiovascular disease due to exposure to sonic booms in 1964.

Based on an annual hypertension incidence rate for these women of about 30 per thousand (Dannenberg et al., 1988), Table 2-2 suggests that if all 251 survivors could be located, and if all information were reliable and valid with no misclassification bias, the risk among those exposed to sonic booms would have to be about 4 times greater than for those not exposed in order to detect a cardiovascular effect of sonic boom exposure on hypertension.

Since individuals at risk were not in fact identified at the time of exposure, misclassification bias can be expected to be large although unmeasurable. Even assuming reliable memory as to exposure among these now very elderly women, additional sources of misclassification bias exist with hospital and physician records. As seen in Table 2-4 even if the true risk of developing sonic boom-induced cardiovascular disease were 2.0, misclassification bias would reduce the observed risk ratio to a figure between 1.31 and 1.73, clearly beyond detectability for this cohort.

The possibility exists, however, that surviving relatives and friends could be contacted to expand the sampling pool to include information about older residents who were exposed to sonic booms in 1964 but who are no longer alive. Tables 5-2 and 5-3 show a total population of 44,512 in 1964 in the highest risk cohorts: men 65-74 and women 55-74. At an average annoyance rate of 12.5%, about 5564 residents may have been at risk. Assuming that about 10% of these residents or their surviving relatives or friends could be located, Table 2-2 suggests that the relative risk would have to be greater than 2.5 to be detectable. Lower relative risk rates

could be detectable if a larger proportion of surviving relatives and friends could be located, as might be possible with a massive publicity campaign.

Without access to residents themselves, however, misclassification bias can be expected to be even higher than for samples consisting only of surviving exposed residents. To the usual sources of misclassification bias are added:

- the difficulty in locating surviving relatives and friends who may have since changed residences and/or names;
- the unreliability of death records;
- the unreliability of memory about a friend of relative who may have died a decade or more ago; and
- the likelihood that younger relatives would be unaware of the temporal sequence of cardiovascular disease and sonic boom exposure.

To achieve an observed relative risk of 2.5, then, would probably require a true risk well in excess of 3.5, one so anomalously high that it would almost certainly have been long since widely recognized.

5.3.6.2 Cohort of 1964 Oklahoma City Residents at Lesser Risk

Middle aged men and women constitute another subpopulation from which samples could be drawn. Although this group experiences lower CVD mortality than elderly men and women, they are more likely to be alive today, so that they are more easily traced and interviewed.

Extrapolating from Tables 5-2 and 5-3, the 43,000 survivors in 1986 who were over 40 in 1964 would be expected to be reduced to about 29,000 in 1990. If all of these survivors are considered, the adjusted incidence rate (weighted by number of survivors in each age-gender cohort) is about .021, based on the Framingham study (Dannenberg et al., 1988). Table 5-4 shows the proportion of those residents over 40 in 1964 that would have to be located to provide sufficient power to detect a given relative risk. As can be seen in Table 5-4, one would have the power to detect an effect of the sonic booms given a relative risk of about 2.0, without adjustment for misclassification bias if all of the following conditions were met:

- if 21% of the 28,731 residents of the overflown portion of the Oklahoma City area who were over 40 in 1964 could be located;
- if there were no selective migration out of the exposed area (that is, if those exposed were no more likely to leave the area than those who were not exposed);
- if all 6,100 (21% of 29,000) of those located were willing to participate in a study; and
- if 12.5% of these people had been seriously annoyed by the sonic boom exposure.

A true relative risk substantially greater than 2.0 would be required to detect an effect of sonic boom exposure under realistic assumptions about misclassification bias (cf. Table 2-4). The aircraft noise health effects literature contains no credible evidence of relative risks of this magnitude.

Table 5-4: Proportion of Overflown Population Over 40 Years Old in 1964 Needed for Sampling.

Sample Size and Proportion of Overflown Population				
Relative Risk	*Cohort Sample Size Needed*	*Annoyance Rate* 22%	12.5%	3%
1.5	4866	.77	>1.00	>1.00
2.0	1450	.23	.40	>1.00
2.5	747	.12	.21	.87
3.0	477	.08	.13	.55

Notes:

α = .05, 2-sided test; β = .10.

P(Disease) assumed to be .021 in nonexposed.

Whether the study is limited to the cohort of exposed women aged 55-64 in 1964 still expected to be alive, or is expanded to include the surviving friends and relatives of the cohort of men aged 65-74 and women aged 55-74, or even the larger cohort of surviving men and women aged 40-74 in 1964, there is no straightforward means of identifying those who were residents of Oklahoma City in 1964. A massive effort would still be necessary to locate as many of the 400,000-odd persons overflown as affordable (cf. Table 5-6).

Under realistic assumptions, then, even a massive retrospective cohort study conducted in Oklahoma city today would have insufficient power to detect an effect on cardiovascular disease of the 1964 sonic boom exposure, even an effect far stronger than could reasonably have escaped notice at the time.

Another difficulty posed by a retrospective cohort study is identification of an appropriate

nonexposed comparison group. Since a great many residents of Oklahoma City and vicinity in 1964 were almost certainly exposed to the sonic booms in places other than their own residences, the two most likely sources of nonexposed controls are (1) individuals in the 40-74 age range residing in communities similar in all respects to Oklahoma City, and (2) individuals who were born between 1889 and 1924 and who immigrated to Oklahoma City after 1964.

No nearby municipalities exist that are highly similar in size and composition to Oklahoma City, nor is there any obvious mechanism for identifying all those who moved to Oklahoma City late in life two decades ago. Even if such persons could be identified, they could not be expected to be alive today, nor would they be as likely as the those exposed in 1964 to have surviving relatives who could be located. Obtaining medical information about them (especially prior to their arrival in Oklahoma City) would also be more difficult than for the exposed cohort. Further, there would be far fewer of these people than of the exposed cohort.

Finally, there is little likelihood that the exposed cohort in Oklahoma City could be identified today. Thus, retrospective cohort studies conducted in Oklahoma City today cannot support a reasonable test of sonic boom effects on health.

In short, the nature of exposure and of information available in Oklahoma City do not lend themselves to a retrospective cohort study, since the primary premises of this study design clearly cannot be satisfied. There is no specifically identifiable cohort of people known to have been exposed to sonic booms in 1964 who can also be shown, from medical or other records, to have been free of cardiovascular disease prior to the exposure. Even if identification of a sample of (hypothetically) exposed and nonexposed 1964 residents or their relatives were possible to construct, locating and tracking very large numbers of individuals, many of whom would not be eligible for participation in the study, would be highly impractical.

Use of such an approach makes it impossible to know if some individuals in the original cohort whose exclusion would drastically alter the results of the study had been missed. Interviews with surviving relatives, even if achievable, could not be expected to provide complete or reliable health information, either for the time period prior to 1964 or afterward, nor could medical or death records. Given the misclassification bias inherent in such sketchy data, the 1964 sonic boom exposure would have to have created an effect of epidemic magnitude (one far greater than any reported in the literature, and far greater than could reasonably have escaped notice at the time) to be detected by statistical analyses of health data available for study today.

Although information on exposure at the individual level is of the greatest value in quantifying the relationship between exposure and health outcomes, well documented place exposures (e.g., mean values of distributions of probabilities of sonic boom overpressures) for the entire cohort are not valueless provided that they represent a reasonably good estimate of the noise levels to which cohorts were exposed. However, use of group rather than the individual data severely limits the potential for drawing causal inferences from the study results.

A retrospective cohort study design employing place measurements of exposure could be greatly enhanced by documentation that individuals classified as exposed were present in specified places and times during production of the sonic booms, and likewise, that individuals classified as nonexposed were absent from such places at such times. It is not now possible to obtain such documentation of place exposure for individuals residing in Oklahoma City in 1964.

5.3.7 Examination of Feasibility of a Case-Control Study

Although a cohort study design is infeasible in Oklahoma City for reasons just discussed, it is conceivable that a case-control study design might be feasible. This section summarizes the results of efforts to ascertain whether sufficient information can be recovered from sources available today to permit such a study. The information of immediate interest is that required to perform sample size calculations: the numbers of available cases and controls. (The reader should bear in mind, however, the discussion of Section 5.2.2 concerning the unreliability of sonic boom exposure classification on the basis of residential address.)

5.3.7.1 Sources of Health Information in Areas Overflown Supersonically in 1964

Of approximately 30 general hospitals in the counties overflown supersonically in 1964, only 7 had a capacity of more than 300 beds. Since it is important that cases be selected in a manner that does not introduce bias, systematic differences among the locations, patient populations, and nature of exposure of patients admitted to hospitals of different sizes require some consideration. The most likely biases associated with hospital size are location (smaller health care facilities are more likely to be located in less densely populated areas) and socioeconomic level of patients. Although there is a possibility that personal sonic boom exposure might differ in urban and rural areas (with greater indoor exposure likely in urban areas and greater outdoor exposure likely in rural areas), there is no empirical means of determining the extent of such differences in exposure. Potential selection biases associated with hospital location are overlooked for the time being to permit further development of estimates of numbers of cases and controls.

A written questionnaire was sent in late 1988 to Directors of Medical Records at the 7 largest hospitals[11] in the area overflown in 1964 seeking information about the availability and accessibility of individual patients' medical records in the 1964-1970 time frame.

Five of the 7 largest hospitals reported that they had records in storage from the time period

[11]Since smaller hospitals would contribute relatively few cases to a potential study, their exclusion for the present analysis would affect sample size calculations little. Larger hospitals also tend to have larger catchment areas that are more likely to include patients living in both exposed and nonexposed areas.

of interest. None of the hospitals had computer-based medical record keeping systems in operation in the mid-1960s. Only three of the five hospitals which had retained records from the 1960s now have means for retrieving medical records by diagnosis from 1964 to 1970. There are no data from which to estimate baseline prevalence rates (prior to sonic boom exposure) because there is no information as to whether pre-1964 records exist, nor if they exist, whether they can actually be retrieved. Further, none of the hospitals had any information about their catchment areas during the time of sonic boom exposure or in the years immediately thereafter.

The three hospitals that could retrieve records by diagnosis were asked to review 100 patient records from a single year (1968) to permit estimates to be made of their catchment areas. It appears from these records that 73%, 81%, and 40% of the patients admitted to the three hospitals resided in locations within 16 miles of the 1964 supersonic flight track. It is, therefore, likely that large proportions of both cases and potential controls resided in areas exposed to sonic booms, complicating the task of attributing the findings of a case-control study to impulsive noise exposure *per se*.

The completeness of stored medical records for the years following sonic boom exposure was not verified. However, it is common practice to retain only face sheets and discharge summaries in long-term storage and to discard detailed charts, patient histories and physicians' and nurses' notes. Only two of the three hospitals which could retrieve medical records by diagnosis required the patient's Social Security number or the address of a relative. None of the hospitals required patients' height, weight, or smoking history. Further, none of the hospitals' business offices have kept billing records from the 1960s, precluding linkage of medical records to financial records for purposes of locating patients.

The accessibility to researchers of medical records that can in principle be retrieved from off-site archival storage for the period of interest is governed by institutional review boards at all hospitals contacted. Institutional review boards at two of the three hospitals require consent of the patient (or next of kin if the patient is deceased) for use of medical records. This creates a certain circularity: patients and next of kin cannot be contacted to arrange consent until they have been identified, but hospital policy requires consent before medical records can be examined so that individual patients may be identified in the first place.

5.3.7.2 Examination of Adequacy of Sample Size for a Case-Control Study

Conclusions that can be drawn from sample size calculations are sensitive to a number of assumptions. Assumptions must be made about the following issues to evaluate the adequacy of the sample available for a case-control study include the following:

- the number of archived medical records that can actually be retrieved from off-site storage;

- the completeness and verifiability of diagnoses of such records;
- the number of prevalent vs. incident cases included in the records;
- the number of multiple admissions of the same patients in these records;
- the ability to locate patients or next of kin;
- the willingness of located patients or next of kin to consent to the use of their records in an epidemiologic study; and
- the proportion of potential controls whose exposure to sonic booms resembles that of cases.

Sample size and relative risk estimates are also sensitive to a number of technical assumptions for which no clear guidelines are available:

- Although exposure rates can be estimated for entire catchment areas of particular hospitals (v.s.), relative risk determination for sample size calculations depend on knowledge of exposure rates for nondiseased persons. There is no basis for such estimation in Oklahoma City.
- Sample size and relative risk estimates need to be adjusted for potential confounding variables. Failure to adjust for multiple confounders leads to overly optimistic estimates of power and the relative risk that can be detected for a given sample size.

Since the following sample size and relative risk estimates are based on simplified assumptions, they should be interpreted with caution.

It is estimated that potentially accessible hospital records for the 6.5 years from late 1964 through 1970 contain a total of approximately 2,500 cases of hypertension, 650 cases of myocardial infarction, and 1,600 cases of cardiac dysrhythmias; or on an annualized basis, about 385, 100, and 250 cases of hypertension, myocardial infarction, and cardiac dysrhythmias, respectively. Assuming (1) that each of these records is that of a different individual; (2) that 1964 addresses and consent for participation could be obtained for about half of these, as well as for half of a set of hypothetically nondiseased controls; and (3) that 80% of the patient population had been exposed to sonic booms, then relative risks of about 1.3, 1.7, and 1.4 could be detected at a conventional error rate of $\alpha = .05$ (one-sided) and power of .80 for hypertension, myocardial infarction, and cardiac dysrhythmias, respectively.

At more stringent levels of Type I error ($\alpha = .01$, two-sided) and power of .90 (cf. Section 5.2), the 325 cases of myocardial infarction and their controls would not suffice to detect relative risks much smaller than 2.5, but sample sizes would be adequate for detection of risk of hypertension and cardiac dysrhythmias at risk ratios of 1.52 and 1.71, respectively.

All of these calculations assume no misclassification bias in assessing either personal noise exposure (from place of residence) or disease status (e.g., discrimination of prevalent from

incident cases, or adequacy of diagnosis). Under less sanguine assumptions, the relative risks detectable in a retrospective case-control study based on information from three Oklahoma City hospitals are considerably larger. For example, if next of kin can be located for only 20% of the cases that can be retrieved from off-site storage, the number of cases of myocardial infarction for the half-dozen years following sonic boom exposure is only 130, or less than 2 per month. The relative risk that can be detected with power of .90 and $\alpha = .01$ (two-sided) in a sample of this size is on the order of 5.5 (or greater). The relative risk detectable for hypertension with 500 cases is about 2.0, while the relative risk detectable for cardiac dysrhythmias with 320 cases is 2.5.

Note that all of these calculations are based on *observed* relative risks. The detectability of an effect is severely attenuated by the difficulty in retrospectively classifying both exposure and disease status. The only source of exposure information available from hospital records is place of residence in 1964. Defining exposure as residence within some distance of the flight path provides, at best, a crude estimate of exposure (cf. Section 2.1 and Appendix A) likely to produce substantial misclassification.[12] Hospital records cannot be expected to reveal amounts of time spent at home. While some records might indicate whether patients worked in a geographic area exposed to sonic booms, they could not be expected to indicate the amount of patients' recreational or occupational impulse noise exposure (e.g., gunfire, industrial impact noise). Since many of the cardiovascular patients of the 1964 era were probably among the older members of the community, few of these patients will be alive today, as described in the preceding examination of the cohort study. Thus, verification of these patients' actual sonic boom exposure is essentially unobtainable. Although some relatives might be located and interviewed in an effort to refine exposure information, such interviews cannot be counted on to yield reliable information of the type needed.

Such crude measures of exposure status pose two obstacles to detection of an adverse effect of noise on health. First, they greatly reduce the likelihood of deriving a quantitative dosage-effect relationship from a study. Second, the attendant misclassification bias attenuates the size of the risk ratio that can be detected. Note from Table 2-4 that if the true risk ratio is 2.0, the observed risk ratio could be as small as 1.3 as a result of misclassification bias. Even for hypertension, with 500 cases potentially available to provide usable data, the sample size would be insufficient to detect a sizable effect of sonic booms on cardiovascular health.

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[12]Misclassification of nonexposed is an especially serious concern, since hospitals chosen must be in the exposed area to maximize sample size of exposed cases (violating the requirement that cases and controls be sampled independently of exposure). As noted in Section 5.2.2, even patients who resided more than 16 miles from the flight path cannot be considered free of exposure, since they may have spent substantial amounts of time in boom-exposed areas during the day. An alternative design, in which all hospital cases are considered exposed and controls are randomly sampled from remote nonexposed areas, suffers from many of the same difficulties discussed earlier in the context of a cohort study.

5.3.7.3 Examination of Adequacy of Sampling Methods for a Case-Control Study

According to Breslow and Day (1987, p. 3) the case-control and cohort study designs share the same logical framework of inference, although they appear to be distinct. The rates estimated in a case-control study should refer to rates in some defined population. Inferences drawn from "results of a case control study depend logically upon the interpretation one can give to it as having arisen by sampling from some underlying cohort. The less clear the definition of the underlying population, the less confidence can be put in the results of the case control study."

Thus, a major constraint on development of a meaningful sampling process for a case-control study of the cardiovascular effects of sonic booms experienced by residents of Oklahoma City is the same as that described for a cohort study: namely, the difficulty of defining an appropriate population at risk from which to sample, when exposure occurred in the distant past, was place-related, and of relatively short duration. The epidemiologic literature offers little practical guidance in this respect.

The population at risk is that group of individuals who are at a defined risk of becoming diseased. Cases and controls should ideally be comparable on the *a priori* probability of exposure; that is, they should be drawn from the same population. Breslow and Day (1980, p. 16) expand on this criterion as follows (in the context of detection of cancers):

> "...even a case-control study which is not population-based does derive from a hypothetical population, being those individuals who, if they were to develop the cancer under study, would be included as cases but are otherwise potential controls."

It is obvious that cases whose disease develops outside the population at risk (in the present case, outside the 1964 population of Oklahoma City who could have been exposed to sonic booms) are irrelevant to etiology in the study of the population of interest.

A frequently used approach for assuring that rate ratios estimated in a case-control study refer to some defined population is to sample from all cases in a defined area for a given time, and to select controls from this same referent population. This approach assumes that the population is relatively stable. This assumption is critical when the exposure is of relatively short duration and is quantified by place measurements. The data available from existing health facilities in Oklahoma City (in exposed and nonexposed areas) do not allow identification of all cases of cardiovascular disease which developed in the area during the period of interest. High migration rates and sketchy data about past health service patterns make it extremely difficult to estimate whether cases and controls can be selected such that they were equally likely to have been residents of Oklahoma City in 1964. Even if all cases in the area were determinable, and if an unbiased sampling of controls were possible, medical record reviews or personal interviews would be required to verify that each subject belonged to the desired population at risk. Even this very costly and time-consuming process would not resolve the issue, however, since

individuals in the study population at risk who moved out of Oklahoma City or received health care outside of the area would remain unknown.

Under realistic assumptions, then, even a case-control study sufficiently large to detect small risks would be very unlikely to provide data from which causal inferences could be drawn. Any observed association, whether positive or negative, could be questioned because of the potential for selection bias. Since available information does not permit a demonstration of the absence of selection bias, it would be impossible to refute criticism of this sort.

5.3.7.4 Approximate Cost of a Case-Control Study

Despite the considerable likelihood of uninterpretable results, the merit of conducting a case-control study in Oklahoma City might still be argued if one could be conducted at little cost; however, such is not the case. One estimate of the total cost of conducting such a study[13] exceeds $1,500,000. Approximately 17 person-years of effort would be required over a period of about three calendar years, of which more than half (about 24,000 person-hours) would be required to locate case files and find 1964 and current addresses of potential study participants.

5.3.8 Summary of Impediments to Conduct of Epidemiologic Study in Oklahoma City

The following logical and statistical constraints on the feasibility of epidemiologic study of the health consequences of sonic boom exposure in Oklahoma City are noteworthy:

- Since medical records of individuals for the time period preceding sonic boom exposure are reported by hospitals to be unobtainable, prevalent cases would be difficult to distinguish from incident cases. Given that the induction period for cardiovascular disease is almost certainly several years or more, there is little biologic basis for considering many of patients hospitalized with cardiovascular disease in the first 2-3 years following the 1964 sonic boom exposure as incident cases in any event.
- The population at highest risk of developing sonic boom-induced cardiovascular disease cannot yield adequate samples for a cohort study to reveal the existence of any disease which has a latency of more than a few years or which affects only a small proportion of the susceptible population.
- An appropriate sampling scheme cannot be constructed for either a retrospective (historical) cohort or a case-control study. A cohort of individuals cannot be identified who are known to have been exposed to sonic booms in 1964, who can be shown through records to have been free of cardiovascular disease prior to exposure to the sonic booms, and who can be followed through records for the last 10-20

[13]This estimate is based in part on information prepared in November, 1988 by ENSR Health Sciences.

years after exposure to ascertain cardiovascular outcomes. Similarly, a scheme that would allow sampling in a case-control study to be independent of exposure (and thereby allow estimation of of the relative magnitude of incidence rates of disease in the exposed and nonexposed groups) is not feasible, since (1) the specific population at risk is unknown, (2) there has been considerable migration into and out of Oklahoma City since 1964, and (3) catchment areas for health facilities used by exposed and nonexposed individuals in Oklahoma City in 1964 cannot be adequately specified because data were not systematically obtained by hospitals in the 1960s and what little may have been collected is not now available.

- The greatest noise dose any individual could have experienced was 1,253 booms of moderate intensity within a 6 month period.[14] If development of sonic boom-related cardiovascular symptoms requires more than 6 months' exposure to a maximum of 8 sonic booms per day, inferences about the existence of such a disease cannot be drawn from information available in Oklahoma City.

Although it is perhaps tedious to do so, it is also important to list the practical impediments to study of disease that might have been created by the circumstances of sonic boom exposure in Oklahoma City:

- No documentation is available of the actual noise exposure (that is, the impulsive noise dose) of any individual or class of individuals in the overflown areas. This lack of information creates errors of misclassification of exposure that cannot be well quantified, but are almost certainly appreciable.
- Only coarse scale (i.e., areas of scores of square miles), place-oriented, probabilistic estimates of population noise doses can be constructed from available information. Inferences drawn from such crude noise exposure estimates are very likely to suffer from ecologic fallacies.
- Since no accurate definitions of populations at varying degrees of risk can be established, reliable dosage-effect relationships cannot be developed.
- Locating and obtaining consent from sufficient numbers of individuals or physicians for access to medical records and participation in a study of the sort under discussion is problematical.
- The limited information about confounding variables such as obesity and smoking history that is likely to be preserved in medical records of the period make it difficult to interpret potential positive findings. The incomplete recording of Social Security number and other identifying information reduces the likelihood of locating relatives and survivors for interviews which could conceivably yield such information.
- Net immigration and natural population increase have greatly increased the sizes of populations in overflown areas, making it more difficult to discern the prevalence of

[14]This approximates the total subsonic aircraft noise exposure of just a few days in the vicinity of a major civil airport.

morbidity attributable to cardiovascular disease in the surviving members of the cohort of 1964 residents who may have been exposed to sonic booms.

5.4 Chronic Exposure to Sonic Booms

5.4.1 White Sands Missile Range

People working and living in the vicinity of White Sands Missile Range have been chronically exposed to sonic booms for many years. The total number of people so exposed is likely to number in the hundreds, if not thousands. Members of this population include the range work force; military personnel who work in or near range facilities (including some stationed at Stallion Gate, Holloman Air Force Base, and elsewhere in proximity to the range); contractor personnel who service range facilities (such as those who maintain the ACMI equipment on Oscura Peak); and ranchers and other civilians who live north of the range in the vicinity of the hamlet of Bingham, New Mexico (population 50), and elsewhere in the vicinity of the range boundaries (e.g., Socorro, New Mexico). This population has probably been stable for a lengthy period of time, since the range is a major source of employment in south central New Mexico, and the area has not experienced major population shifts. A disadvantage of this site for prospective study, however, is the wide dispersal of the population within the range, and the fact that flight paths are deliberately designed to avoid populated areas.

It is possible that a meaningful prospective study of health consequences of sonic boom exposure could be conducted in the vicinity of White Sands Missile Range. The attractiveness of this opportunity is enhanced by (1) the existence of an elaborate ACMI system for tracking and recording three dimensional positional information for aircraft engaged in supersonic training exercises, and (2) a recently terminated large scale acoustic measurement program for sonic booms conducted in the area for nonepidemiologic purposes by the Tactical Air Command.

It may, therefore, be worthwhile to determine through interviews with range and local government personnel whether the following issues can be resolved:

- What historical information can be obtained (or inferred) about the nature and frequency of supersonic flight activity in the vicinity of the Missile Range?
- What information and estimates are available about the size, composition, stability and geographic distribution of the population currently exposed to sonic booms?
- Where does this population obtain health care, and what health care records are available from what sources for what periods?

If the adequacy of exposed and control populations, exposure conditions, acoustic

measurement, and accessibility of medical information can be documented, it would be reasonable to suggest a prospective epidemiologic study. The two key elements of such a study would be:

- initial and periodic (perhaps annual) blood pressure measurements or more detailed physical examinations of test participants; and
- continuous monitoring of exposure to sonic booms.

One major disadvantage of a prospective study at White Sands Missile Range is the wide dispersal of the affected population. There are no sizable towns in which even moderate numbers of residents can be recruited for study. As a result, study in this site would be more costly than in some of the alternate sites discussed in Chapter 6.

5.4.2 Areas Overflown by SR-71

The SR-71 supersonic reconnaissance aircraft has overflown a number of small towns in the northern United States (e.g., Darby, MT) for a number of years. Although the frequency and timing of overflights is sensitive information, there is reason to believe that overflown towns have experienced as many as several booms per month. Since SR-71 operations may be discontinued in the near future, opportunities for prospective study of the overflown population are uncertain. Retrospective analyses are likely to be complicated by national security issues.

5.5 Chronic Exposure to Subsonic Civil Aircraft Noise

Between one and two million people in the United States are believed to reside in airport neighborhoods exposed to high levels of subsonic aircraft noise (EPA, 1978). Several health effects studies that have been conducted in such environments are reviewed in Section 3.3. As noted in Section 3.4, even the most carefully conducted of these studies have failed to provide clear evidence of adverse effects of aircraft noise on health. The feasibility and merit of yet another ecologic airport study are discussed in the following sections.

5.5.1 Design of an Ecologic Study in Airport Environs

To represent a worthwhile improvement over previously conducted studies, a newly designed ecologic study of the effects of aircraft noise on public health in airport environs would have to overcome some of the well-known limitations of these studies. For example, it would be necessary to develop very detailed estimates of noise exposure patterns in several neighborhoods in the vicinity of a large civil airport over extended periods. In practice, this requirement implies careful documentation of changes in aircraft operations from about the time of introduction of jet transports into the civil fleet (say, 1960) to the present. Quantitative estimates of exposure levels in several neighborhoods with varying levels of aircraft noise exposure would have to be developed and reconciled among different information sources (published noise contours, air travel guides, airport records, etc.) for several subintervals of several years' duration. Confidence intervals for the exposure estimates would also be developed so that neighborhoods could be selected for study in a manner that minimized misclassification error.

Since the most likely sources of information about prevalence rates of various health conditions in these airport neighborhoods are insurance carriers, HMOs, hospitals and physicians in private practice, a comprehensive list of names, addresses, catchment areas, and nature of such organizations is required. From this list, a set of appropriate organizations might be chosen. Criteria for selection would include:

- A catchment area for exposed groups which includes neighborhoods close to airport flight paths; or a catchment area for nonexposed groups which includes neighborhoods at suitable distances from major airports and noisy highways that is similar in demographic characteristics and stability to the exposed neighborhoods;
- Quality (completeness and temporal resolution) of records kept during the relevant period;
- Availability of residential addresses for individuals presenting symptoms of cardiovascular morbidity;
- Absence of nontechnical impediments to providing access to required records, such as confidentiality and cooperation.

Each cooperating organization would have to provide information from which cardiovascular morbidity could be inferred for the period of interest: incidence rates of arrhythmias, hypertension, and myocardial infarcts. Detailed information would also be needed about other characteristics of the selected neighborhoods, such as stability, socioeconomic level, and other demographic characteristics.

Detailed information would also be needed about such cardiovascular risk factors as smoking, diet, cholesterol level, exercise, alcohol use, and the like. Such information would preferably be obtained from a sample of long term neighborhood residents. At best this

information would allow an aggregate estimate or mean level of risk on each factor for the exposed and unexposed areas.

One location at which a study of this type might be possible is the vicinity of Los Angeles International Airport (LAX), for which extensive records of flight activity are available for simple flight paths that overfly densely populated residential areas. This location or a similar civil airport area might, therefore, be amenable to ecologic study, using aggregate data collected retrospectively.

At the time of this writing not enough is known to make a detailed assessment of the feasibility of an ecologic study of airport noise effects on health at any particular site. However, certain aspects of the utility of such study are apparent even without site-specific investigation. For example, many of the logical and practical difficulties of case-control and cohort studies noted earlier in this chapter may also be expected in airport environs.

The feasibility of a ecologic study depends in part on the quality of morbidity and mortality data available over an extended period, preferably 10 to 20 years. Once collected, data can be evaluated through multiple time series analysis.

Use of incidence data in an ecologic analysis gives greater validity to inferences derived from such studies. The Anton-Guirgis et al. (1986) study clearly demonstrates the difficulties of obtaining morbidity data, either incidence or prevalence, for areas small enough to be homogeneous on many confounding variables. In the case of a complex multifactorial disease such as hypertension, conclusions drawn from ecologic studies of airport environs where data do not allow adjustment for confounding variables are likely to exaggerate the true impact of aircraft noise on health.

5.5.2 Preliminary Evaluation of Studies of Effects of Civil Airport Noise on Cardiovascular Health

Although a detailed investigation of an ecologic study of aircraft noise effects on residents of airport neighborhoods has not yet been done, it is possible to identify several a priori grounds for believing that such a study might not provide clear evidence of the presence or absence of adverse effects of exposure:

- Ecologic study of airport noise suffers from the usual problems of misclassification due to exposure uncertainty. Personal noise exposure may be quite different from the outdoor noise that serves as the measure of exposure in an airport study.
- Any study based on health records assumes that people are treated near their place of residence rather than elsewhere (e.g., near their place of employment).

- Mobility of residents is often high in airport environs. For example, Fidell and Jones (1973, pp. 29-30) noted that a third of the respondents in a panel sample in an airport neighborhood with a noise exposure between L_{dn} = 80 dB and L_{dn} = 85 dB could not be relocated by telephone after only 2 months. A study designed to track residents for a long time is, therefore, likely to suffer from substantial attrition rates that differ for exposed and nonexposed groups. Worse yet, attrition in the exposed group is likely to be function of exposure.

- Matching of unexposed neighborhoods to those that are exposed is likely to be quite difficult. Control of confounders is especially problematical, as is the bias produced by self-selection. People who live near airports do so for diverse reasons. They may well be less sensitive to noise than others, or of lower socioeconomic level than others. While statistical control can be attempted for some confounders, the results of an ecologic study conducted in airport environs can always be disputed on the basis of yet another confounder that was left uncontrolled.

- Classification of those who do vs. those who do not exhibit cardiovascular disease may be substantially erroneous, depending on the quality of the recorded data. If data are gathered from health providers, accurate classification is based on the assumption that those who show cardiovascular symptoms are equally likely to visit health professionals as are those who do not exhibit symptoms, and that professionals use similar diagnostic criteria and record these diagnoses with sufficient detail to classify cases.

A final difficulty associated with an ecologic airport study is the difference in the nature of exposure between airport neighborhoods and communities near MOAs and MTRs (cf. Section A.4.2). In the vicinity of a large civil airport, noise is stable and predictable. Noise produced by flight operations in MOAs and along MTRs, however, is sporadic and largely unpredictable. If the nature of the exposure mediates health consequences, little would be gained from the conduct of such a study. Should aircraft noise in the vicinity of a large civil airport be found to affect health in an urban population, environmental planners would still not be able to predict the extent to which occasional, unpredictable aircraft noise affected health in a rural population. If no evidence were found of adverse health consequences of noise exposure at a large airport, one could easily argue that predictable noise of this sort is less likely to affect health than the potentially startling aircraft noise heard near MOAs and MTRs.

Ecologic studies of airport environs which control for major confounders, verify stability of the population overflown and apply current statistical testing as suggested by Morgenstern (1982) may be useful in suggesting the maximum or upper bounds of the impact of aircraft noise on cardiovascular health if more definitive studies cannot be designed. The problem of designing etiologic-based epidemiologic studies needed to predict the impact of environmental exposures is very real because of the enormous internal migration of the U.S. population, the changes in exposures to noise and other CVD risk factors individuals experience over a period of 5 to 10 years, and the low response to and compliance with study protocols (Erdreich, 1985, p.

87). The inherent danger in using ecologic data to predict impact of aircraft noise on humans is in exaggerating or minimizing the problem. Although policy decisions should constantly be reevaluated in light of new evidence, the difficulty in changing and/or rescinding policies must be considered in deciding what is acceptable for impact analyses. Given the nature of the ecologic design and the data typically available, overestimation of the effect of noise *per se* is more likely than underestimation. For example, if cohort studies in Europe documented a weaker effect under higher aircraft noise exposure than an ecologic study around the LAX airport, could the general public be convinced that noise confers less risk than previously estimated? Nevertheless, ecologic data are better than a hypothetical guess and needless to say, interventions should be made if noise truly increases risk to cardiovascular diseases.

5.5.3 Feasibility of Epidemiologic Studies Abroad

Low altitude, high speed overflights and sonic booms are audible to residential populations in several parts of the world, including Great Britain, Germany, Turkey, Labrador, and Israel among others. However, detailed assessments of the feasibility of studying effects of aircraft noise exposure on nonauditory health outside of the United States were beyond the scope of the present effort.

Ongoing studies of effects of noise exposure on public health are underway in Great Britain and Germany. Opportunities for cooperation in these studies are limited by a number of factors, including the noise sources of interest and the extreme sensitivity of some of the work.[15] Differences in risk factors for cardiovascular disease between American and other populations (e.g., native populations of rural Turkey or Labrador) would also complicate interpretations of study outcomes for Air Force environmental planners.

[15]Aviation Week and Space Technology (12 December, 1988, p. 40), for example, cites intensified public outrage about low-level military flying following the crashes of more than 20 military aircraft during training flights in Germany in 1988.

6. Potential Study Sites for Prospective Epidemiological Study

This chapter explores the availability of sites in the vicinity of domestic MTRs and MOAs suitable for the study of the effects of residential exposure to aircraft noise on nonauditory human health. Such sites must at a minimum contain an adequate population exposed to sufficient aircraft noise levels to support long term prospective epidemiologic studies.

Sites were sought according to the following plan of overlapping activities:

- Develop criteria for rank ordering military airspaces (MTRs and MOAs) listed in the Military Airspace Data Base developed by Oak Ridge National Laboratory (ORNL), (Schweitzer & Saulsbury, 1989);
- Search and link databases to create separate matrices for MTRs and MOAs of rank orderings of military airspaces according to criteria developed;
- Select the highest ranked MTRs and MOAs to pursue identification of military airspaces for epidemiologic study;
- Review selected MTRs and MOAs using detailed sectional aeronautical charts to identify towns overflown by military aircraft as well as nearby nonexposed towns that could serve as controls;
- Verify the accuracy and adequacy of database information about the candidate military airspaces and determine location and size of hospital facilities for identified towns;
- Identify the most suitable sites and evaluate the feasibility of conducting such study.

6.1 Selection of Candidate Military Airspaces

The selection process was designed to identify the dozen most desirable sites for epidemiologic study of military aircraft noise on human nonauditory health, particularly cardiovascular disease. While a study based on a single exposed locale paired with a single nonexposed control locale is economically appealing, such a study is undesirable for a number of reasons.

1. Changes are likely either in the characteristics of military flight patterns or in the willingness of the community to participate in the study over the 5-10 year study period, cutting short research at one or more sites. Selection of multiple sites raises the probability that at least part of the study can continue in the event of loss of a portion of the data collection pool.
2. Findings of a study based on a single exposed and single nonexposed site are

vulnerable to the criticism that they are idiosyncratic to the selected sites and not generalizable to other, unselected populations. Conducting the study at multiple sites enhances the generalizability of results.

3. In the likely event that no statistically significant relationship is found between noise exposure and cardiovascular functioning, the study is subject to the criticism that power was too low--the sample size was inadequate to show small but meaningful change in cardiovascular health as a result of aircraft noise. Use of multiple sites will produce larger sample sizes, resulting in a study less vulnerable to the criticism of low power.

6.1.1 Development of Criteria

The most suitable sites for epidemiologic study are those with population centers containing the greatest and densest concentrations of people with the highest exposure to military aircraft noise. The ORNL database includes information about noise exposure associated with various aircraft as well as rough estimates of total overflown population and number of overflights. The database also contains information which allows several other factors to be considered in evaluating the relative suitability of these sites.

- The effects of overflights on human health, if any, are more likely to occur under low altitude portions of flight tracks. For MTRs only, the database estimates population in the low altitude portions of flight tracks.
- Time of day of overflights may also have an influence on any relationship between noise exposure and human health.[16]
- Day of the week is assumed to be associated with both accurate measurement of noise exposure and likelihood that such exposure is annoying. Weekend overflights may produce more reliable data for epidemiologic study.
- For MOAs only, the data base contains information about the proportion of overflown population that is rural (fewer than 500 people per square mile). Epidemiologic study of the type most likely to produce unambiguous results is inefficient unless conducted in areas of relatively high population density.
- For MTRs only, the data base contains information on the population beneath the area in which the route duplicates itself. Noise exposure is greater for those sites under duplicated routes.
- Not all overflights produce equal amounts or kinds of noise; differences are

[16]As described in Chapter 4, any effects of noise exposure are likely to be mediated by annoyance. People may be more annoyed by nighttime than by daytime overflights. Moreover, they are more likely to be at home during nighttime hours. The relationship between outdoor noise measurement and indoor noise exposure is tenuous at best. It becomes slightly stronger during nighttime hours when noise produced by human activity is diminished. The relationship becomes nonexistent if residents are elsewhere during the noise-producing overflights, as they are more likely to be during daytime hours.

associated with type of aircraft. Types of aircraft operating at each site affect estimates.

The ORNL database is described in detail in Scweitzer & Saulsbury (1989). Portions of the database used for developing exposure criteria are described in Appendix C.

6.1.2 Ranking of MTRs and MOAs

The results of the rank orderings may be found in Appendix C. Table 6-1 shows rank orderings of MTRs according to the exposure criteria described. Rankings are limited to the upper 64 of the 702 routes listed in the database in terms of total overflown population. Rows list routes (MTRs). Columns show rank ordering according to the following scheme.[17]

- Total ranks routes by the most rudimentary measure of noise-exposed population, the product of values for total population overflown by the MTR and values for average number of sorties scheduled per month.
- Weekend ranks routes by the product of values for population for routes scheduled on weekends and values for average number of sorties scheduled per month.
- Nighttime ranks routes by the product of values for population overflown at night and average number of sorties.
- Low Altitude ranks routes by the product of values for population in low altitude portions of the MTR and values for low altitude noise.
- Low Altitude Night ranks routes by the product of values for population in low altitude portions of the MTR overflown at night and values for low altitude night noise.
- Duplicated ranks routes by the product of values for population in portions of the MTR which duplicate themselves and double the values for average number of sorties scheduled per month.
- Duplicated Low Altitude ranks routes by the product of values for duplicated low altitude population and double the values for low altitude noise.

Table 6-2 shows orderings of MOAs according to the exposure criteria described in Section 6.1.1. Listings are limited to the top 23 of the 126 database MOAs in terms of total overflown population. Rows list areas (MOAs). Columns show rank ordering according to the following:

Non-Rural ranks areas by the product of values for nonrural populations beneath the MOA and values for average number of sorties schedulaed per month.

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[17]Appendix C, Section C.1 shows tables of rankings by population and noise criteria as well as tables of raw values on which rankings are based. Small numbers denote highest ranks.

Table 6-1: Rankings of MTRs by Exposure-Related Criteria.

Route	*Total*	*Week-ends*	*Night-time*	*Low Alt. Night*	*Low Altitude*	*Dupli-cated*	*Low Alti-tude Dup.*
IR0023	84	83	71	79	67	26	24
IR0042	64	63	54	49	42	26	24
IR0062	9	9	9	101	84	9	24
IR0069	31	31	25	27	27	5	3
IR0074	52	52	42	71	59	26	24
IR0075	4	4	4	5	5	1	1
IR0084	39	39	31	101	84	26	24
IR0089	73	72	62	82	70	26	24
IR0090	69	68	59	80	68	26	24
IR0133	2	2	2	2	2	24	22
IR0715	25	25	20	101	84	26	24
IR0721	11	11	11	18	18	26	24
IR0726	53	53	43	41	38	23	21
SR0035	81	80	69	77	65	11	10
SR0036	100	98	86	95	80	14	13
SR0037	67	66	57	68	57	11	10
SR0040	71	70	61	70	84	26	24
SR0059	99	97	85	93	79	26	24
SR0225	102	100	88	97	81	26	24
SR0701	59	59	49	59	50	26	24
SR0702	55	55	45	54	45	21	19
SR0707	90	88	77	87	74	26	24
SR0732	51	51	41	53	84	26	24
SR0771	27	27	21	28	28	26	24

Table 6-1: continued.

Route	*Total*	*Week-ends*	*Night-time*	*Low Alt. Night*	*Low Altitude*	*Dupli-cated*	*Low Alti-tude Dup.*
SR0774	91	89	78	88	84	8	8
SR0801	48	48	38	48	84	20	18
SR0823	82	81	70	81	69	26	24
SR0824	88	86	75	85	73	26	24
SR0825	85	84	72	83	71	26	24
SR0826	47	47	37	47	41	10	9
SR0845	66	65	56	63	52	26	24
SR0847	63	62	53	58	49	26	24
SR0873	38	38	30	39	84	7	6
SR0900	1	1	1	1	1	26	24
SR0901	32	32	26	32	31	4	4
SR0902	76	75	64	75	63	26	24
SR0904	37	37	29	38	36	26	24
VR0058	23	23	19	20	20	26	24
VR0086	78	77	66	73	61	26	24
VR0087	7	7	7	7	7	26	24
VR0088	6	6	6	6	6	12	11
VR0092	23	23	19	20	20	26	24
VR0093	75	74	63	69	58	25	23
VR0095	12	12	12	10	10	26	24
VR0704	19	19	90	16	16	26	24
VR0705	15	15	90	14	14	26	24
VR0707	34	34	90	31	30	26	24
VR1016	83	82	90	78	66	26	24

Table 6-1: continued.

Route	*Total*	*Week-ends*	*Night-time*	*Low Alt. Night*	*Low Altitude*	*Dupli-cated*	*Low Alti-tude Dup.*
VR1023	87	102	74	23	23	13	7
VR1050	42	42	33	40	37	26	24
VR1051	33	33	27	34	33	26	24
VR1052	17	17	15	25	25	26	24
VR1055	18	18	16	17	17	26	24
VR1056	17	17	15	25	25	26	24
VR1059	10	10	10	11	11	26	24
VR1060	29	29	23	29	29	26	24
VR1064	20	20	17	15	15	26	24
VR1068	49	49	39	50	43	26	24
VR1145	16	16	90	21	21	26	24
VR1146	43	43	90	42	39	26	24
VR1653	20	20	17	15	15	26	24
VR1721	97	95	83	91	77	26	24
VR1751	21	21	18	19	19	26	24
VR1752	5	5	5	4	4	19	15

Weekend ranks areas by the product of values for nonrural population for areas scheduled on weekends and values for average number of sorties scheduled per month.

Nighttime ranks areas by the product of values for nonrural population in areas overflown at night and average number of sorties.

Low Altitude ranks areas by the product of values for nonrural population and values for low altitude noise.

Low Altitude Noise ranks areas by the product of values for nonrural population and in areas overflown at night and values for low altitude night noise.

6.2 Selection of Highest Ranked Candidates

Identification of the most desirable candidate sites for epidemiologic study is based on rankings in Tables 6-1 and 6-2, which give greatest weight to low altitude nighttime and weekend noise exposure. MTRs and MOAs without nighttime activity were not considered further.

6.2.1 Review of Selected Military Airspaces

The most highly ranked sites derived from the ORNL Military Airspace Data Base were further reviewed for purposes described below.

6.2.2 Accuracy and Completeness of Database Information

The documentation for the ORNL Military Airspace Data Base (Schwietzer & Saulsbury, 1989) lists sources for much of the information contained in the files. Where possible, such sources were consulted to spot check the accuracy of transfer of information to disk files.

Air Force Magazine's "Guide to U.S.A.F. Bases at Home and Abroad" (May, 1987, pp. 188-195) was listed in the data base as the source for scheduling commands. A check revealed several discrepancies, noted in Appendix C, Section C.2. Contacts with airspace managers to update information (see following section) revealed that most scheduling telephone numbers listed in the database were out of date. Several discrepancies were found between the database and the Area Planning document reported as the source for much of the information on MTRs, AP/1B, 18 Dec 1986 (DMA Aerospace Center, 1986b). Similarly, some of the information in

Table 6-2: Rankings of MOAs by Exposure-Related Criteria.

MOA Name	*Non-Rural*	*Week-end*	*Night-time*	*Low Altitude*	*Low Alti-tude Night*
BIRMINGHAM 2	14	6	9	11	11
BISON	12	11	12	19	19
BRADY LOW	--	29	--	--	--
BRUSH CREEK	15	3	4	10	10
BULLDOG A	5	29	3	5	5
COMPLEX 1	1	1	1	1	1
EGLIN B	11	29	20	19	19
EGLIN E	17	29	27	13	13
EUREKA LOW	--	--	30	--	--
FARMVILLE	9	29	8	8	8
FREMONT	--	29	--	19	19
GAMECOCK C	7	4	7	6	6
GAMECOCK I	19	29	19	15	15
HOTROCK 2	2	2	30	3	3
JENA 1	8	7	30	7	7
MORENCI	3	29	14	19	19
QUICK THRUST E	16	29	30	12	12
QUICK THRUST F	18	29	30	14	14
QUICK THRUST I	1	29	30	2	2
SNOWBIRD 2	--	29	--	19	19
SYRACUSE 2	4	5	30	4	4
SYRACUSE 3	10	12	30	9	9
TYNDALL F	13	29	18	19	19
WILLIAMS 1	--	29	--	19	19

the Restricted Area database files failed to correspond to data in the appropriate Area Planning document, AP/1A, 23 Oct 1986 (DMA Aerospace Center, 1986a). While the latter document was also cited as the source for much of the information in the MOA files, AP/1A contains only scheduling information. The actual source of information about MOAs in the ORNL database appears to be a document from DMA Aerospace Center (1987). Comparisons between that document and the MOA files in the database revealed a fairly high degree of accuracy. Spot checks of the database files against the 3 documents revealed errors in about 10% of the routes/areas, detailed in Appendix C, Section C.2.

Other database files listed sources not readily available, such as "ORNL's Computing and Telecommunications Division." Data in those files were therefore checked for internal consistency (if an item was listed in more than one portion of the file) and completeness. A number of problems were found in this check, some of which are noted in Appendix C, Section C.2.

Accuracy of database information was also evaluated as part of the search for a provisional list of towns compiled from sectional aeronautical charts. Obvious discrepancies were noted. This finding led to a search of sectional aeronautical charts in the vicinity of MTRs and MOAs for large towns, and a scan of large towns to see if they were in MOAs or under MTRs. This search identified 5 additional towns exposed to numerous low altitude night overflights which were added to the ranking process.

Attempts made to rank order Restricted Areas (RA) using the ORNL database files were abandoned due to large amounts of missing or incomplete data in a number of fields critical to the ranking process (cf. Appendix C, Section C.2).

6.2.3 Updating Database Information

Data sources cited in the documentation for the ORNL Military Airspace Data Base (Schweitzer & Saulsbury, 1989) for which dates were listed dated from late 1986 to midyear 1987. ORNL sources were undated. Even if accurate, there is considerable likelihood that characteristics of overflights as well as overflown populations may have changed in the interim. Further, there is similar likelihood that such characteristics will change over the course of the 5-10 year duration of a prospective epidemiologic study.

The current copy of AP/1B (DMA Aerospace Center, 1989b) for MTRs was therefore consulted to verify the current altitude ranges, extents, and hours of operation of candidate routes. Personnel at the airbases responsible for scheduling each candidate MTR and MOA provisionally selected were contacted to verify actual minimum altitudes in the vicinity of affected towns and current flight usage. Little consistency was observed in statements about

minimum altitudes for overflying towns, suggesting some degree of pilot discretion in appropriate minimum flight altitudes.

Many of the provisionally selected towns are located in airport "bubbles" on sectional aeronautical charts. While the charts specify a minimum altitude of 700 feet in these bubbles, most of the airspace managers interviewed referred to an Air Force limitation of 1,500 feet above or three nautical miles beyond public use airports. Some airspace managers cited an Air Force limitation of 1,000 feet in such areas. Other airspace managers referred to "the FAA guidelines", usually quoted as 1,000 foot minimum altitude for any congested area (or settlement) within a 2,000 foot radius, and 500 foot minimum altitude for an uncongested area or any object on the ground. One manager cited a 2,000 foot minimum altitude for congested and 1,000 foot minimum for uncongested areas.[18] In any event, no definition of "congested" is available. For one MOA, a minimum of 3,000 feet over any community was cited by the airspace manager, although the restriction shown in the database was 1,200 feet.

Minimum altitudes quoted by managers for the selected military routes and operating areas are well above the minimums listed in the database in most cases. For estimating noise exposure, it cannot be assumed that any town will be consistently overflown at altitudes less than 1,000 feet, an altitude that cannot be considered "low" for noise computation from information in the ORNL database. Exposure estimates based on numbers of monthly overflights are also subject to some uncertainly. The documentation for the ORNL Military Airspace Data Base (Schweitzer & Saulsbury, 1989) makes it clear that only scheduled flights for each aircraft type are recorded in the files. Flight schedulers were thus contacted to estimate the relationship between scheduled and actual overflights for the provisionally selected sites.

Usage varies considerably over the course of the year for many routes and areas. Most of the values used to estimate noise exposure are based on figures for the first half of 1989. Although averages are reported, they may be based on highly discrepant monthly usage. Some of these averages correspond quite closely to database values, while others diverge sharply. For example, the ORNL database reports 23 flights per month for one MTR while the current airspace manager reports 76 overflights. For another, flight usage decreased from 200 scheduled flights listed in the database to current usage of 80 flights per month.

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[18]The actual Federal Aviation Regulation is as follows: "Except when necessary for takeoff or landing, no person may operate an aircraft below the following altitudes: (a) *Anywhere*. An altitude allowing , if a power unit fails, an emergency landing without undue hazard to persons or property on the surface. (b) *Over congested areas*. Over any congested area of a city, town, or settlement, or over any open air assembly of persons, an altitude of 1,000 feet above the highest obstacle within a horizontal radius of 2,000 feet of the aircraft. (c) *Over other than congested areas*. An altitude of 500 feet above the surface except over open water or sparsely populated areas. In that case, the aircraft may not be operated closer than 500 feet to any person, vessel, vehicle, or structure."(Federal Aviation Regulations, 1989, paragraph 91.79.)

In general, information collected in interviews with flight schedulers was far less precise for MOAs than for MTRs. For some MOAs, total numbers of operations were reported without regard to aircraft type. In other MOAs, airspace managers claimed that no records were kept of number of sorties. Some MOAs are assigned to different users by time period, with no tracking of type of use during those times. One manager who kept no records reported "heavy daily usage." Another reported that usage had not changed in the past three years, and thought the database value quoted to him sounded "about right." For such MOAs, ORNL database values were used for estimating number of sorties.

It is apparent that prospective epidemiologic studies cannot rely solely on reported military airspace usage for estimates of noise exposure. To be useful in developing dose-response relationships, actual noise exposure must be periodically monitored on site as part of the data collection process.

6.2.4 Supersonic MOAs and Corridors

Information was sought outside the ORNL database to identify supersonic MOAs, ranges, and corridors, in an attempt to identify sites subjected to sonic booms.

With few exceptions, supersonic MOAs are vast areas lacking sizable communities. White Sands Missile Range is a typical example. People living and working in the vicinity of the range have been chronically exposed to sonic booms for many years. While the total number of people so exposed is likely to number in the hundreds if not thousands, residents are widely dispersed and flight paths are deliberately designed to avoid populated areas. As a result, there are no sizable towns in which even moderate numbers of residents can be recruited for participation in a long term study.

There are a few communities with populations ranging from 3,500 to 5,000 in the Sells Low and Baghdad 1 MOAs in Arizona, and within the Edwards AFB supersonic corridor in California. These are considered in the ranking process of the following section.

6.3 Identification of Exposed and Control Towns

Sectional aeronautical charts were used to trace MTRs and locate MOAs with highest ranked exposure. Only those routes and areas with minimum altitude levels under 3,000 feet were selected for further review (with the exception of a supersonic MOA with a minimum altitude of 4,000 feet.) Towns were identified within the boundaries of the selected military overflight areas. The same charts were used to identify nearby towns of similar size and characteristics distant enough from military aircraft overflights to provide relatively unexposed, control populations for comparison. The charts were also used to identify overlapping military airspaces (e.g., MOAs which also contain MTRs, as well as overlapping MTRs.) Also identified were RAs which overlap MTRs and MOAs.

The ORNL database lists population sizes for entire MTRs and MOAs rather than figures for individual towns. Initial selection of towns was based therefore on the size of designated symbols in sectional aeronautical charts. Populations for each of these towns were checked in the 1988 County and City Data Book (U.S. Bureau of the Census, 1988) which reports 1986 population for all "places" with populations greater than 2,500. Contacts were made with such civic organizations as county or town planners, chambers of commerce, or city clerks to ascertain populations for unlisted towns, and to update population estimates.

The U.S. County and City Data Book (U.S. Bureau of the Census, 1988) also lists number of hospitals and hospital beds for communities with more than 25,000 inhabitants. For smaller towns, the sources contacted for population updates were also asked about hospital facilities. If a community had no hospital, information about nearby hospitals was sought. Hospitals were not contacted for detailed information about size, catchment area, record keeping practices, accessibility of records, or willingness to participate in a long term study.

6.4 Selection and Ranking of Towns

Table 6-3 shows 38 towns selected for consideration for epidemiologic study on the basis of the ORNL database and the verification/updating processes. The columns of this table show:

- Rankings by current population estimate;
- Rankings by estimated number of overflights per month;
- Rankings by the product of population and number of overflights;

- Whether overflights are scheduled 24 h per day (T = yes, F = no)[19];
- Whether weekends are scheduled (T = yes, F = no); and
- Overall ranking of suitability of town for prospective epidemiologic study. Double weight is given to 24-h scheduling; 1.5 weighting is given for weekend scheduling. MOAs are given a weight of 0.33 to compensate for an average area approximately 3 times the width of the average MTR.

Table 6-4 provides the values underlying the rankings in Table 6-3, in addition to several other characteristics associated with these towns. The columns of this table show:

- Estimated 1989 population of exposed town;
- Estimated number of overflights;
- The product of population and overflights;
- Number of hospitals accessible to the exposed town,
- Distance to the nearest hospital in miles (with zero entered if a town contains one or more hospitals),
- Estimate of minimum altitude in feet (altitudes less than 1000 ft may not actually be attained); and
- Identification of military airspace over town (MTR, MOA and/or RA).

None of the duplicated routes or areas identified from the ORNL database contained towns appropriate for epidemiologic study. No "low altitude noise exposure" values are reported since, as discussed previously, it is not clear that any communities are consistently overflown at altitudes below 1,000 feet.

Table 6-5 provides population and hospital information for control towns associated with the selected exposed towns. The columns of this table show:

- Name of exposed town;
- Associated control (unexposed) town;
- Estimated current population of control town;
- Number of hospitals accessible to the control town; and
- Distance to the nearest hospital in miles (with zero entered if a town contains one or more hospitals).

[19]All of the provisionally selected exposed towns are subjected to some level of evening exposure (beyond sunset). For a few of the relevant MTRs and all the MOAs, however, operations usually end sometime before midnight, often between 2200 and 2300 local time.

Table 6-3: Rankings of Towns Identified for Consideration for Epidemiologic Study.

Town Name	*State*	*Current Pop.*	*No. of Sorties*	*Pro-duct*	*Sched. 24 h*	*Week-end*	*Overall Ranking*
RIDGECREST	CA	2	1	1	F	T	1
TEHACHAPI	CA	13	1	2	F	T	2
MOJAVE	CA	16	1	3	F	T	3*
HENDERSON	NC	4	9	8	T	T	4
SALISBURY	NC	3	15	9	T	T	5
HAINES CITY	FL	6	6	5	F	T	6
NEWBERRY	SC	5	13	15	T	T	7
ATHENS	TN	7	14	16	T	T	8
McMINNVILLE	TN	8	14	17	T	T	9
CORDELE	GA	5	16	19	T	T	10
BURLINGTON	NC	1	18	10	F	T	11
PIKEVILLE	KY	8	6	12	F	T	12
DAYTON	TN	14	20	18	T	T	13
BAGHDAD	AZ	19	2	7	F	F	14*
SO. PITTSBURGH	TN	20	11	22	T	T	15
BRIDGEPORT	AL	21	11	23	T	T	16
DENMARK	SC	17	13	24	T	T	17
ABBEVILLE	SC	12	16	25	T	T	18
BAMBERG	SC	22	13	26	T	T	19
TABOR CITY	NC	27	10	27	T	T	20
MURPHY	SC	29	9	28	T	T	21
WADESBORO	NC	18	15	29	T	T	22
WOODRUFF	SC	14	16	30	T	T	23

Table 6-3: continued.

Town Name	*State*	*Current Pop.*	*No. of Sorties*	*Pro-duct*	*Sched. 24 h*	*Week-end*	*Overall Ranking*
FORSYTHE	GA	16	16	32	T	T	24
MONTEZUMA	GA	16	16	30	T	T	24
WASHINGTON	GA	16	16	32	T	T	24
SANDERSVILLE	GA	10	5	6	F	F	27
BLACKVILLE	SC	25	13	33	T	T	28
ASHBURN	GA	20	16	34	T	T	29
GREENFIELD	OH	15	7	11	F	T	30
ROWLAND	SC	30	8	35	T	T	31
AJO	AZ	23	3	4	F	F	32*
ROANOKE	AL	11	17	31	F	T	33
WADLEY	GA	24	5	13	F	F	34
YADKINVILLE	NC	28	15	36	T	T	35
LOUISVILLE	GA	26	5	14	F	F	36
JOHNSONVILLE	SC	31	4	18	F	T	37
HEMINGWAY	SC	32	4	21	F	T	38

*Subject to supersonic overflights.

Table 6-4: Characteristics of Highly Ranked Towns.

Town Name	*Current Pop.*	*No. of Sorties*	*Pro-duct**	*No. of Hospitals*	*Dist. to Hospitals*	*Min. Altitude*	*MOA/ MTR/RA*
Ridgecrest, CA	28639	4166	80.77	1	0	500	Complex 1 IR-2506
Tehachapi, CA	5500	4166	76.60	1	0	1200	Complex 1
Mojave, CA	5000	4166	73.18	3	25	1200	Complex 1
Henderson, NC	16000	142	63.56	1	0	1000	VR1758
Salisbury, NC	25000	66	62.17	2	0	1500	IR0721, VR1721
Haines City, FL	12700	270	65.35	1	0	1000	IR0046
Newberry, SC	13000	80	60.17	1	0	1500	VR0088
Athens, TN	12152	76	59.65	1	0	1500	VR0092, VR0058
McMinnville, TN	11500	76	59.42	2	0	1500	VR0092, VR0058
Cordele, GA	13000	50	58.13	1	0	1500	VR0095
Burlington, NC	39700	38	61.79	2	0	3000	IR0062
Pikeville, KY	6500	185	60.80	1	0	400	IR0075
Dayton, TN	6500	76	56.94	1	0	1500	VR0092, VR0058
Baghdad, AZ	4200	1043	66.42	1	65	4000	Baghdad 1
So. Pittsburgh, TN	4000	99	55.98	1	0	500	VR1052, 1056,92,58
Bridgeport, AL	3858	99	55.82	2	5	500	VR1052, 1056,92, 58,77,78

* 10 $\log_{10}$ of product of current population x number of sorties.

Table 6-4: continued.

Town Name	*Current Pop.*	*No. of Sorties*	*Pro-duct**	*No. of Hospitals*	*Dist. to Hospitals*	*Min. Altitude*	*MOA/ MTR/RA*
Denmark, SC	4434	80	55.50	1	7	500	VR0088
Abbeville, SC	6000	50	54.77	1	0	1500	VR0095
Bamberg, SC	3740	80	54.76	1	0	500	VR0088
Tabor city, Nc	2707	109	54.70	2	10	1500	VR0087
Murphy, SC	2076	142	54.70	1	0	1500	VR0092, 58,1052, 1055,1056
Wadesboro, NC	4404	66	54.63	1	0	1500	IR0721, VR1721
Woodruff, SC	5276	50	54.21	1	0	500	VR0095
Forsythe, GA	5000	50	53.98	1	0	1500	VR0095
Montezuma, GA	5000	50	53.98	1	0	1500	VR0095
Washington, GA	5000	50	53.98	1	0	1500	VR0095
Blackville, SC	2840	80	53.56	1	11	500	VR0088
Ashburn, GA	4000	50	53.01	2	26	500	VR0095
Greenfield, OH	5150	256	61.20	1	0	500	Brush Creek, R-5503A
Rowland, SL	2003	94	52.75	1	10	500	VR0087, IR0062
Ajo, AZ	3500	538	62.75	1	90	3000	Sells Low
Roanoke, AL	6049	43	54.15	1	0	1500	VR1055
Wadley, GA	2960	388	60.60	1	10	1500	Bulldog A

* 10 $\log_{10}$ of product of current population x number of sorties.

Table 6-4: continued.

Town Name	*Current Pop.*	*No. of Sorties*	*Pro-duct**	*No. of Hospitals*	*Dist. to Hospitals*	*Min. Altitude*	*MOA/ MTR/RA*
Yadkinville, NC	2337	66	51.88	1	0	500	IR0721, VR1721
Louisville, GA	2800	388	60.36	1	0	500	Bulldog A
Johnsonville, SC	1750	402	58.47	1	20	500	Game-cock C
Hemingway, SC	1200	402	56.83	1	25	500	Game-cock C

* 10 $\log_{10}$ of product of current population x number of sorties.

Table 6-5: Characteristics of Towns Identified as Possible Controls for Exposed Towns.

Exposed Town	*Control Town*	*State*	*Control Pop.*	*Control Hospital*	*Control Hosp Dist*
RIDGECREST	PALMDALE	CA	55000	1	0
TEHACHAPI	ARVIN	CA	8100	10	25
MOJAVE	ARVIN	CA	8000	10	25
HENDERSON	EDEN	NC	15658	1	0
SALISBURY	SANFORD	NC	16500	1	0
HAINES CITY	NORTH PORT	FL	9940	3	10
NEWBERRY	BARNWELL	SC	19868	1	0
ATHENS	SHELBYVILLE	TN	15000	1	0
McMINNVILLE	SHELBYVILLE	TN	15000	1	0
CORDELE	TIFTON	GA	14000	1	0
BURLINGTON	GREENVILLE	NC	45765	1	0
PIKEVILLE	HARRODSBURG	KY	8300	1	0
DAYTON	SPARTA	TN	9280	1	0
BAGHDAD	CLARKDALE	AZ	2000	1	10
SO. PITTSBURGH	MANCHESTER	TN	8000	2	0
BRIDGEPORT	OLIVER SPRINGS	TN	4000	1	7
DENMARK	BARNWELL	SC	5572	1	0
ABBEVILLE	CHESTER	SC	5500	1	0
BAMBERG	BARNWELL	SC	5572	1	0
TABOR CITY	HOLLY HILL	SC	2500	2	35
MURPHY	OLIVER SPRINGS	TN	4000	1	7
WADESBORO	CHERRYVILLE	NC	5000	2	7
WOODRUFF	CHESTER	SC	5500	1	0

Table 6-5: continued.

Exposed Town	*Control Town*	*State*	*Control Pop.*	*Control Hospital*	*Control Hosp Dist*
FORSYTHE	METTER	GA	4000	1	0
MONTEZUMA	METTER	GA	4000	1	0
WASHINGTON	METTER	GA	4000	1	0
SANDERSVILLE	THOMSON	GA	10500	1	0
BLACKVILLE	NORTH	SC	1290	1	17
ASHBURN	PELHAM	GA	4306	1	9
GREENFIELD	LOGAN	OH	6000	1	0
ROWLAND	GREAT FALLS	NC	2500	1	15
AJO	COOLIDGE	AZ	6700	1	5
ROANOKE	CHILDERSBURG	AL	5082	1	0
WADLEY	METTER	GA	4000	1	0
YADKINVILLE	NO. WILKESBORO	NC	3659	1	0
LOUISVILLE	UNION POINT	GA	1800	1	7
JOHNSONVILLE	HOLLY HILL	SC	2500	2	35
HEMINGWAY	HOLLY HILL	SC	2500	2	35

For each of the exposed towns, efforts were made to locate nearby control towns unexposed to military overflights and, to the extent possible, unexposed to commercial air traffic. In some sections of the United States (particularly in the Southeast, where the greatest numbers of candidate towns are found) such unexposed areas are difficult to find. These, of course, are the very locales containing the highest concentration of candidate exposed towns. Some control towns, as a result, may lie within the path of high altitude MTRs (minimum altitude greater than 6,000 feet). Additionally, these towns may be exposed to flight operations which stray from designated airspace. As a result, monitoring of aircraft noise exposure is as important for unexposed as for exposed towns.

It also should be noted that noise exposure estimates are less precise for MOAs than for MTRs. Flight paths within MOAs are not well defined, so that not all operations may take place in the vicinity of towns within the MOA.

The dozen most highly ranked sites from Table 6-3 and their most suitable control towns are described below:

1. Ridgecrest, CA is the highest ranked town by a large margin. The town has a number of shortcomings as a test site for epidemiologic study of the effects of military aircraft noise on community health, however. First, Ridgecrest is adjacent to and derives much of its support from the China Lake Naval Air Station. As a result, the population consists largely of military personnel and dependents, as well as civilian personnel in direct and indirect service to the military. Ridgecrest may, therefore, be more tolerant of noise produced by military overflights, and if less likely to be annoyed by exposure, is less likely to suffer adverse health effects than a population with less dependence on the military. Second, the actual aircraft noise exposure of Ridgecrest is not necessarily as great as suggested by the information in the ORNL database. The town lies at the northeast corner of R-2506, and near the northeast corner of Complex 1 MOA. Edwards AFB scheduling personnel suggest that most military sorties tend to avoid that area because of the town itself.

The town of Palmdale, while the most appropriate nearby control town for Ridgecrest, is also less than ideal as an epidemiologic study site. Palmdale is a rapidly growing suburb of Los Angeles that is almost twice as large as Ridgecrest. While not strictly in the path of an MTR, MOA, or restricted area, Palmdale is not wholly removed from military airspace. Palmdale is about 15 miles from the edge of the Edwards AFB high altitude supersonic corridor, and about 25 miles from Edwards AFB itself. Palmdale is also the site of a large aircraft manufacturing facility.

2. Tehachapi, CA is a smaller (5500 population), isolated town in a valley at the southern end of the Sierra Nevada mountain range, near the western border of Complex 1 MOA. The closest control town, Arvin, lies well outside the Edwards AFB military airspace. Arvin is a

farming community, possibly of different demographic composition from Tehachapi. Arvin is considerably larger than Tehachapi, but unlike Tehachapi, has no hospitals of its own. Instead, residents of Arvin obtain medical care at a dozen-odd hospitals in Bakersfield, a city about 25 miles distant. Medical monitoring of residents of Arvin would pose greater difficulties than monitoring of residents of Tehachapi.

3. Mojave, CA is a town of about about 5,000 residents well within the borders of Complex 1 MOA and also lies within the high altitude supersonic corridor of Edwards AFB. Mojave has no hospitals, although Lancaster, 25 miles away has 3 hospitals. There are no nearby towns of equivalent size suitable as controls. Arvin is the control town closest in population size, but as a farming community may differ demographically from Mojave. Arvin also poses problems as a control town because of access to the many hospitals in Bakersfield, 25 miles away. An alternate control town is Adelanto, with access to 2 hospitals in the Antelope Valley, about 25 miles distant. Adelanto, however, has a population of about 11,000, making it more than double the size of Mojave and possibly of different demographic composition.

4. Henderson, NC is the most populous of the candidate towns and lies at the southern edge of VR1758, with all except the southernmost portion of the town within the boundaries of the MTR. Although residents are subjected to only 142 fights per month (on average), operations occur both nights and weekends. The major drawback is the 4 nm distance of the center of town from the centerline of the MTR. The most appropriate control town, Eden, also has a single hospital and is quite close in population to Henderson.

5. Salisbury, NC lies beneath IR921/VR1721, a single MTR This is a populous town but is subjected to only 66 overflights per month on average. Flights are scheduled continuously. The town center lies about 5 nm from the centerline of the MTR, so that only the northeast half of the town is within the boundary of the MTR. Salisbury has a single hospital, as does its candidate control town, Sanford. Sanford is somewhat less populous and lies close to the boundary of IR718, a high altitude MTR that is not listed in the ORNL database.

6. Haines City, FL is a large town directly under the centerline of IR46, with an average of 270 flights per month. Flights are scheduled for weekends, but not between midnight and 7:00 AM. Haines City has a single hospital, simplifying medical monitoring. The most suitable control town, North Port, has no local hospital. Residents of North Port have access to 2 hospitals in Port Charlotte, 8-10 miles distant, and a hospital in Venice, about 12 miles away. North Port is a harbor town, and may differ demographically from Haines City, an inland town far from any seaport. North Port lies just beyond the border of IR20, a high altitude MTR not listed in the ORNL database.

7. Newberry, SC is the largest of four candidate towns under VR88 with a population of about 13,000. The town lies directly under the MTR. The 3 towns near the MTR are Denmark,

Bamberg, and Blackville, ranging in population size from under 3,000 to about 4,500. Newberry and Bamberg have hospitals. Denmark residents have access to the hospital facilities at Bamberg, 7 miles away, while Blackville residents access the hospital at Barnwell, 11 miles, away. The 3 smaller towns lie within the boundaries of VR88, but not directly under the centerline of the route.

The most suitable control town for Newberry is Sanford, NC, discussed previously as a control town for Salisbury. The most appropriate control town for both Denmark and Bamberg is Barnwell, a larger town that has its own hospital. For Blackville, the most acceptable control town is North, a smaller town that accesses the hospital facilities at Orangeburg.

8. Athens, TN is fully within the boundaries of VR92/58, a single MTR; the centerline of the MTR crosses the southern end of town. Residents of Athens are subjected to an average of 66 flights per month from that MTR, which is scheduled around the clock. The town lies just beyond the boundary of VR1062/1056, a low altitude MTR with 33 flights per month on average. Athens has a population of about 12,000, with access to a hospital within town limits. The most appropriate control town is Shelbyville, which also has a single hospital. Shelbyville, with a population of about 15,000 is fairly close in size to Athens. Shelbyville lies about 3 nm from the boundary of IR77/78, a high altitude MTR.

9. McMinnville, TN, lies about 3 nm from the centerline of VR92/58, at the point at which the MTR makes a turn of about 80 degrees away from the town. McMinneville has a population of about 11,500 and 2 hospitals within its limits. The most appropriate control town is Shelbyville, previously discussed as a control for Athens.

10. Cordele, GA lies about 9 nm from the centerline of VR95, an MTR that is 10 nm wide on either side of its center. Cordele also lies within Moody 1 MOA, a high altitude military area that is not listed in the ORNL database. Cordele has a single hospital, as does its most appropriate control town, Tifton. The two towns are close in size with population equal to about 14,000 for Tifton and 13,000 for Cordele. Tifton also lies within Moody 1 MOA.

11. Burlington, NC is the second largest town in the list of candidates but has relatively few (38) flights per month, on the average. IR62 lies directly over the west side of town, with the entire town inside the boundaries of the MTR. Flights are scheduled on weekends, but not 24 h per day. Burlington has two hospitals. The most suitable control town, Greenville, is about 30% larger, but has only a single hospital. Greenville is about 6 nm from the boundary of VR1759 and about 8 nm from the boundary of VR85/86; both are low altitude MTRs. About 23 flights per month are scheduled along VR85/86; VR1759 is not listed in the ORNL database.

12. Pikeville, KY is about 3 nm from the centerline of IR75. Pikeville has a sizable number of flights, about 185 per month, but only about 6,500 inhabitants. Flight activity ceases

before midnight. Harrodsburg, with a population of about 8,300 can serve as an appropriate control town, with no nearby military flight activity. Each town has a single hospital.

6.5 Confirmation of Noise Exposure

Aircraft noise measurements were made in Mojave and Tehachapi to attempt to confirm noise exposure produced by military overflights in the most heavily used MOA. Monitoring equipment was set up with SEL thresholds set at 80 dB and L_{eq} recording interval set at 1 h. Continuous monitoring was undertaken for 32 hours in Tehachapi and 44 hours in Mojave.

No aircraft overflights were sufficiently high in level to trigger a recording. With an average of approximately 140 daily flights in the MOA, it is evident that either this was an extraordinarily low usage period, or that flights avoid populated areas within the MOA. Informal interviews with local residents suggested that the latter is the case. Residents reported little aircraft noise, except for an occasional sonic boom heard in Mojave.

6.6 Evaluation of Feasibility of Prospective Epidemiologic Study at Selected Sites

Decisions about the feasibility of epidemiologic research at any of the sites identified must take into account the following:

- No single ideal site or set of sites for prospective epidemiologic study exists. All of the candidates suffer some shortcomings.
- Actual exposure aircraft noise at all of the identified sites is unknown, but probably quite low as compared with exposure near a major civil airport. There are fewer overflights per month in even the most heavily exposed town than are experienced in a single day in the vicinity of an airport like LAX. Since prior studies at such airports have failed to uncover a credible relationship between aircraft noise and cardiovascular health, it is probable that epidemiologic study at the identified MTR and MOA sites may similarly fail to find a relationship. Unless it is unpredictability *per se* that contributes to adverse nonauditory health effects of aircraft noise, epidemiologic research in the vicinity of MOAs and MTRs could well produce the same inconclusive results (i.e., failure to find a statistically significant relationship) already available.
- At best, a long term prospective epidemiologic study of nonauditory health effects of aircraft noise that can be conducted at the identified sites is limited in the kind of information that can be obtained. Without accurate measurement of noise doses, no

quantitative dosage-effect relationship can be established. The most that can be shown without such monitoring is a binary effect; either exposed populations will show statistically significant changes in cardiovascular health as compared with nonexposed towns, or they will not.

- If a study were to be conducted at one or more of the identified sites, it would in a sense be retrospective as well as prospective. Participants will have been exposed for some time prior to commencement of the study. Therefore it will be necessary to obtain estimates of prior exposure and assessment of personal/medical history. In any event, it may be impossible to establish incidence rates (as opposed to prevalence rates) among long-time residents of both noise-exposed and control towns.
- Selection of specific sites for epidemiologic research must await further information about hospital facilities, record-keeping practices, accessibility of records, and willingness to cooperate in epidemiologic research.

7. Alternatives to Community-Based Studies

The potential epidemiologic studies of aircraft noise exposure on the health of residential populations described in the previous chapter have been community-based. Another potential source of data for epidemiologic analyses of noise exposure effects exists, however: large databases of audiometric and other health information maintained by the military services. This chapter examines the feasibility and usefulness of epidemiologic analyses of such military and nonmilitary databases.

Although analyses of information in audiometric databases cannot readily be used to establish causal, quantitative dose-effect relationships between residential noise exposure and health outcomes as described in Chapter 2, it is nonetheless useful for the sake of completeness to inquire (1) whether epidemiologic studies of hearing conservation and other databases could be designed to support causal inferences useful to Air Force environmental planners, and (2) whether studies of such databases that do not possess all of the desired features might be at least peripherally applicable to the main goals of the Air Force.

A number of databases were examined to assess the extent to which they met basic epidemiologic requirements for reliable estimates of:

- aircraft or other occupational noise exposure and audiometric information;
- health outcomes and related information for characterizing the population;
- the effects of possible confounding variables; and
- sampling frame requirements for retrospective cohort and case-control studies.

The adequacy of information in these databases for purposes of epidemiologic research was evaluated in terms of the above requirements. Some of the elements considered in each of the areas were as follows:

- *Noise Exposure*: Type and frequency of noise survey measurements and their availability both for high exposure and low exposure (comparison) populations; availability of surrogate measures such as hours flown by aircraft type, occupational codes, etc.; availability of reference audiograms, TTS (temporary threshold shift) followup audiograms, and audiograms from repeated monitoring; comprehensiveness of noise exposure histories; type, size, and use of personal hearing protection devices and individual compliance evaluations; consistency, completeness, and quality control of individual files; capability for associating personal noise dose to audiometric data; quantity of data actually accessible at present; nature of database software and file structures; capability for linking individual exposure data files with other databases containing health outcomes.
- *Sample Identification and Sampling Frame*: Identification of a total cohort of

individuals such as personnel hired during specified years, either for followup over time or for all cases of hypertension, cardiovascular disease, etc.; location of databases; extent to which databases have been or could be linked by individual record identifiers over time; proportion in cohort who could be followed over time; adequacy of period of followup (preferably ten years minimum for cardiovascular effects); reasons for exclusions from each database; control populations available for comparisons with cases for case-control studies; numbers of individuals in cohorts and numbers of cases of disease.

- *Health Outcomes*: Blood pressure measures, cardiovascular disease diagnoses available; proportion of cohort with complete health record information; standardization, reliability and frequency of measurement of health status; quality control of health data; availability of demographic and confounder information (such as family history, height-weight, age, race, and sex for study of hypertension); capability for linking with other data sets.

The most fundamental limitation of analyses of information in audiometric databases is quantification of the independent variable of current interest, noise exposure. Although it is often impossible to reconstruct noise exposure of individuals, it is reasonable under some conditions to seek a surrogate for noise exposure for which measurements are available. The prime example of a surrogate of this sort is hearing damage.

7.1 Purpose of Audiometric Databases within DoD

It is important to bear in mind that none of the DoD audiometric databases were designed to support epidemiologic analyses of the type of present interest. DoD Instruction 6055.12, dated 6 July 1987, establishes as DoD policy that

> "All personnel routinely exposed to hazardous noise shall be placed in a hearing testing and evaluation program. This program shall include pre-placement, periodic (at least once annually), and termination audiograms."

and also that

> "Results of hearing tests performed for hearing conservation purposes, as well as exposure documentation, shall be a permanent part of an individual's health record."

The Instruction further requires that all audiometric data, as well as noise exposure data, be retained for a minimum of 30 years.

The Air Force's hearing conservation program antedates this Instruction by more than 30 years. The Army and Navy also have long standing hearing conservation programs, and have

recently begun record keeping efforts in accordance with Instruction 6055.12.[20]

The following sections describe audiometric and health-related databases potentially useful for one or more epidemiologic studies. The descriptions are based on site visits to the School of Aerospace Medicine and the Occupational and Environmental Health Laboratory at Brooks Air Force Base, TX; to the U.S. Army Environmental Hygiene Agency at the Aberdeen Proving Grounds, MD and the Directorate of Information Management at Ft. Detrick, MD; and to the U.S. Navy Environmental Health Center in Norfolk, VA and the Navy Regional Data Automation Center, Washington, D.C.

7.2 U.S. Air Force Databases

7.2.1 U.S. Air Force Hearing Conservation Data Registry - HCDR

The origins of this database, maintained by the Occupational and Environmental Health Laboratory, may be traced to a 1956 Air Force regulation requiring at least annual audiometric examinations of Air Force personnel (both military and a smaller number of civilian employees) whose Air Force Specialty Code (AFSC) indicated occupational noise exposure that posed a potential hazard to hearing. Approximately one-third of Air Force military and civilian personnel fall into this category.

The earliest audiometric data centrally received were recorded on Air Force Form 1490. Paper copies of the results of audiometric examinations recorded on these forms were filed alphabetically by Air Force bases in the late 1950s and early 1960s, until the bulk of the paper became unmanageable. Some individuals' records became untraceable for all practical purposes as they were transferred from one duty station to another and were were eventually discarded in the mid-1980s during transfer to the newer forms.

For a number of years thereafter, a subset of audiograms was retained from about 24 bases at which it was believed that audiometric measurements were being conducted in keeping with good practice. Audiograms received from other locations were discarded without processing.

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[20]The armed services have powerful economic incentives to maintain such databases. As seen in Figure 7-1, of the more than $8 billion in total annual compensation paid annually to veterans in recent years for service-related disabilities, hearing damage compensation to retired uniformed personnel accounts for nearly $200 million. These totals do not include Workman's Compensation benefits paid to civilian employees of the armed services for hearing damage suffered during employment. Nearly 10% of living veterans now receive payments for service-related hearing damage. Since these payments are now being charged back to the armed services, appreciable savings can be derived from more effective hearing conservation programs and better documentation of service-connected hearing losses.

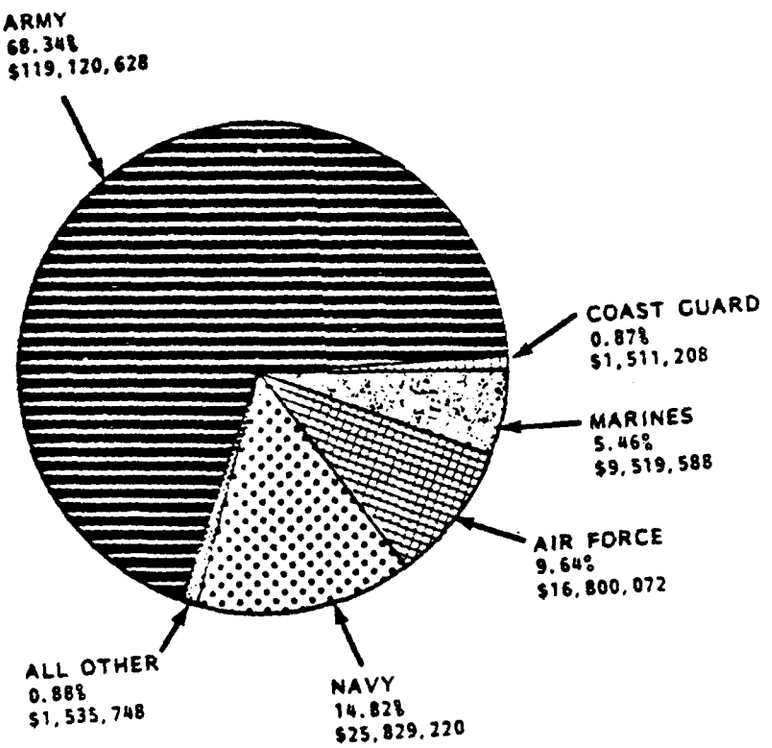


Figure 7-1: Annual Costs for Hearing Damage Compensation, as Reported by Veteran's Administration (Ohlin, 1988).

Initial efforts to transfer information in the paper records to a computer-based system were begun under contract in the early 1960s. A sample of 100,000 records was drawn from the half-million records then available, from which summary information was eventually keypunched onto tabulating cards. Reasonable care was taken to verify keypunched information, but an unknown percentage of records was lost for reasons ranging from incorrect or missing identifiers to partial reporting of audiograms.

Although limited information is available about audiometric records received throughout the late 1960s and early 1970s, fully consistent and complete information in the Registry is available only since March, 1975, the time at which the discarding of audiograms from many installations ceased. A number of technical reports (e.g., Gasaway and Sutherland, 1976, and Sutherland and Gasaway, 1978) have been based on analyses of these post-1975 data.

It was not until July of 1982 that the Registry became a fully computer-hosted database. It now contains in excess of 4 million audiograms. Records of nearly a quarter of a million audiometric examinations are currently being received annually in the Registry. Records in the database are not yet fully accessible, however, for lack of both adequate database management software and computing resources. Work is under way to rehost the database on a DEC VAX mini-computer. The ORACLE database management system will be used for the implementation, rendering the data considerably more accessible for user query and *ad hoc* reporting. Once the new system is operational, records of active duty personnel in the existing database will be converted to the new system. Records of inactive personnel will be archived. The project is focused on optimizing access to active records, but provisions are being made to search archived records. Some data integrity checks will probably be made as data are transferred to the new system, but this process by itself will not materially improve overall credibility and quality of the older data. Newer entries will be based on higher quality data gathered from automated administration of audiometric examinations.

Although details of procedures for administering audiometric examinations in the Air Force have varied over the years, the general nature of the examinations has been fairly consistent. Certified technicians[21] conduct pure tone audiometric examinations using a variety of manually operated, semiautomatic, and (more recently) automatic audiometers.

Adequacy of control over the ambient noise environments in which these examinations are conducted has varied over time and from site to site. The trend over the years has been toward increasing standardization and quality of audiometric procedures consistent with increasing

[21]In the past only audiograms conducted by USAF-certified technicians were considered valid and entered into the Registry. In the future, certification by any of the services will be accepted, but tests conducted by noncertified technicians will continue to be rejected.

availability within the Air Force of prefabricated testing booths and automation of audiometric equipment. One study of several thousand audiograms made with manual audiometers revealed that almost half of these did not meet criteria for tracing (Ohlin, 1988). The Air Force has spent more than $1 million to replace manual audiometers with microprocessor-based units that can operate in fully automatic, semiautomatic and manual modes. These machines also have EIA RS-232C interfaces to transfer audiometric data electronically.

Prior to the acquisition of this equipment, hearing levels observed for each ear at frequencies of 0.5, 1, 2, 3, 4, and 6 kHz had been transcribed manually on a number of forms, as seen in Figures 7-2 and 7-3. Later versions of the forms--Figures 7-4 and 7-5--made provision for recording ancillary information, such as use of hearing protection during noise exposure, calibration dates of audiometric equipment, and a thirteen digit place-of-work code.

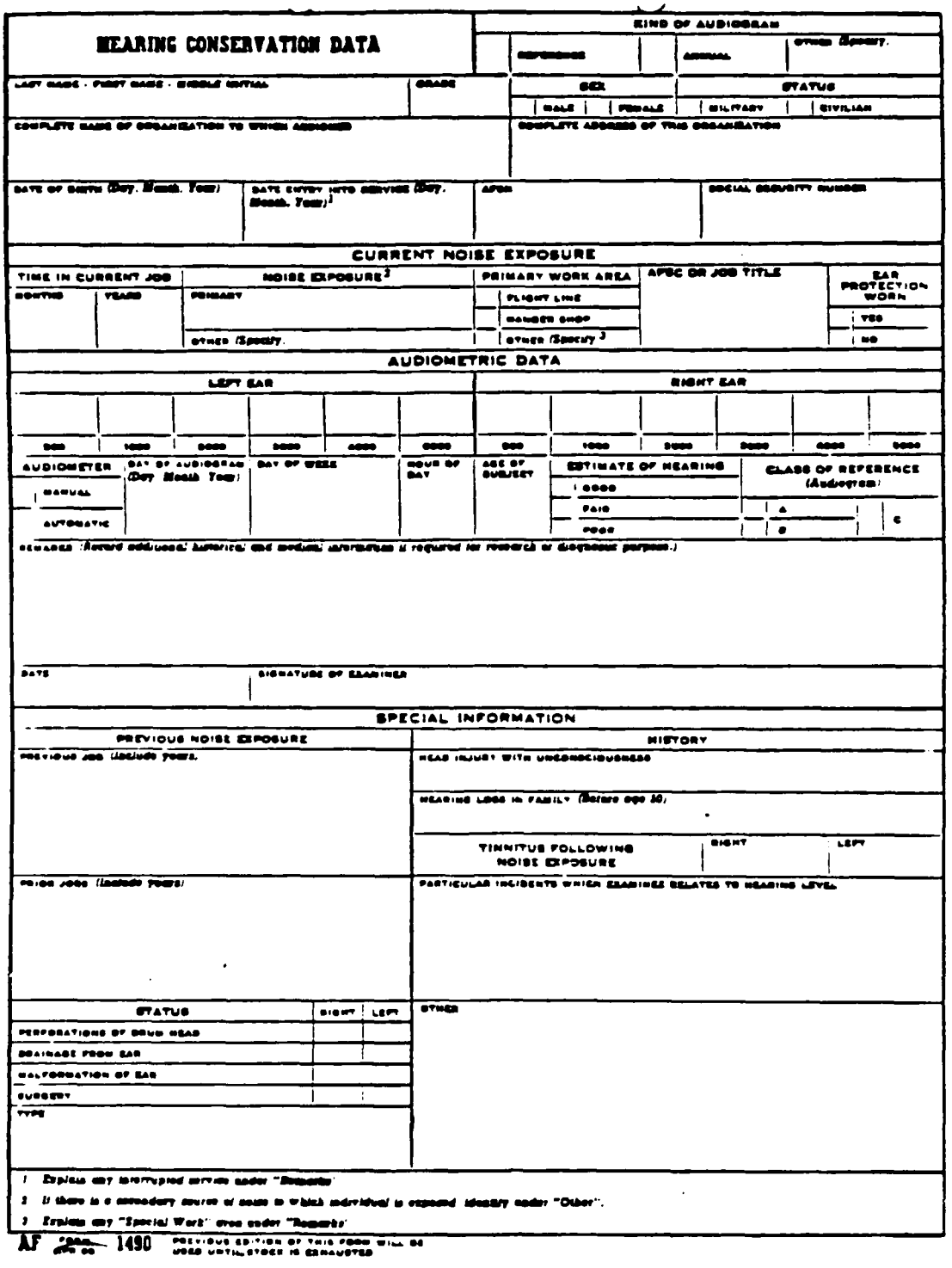
A new Air Force Occupational Safety and Health standard pertaining to audiometric testing was expected in the first quarter of 1989. The new standard requires noise exposure information to be captured along with the audiogram. A locally determined L_{eq} value will be mandated for workplace exposures. All flight personnel are included in the testing program, but no information is expected to be available on interior aircraft levels or exposures of pilots wearing flight helmets.

A microcomputer based system to capture audiometric information in conformity with the new regulation is proposed for deployment in mid-1989, and is at the time of this writing in the final approval stages. It, too, will use database technology (dBASE-IV) to enable local analysis of data. This field system is to be based on the Zenith Z-248.

The field system is modeled after the Army's HEARS (v.i.) system. The design of the Air Force system reflects lessons learned from studying difficulties with early versions of HEARS. While integrated with microprocessor based audiometers now being purchased by the Air Force, the system will also allow manual data entry. This function allows the system to be used by installations which do not yet have the newer equipment, as well as entry of information from referrals from fourteen diagnostic centers and six clinics which now contribute to the Registry.

When this microcomputer based field system is deployed it will allow direct transmittal of digitized information to the Hearing Conservation Data Registry (HCDR) computer at Brooks AFB. Information includes Academy of Otolaryngology classification of hearing loss, as well as reason, result and disposition of cases. The central HCDR system discussed here will not depend totally on automated field data collection; it will also be able to operate with manual transcription of paper records as before.

Hearing levels observed during annual examinations are compared with those recorded in a reference audiogram (generally one taken upon entry to the Air Force) to permit calculation of



HEARING CONSERVATION DATA

KIND OF AUDIOGRAM

REFERENCE | ANNUAL | OTHER (Specify)

LAST NAME - FIRST NAME - MIDDLE INITIAL | GRADE

SEX: MALE | FEMALE

STATUS: MILITARY | CIVILIAN

COMPLETE NAME OF ORGANIZATION TO WHICH ASSIGNED

COMPLETE ADDRESS OF THIS ORGANIZATION

DATE OF BIRTH (Day, Month, Year) | DATE ENTRY INTO SERVICE (Day, Month, Year)[1] | AFSC | SOCIAL SECURITY NUMBER

CURRENT NOISE EXPOSURE

TIME IN CURRENT JOB: MONTHS | YEARS

NOISE EXPOSURE[2]: PRIMARY | OTHER (Specify)

PRIMARY WORK AREA: FLIGHT LINE | MAINTENANCE SHOP | OTHER (Specify)[3]

AFSC OR JOB TITLE

EAR PROTECTION WORN: YES | NO

AUDIOMETRIC DATA

LEFT EAR

500 | 1000 | 2000 | 3000 | 4000 | 6000

RIGHT EAR

500 | 1000 | 2000 | 3000 | 4000 | 6000

AUDIOMETER: MANUAL | AUTOMATIC

DAY OF AUDIOGRAM (Day, Month, Year)

DAY OF WEEK

HOUR OF DAY

AGE OF SUBJECT

ESTIMATE OF HEARING: GOOD | FAIR | POOR

CLASS OF REFERENCE (Audiometer): A | B | C

REMARKS (Record additional numerical and medical information if required for research or diagnostic purposes.)

DATE | SIGNATURE OF EXAMINER

SPECIAL INFORMATION

PREVIOUS NOISE EXPOSURE

PREVIOUS JOBS (Include years)

PRIOR JOBS (Include years)

HISTORY

HEAD INJURY WITH UNCONSCIOUSNESS

HEARING LOSS IN FAMILY (Relation and ID)

TINNITUS FOLLOWING NOISE EXPOSURE | RIGHT | LEFT

PARTICULAR INCIDENTS WHICH EXAMINEE RELATES TO HEARING LEVEL

STATUS	RIGHT	LEFT
PERFORATIONS OF EAR DRUM		
DRAINAGE FROM EAR		
MALFORMATION OF EAR		
SURGERY		

TYPE

OTHER

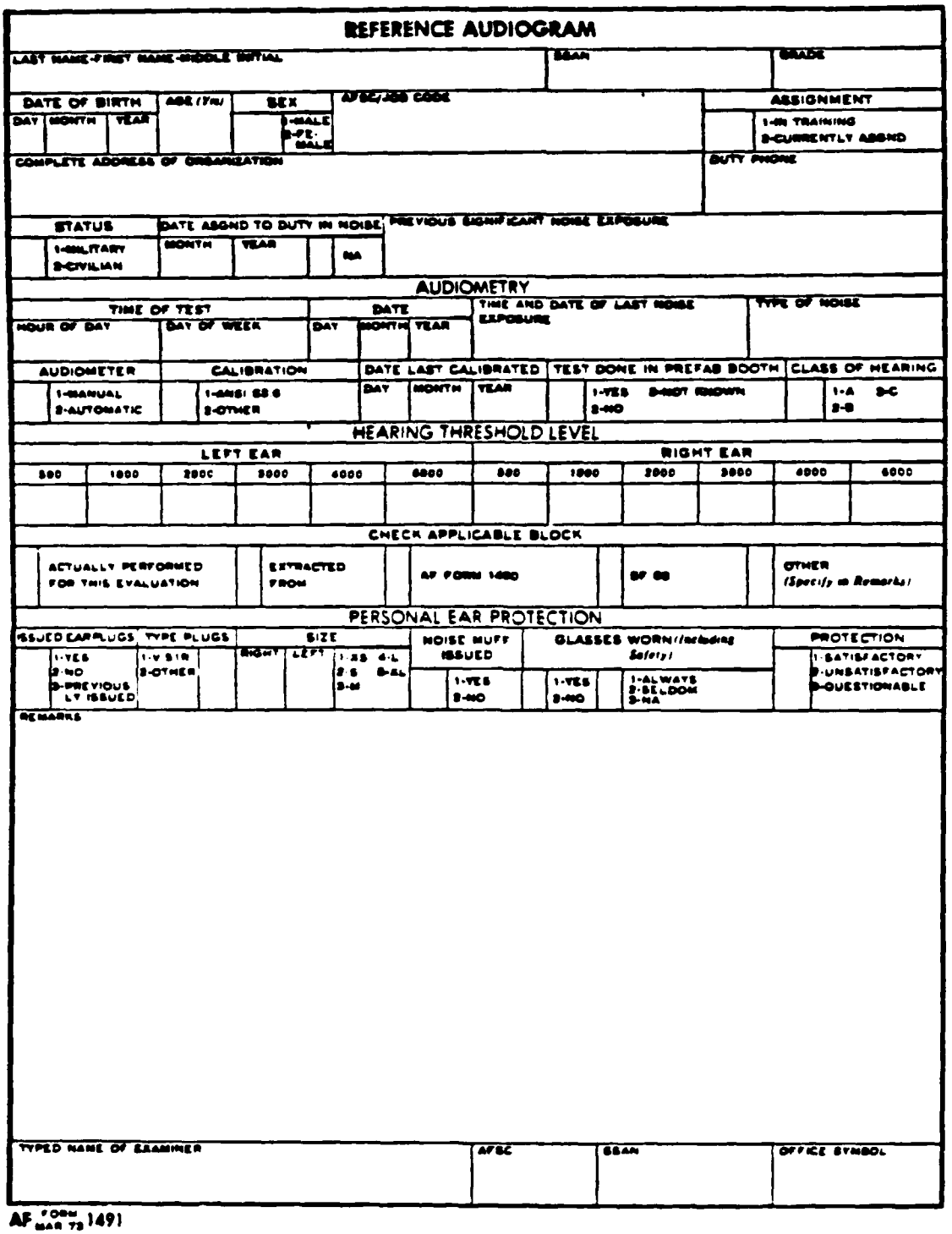
1 Explain any interrupted service under "Remarks"

2 If there is a secondary source of noise to which individual is exposed identify under "Other".

3 Explain any "Special Work" given under "Remarks"

AF FORM 1490 PREVIOUS EDITION OF THIS FORM WILL BE USED UNTIL STOCK IS EXHAUSTED

Figure 7-2: Hearing Conservation Data - DD Form 1490.



REFERENCE AUDIOGRAM

LAST NAME-FIRST NAME-MIDDLE INITIAL | SSAN | GRADE

DATE OF BIRTH (DAY, MONTH, YEAR) | AGE (Yrs) | SEX (1-MALE 2-FEMALE) | AFSC/JOB CODE | ASSIGNMENT (1-IN TRAINING 2-CURRENTLY ASSIGNED)

COMPLETE ADDRESS OF ORGANIZATION | DUTY PHONE

STATUS (1-MILITARY 2-CIVILIAN) | DATE ASSIGNED TO DUTY IN NOISE (MONTH, YEAR, NA) | PREVIOUS SIGNIFICANT NOISE EXPOSURE

AUDIOMETRY

TIME OF TEST (HOUR OF DAY, DAY OF WEEK) | DATE (DAY, MONTH, YEAR) | TIME AND DATE OF LAST NOISE EXPOSURE | TYPE OF NOISE

AUDIOMETER (1-MANUAL 2-AUTOMATIC) | CALIBRATION (1-ANSI S3.6 2-OTHER) | DATE LAST CALIBRATED (DAY, MONTH, YEAR) | TEST DONE IN PREFAB BOOTH (1-YES 2-NO 3-NOT KNOWN) | CLASS OF HEARING (1-A 2-B 3-C)

HEARING THRESHOLD LEVEL

LEFT EAR						RIGHT EAR					
500	1000	2000	3000	4000	6000	500	1000	2000	3000	4000	6000

CHECK APPLICABLE BLOCK

ACTUALLY PERFORMED FOR THIS EVALUATION | EXTRACTED FROM | AF FORM 1480 | SF 88 | OTHER (Specify in Remarks)

PERSONAL EAR PROTECTION

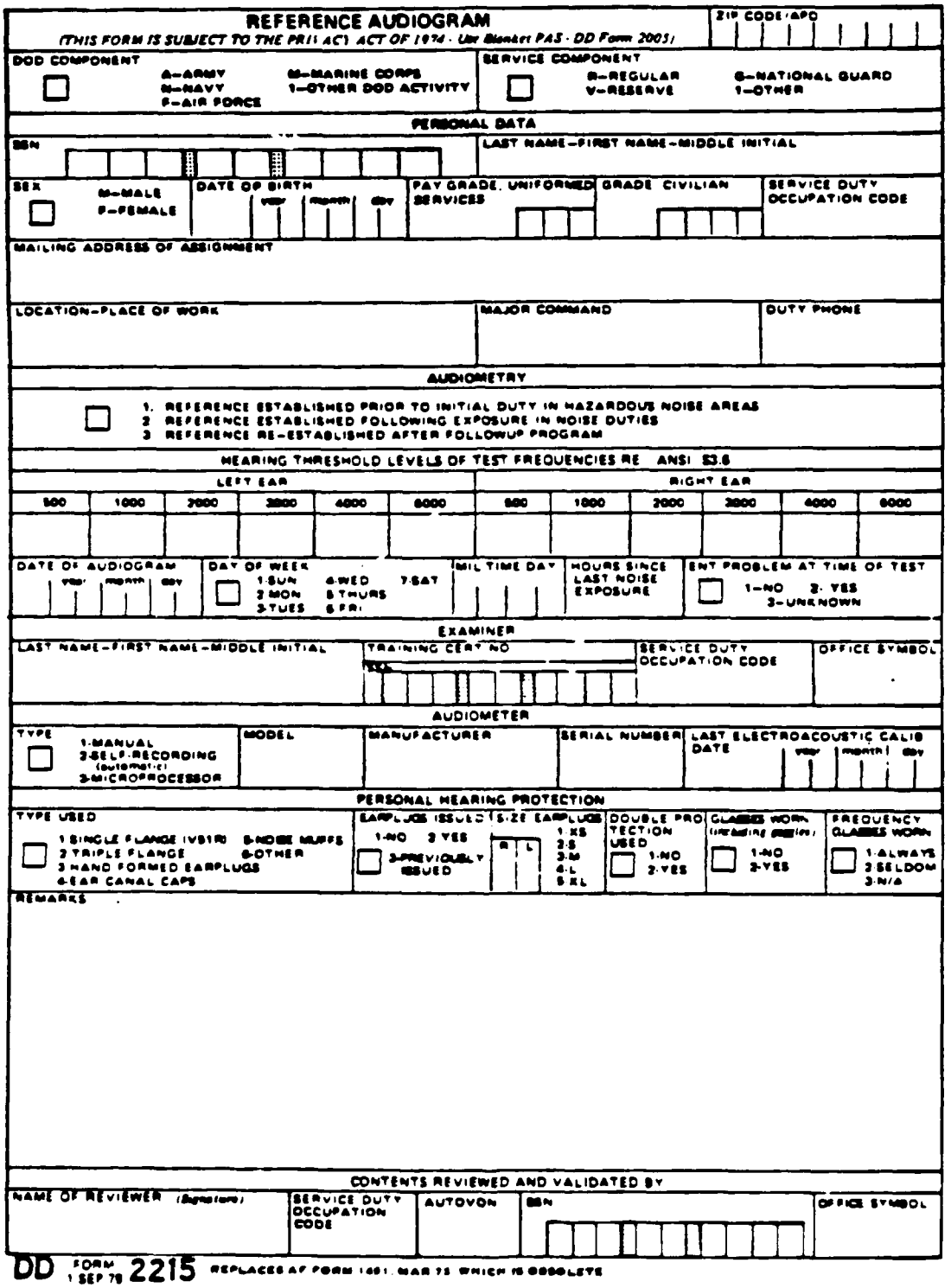
ISSUED EARPLUGS (1-YES 2-NO 3-PREVIOUSLY ISSUED) | TYPE PLUGS (1-V-51R 2-OTHER) | SIZE (RIGHT, LEFT; 1-XS 2-S 3-M 4-L 5-XL) | NOISE MUFF ISSUED (1-YES 2-NO) | GLASSES WORN (including Safety) (1-YES 2-NO) | (1-ALWAYS 2-SELDOM 3-NA) | PROTECTION (1-SATISFACTORY 2-UNSATISFACTORY 3-QUESTIONABLE)

REMARKS

TYPED NAME OF EXAMINER | AFSC | SSAN | OFFICE SYMBOL

AF FORM MAR 73 1491

Figure 7-3: Reference Audiogram - DD Form 1491.



REFERENCE AUDIOGRAM

(THIS FORM IS SUBJECT TO THE PRIVACY ACT OF 1974 - See Blanket PAS - DD Form 2005)

ZIP CODE/APO

DOD COMPONENT
A–ARMY
N–NAVY
F–AIR FORCE
M–MARINE CORPS
1–OTHER DOD ACTIVITY

SERVICE COMPONENT
R–REGULAR
V–RESERVE
G–NATIONAL GUARD
1–OTHER

PERSONAL DATA

SSN

LAST NAME–FIRST NAME–MIDDLE INITIAL

SEX
M–MALE
F–FEMALE

DATE OF BIRTH (YEAR, MONTH, DAY)

PAY GRADE, UNIFORMED SERVICES

GRADE CIVILIAN

SERVICE DUTY OCCUPATION CODE

MAILING ADDRESS OF ASSIGNMENT

LOCATION–PLACE OF WORK

MAJOR COMMAND

DUTY PHONE

AUDIOMETRY

1. REFERENCE ESTABLISHED PRIOR TO INITIAL DUTY IN HAZARDOUS NOISE AREAS
2. REFERENCE ESTABLISHED FOLLOWING EXPOSURE IN NOISE DUTIES
3. REFERENCE RE-ESTABLISHED AFTER FOLLOWUP PROGRAM

HEARING THRESHOLD LEVELS OF TEST FREQUENCIES RE ANSI S3.6

LEFT EAR						RIGHT EAR					
500	1000	2000	3000	4000	6000	500	1000	2000	3000	4000	6000

DATE OF AUDIOGRAM (YEAR, MONTH, DAY)

DAY OF WEEK
1-SUN
2-MON
3-TUES
4-WED
5-THURS
6-FRI
7-SAT

MIL TIME DAY

HOURS SINCE LAST NOISE EXPOSURE

ENT PROBLEM AT TIME OF TEST
1–NO
2–YES
3–UNKNOWN

EXAMINER

LAST NAME–FIRST NAME–MIDDLE INITIAL

TRAINING CERT NO

SERVICE DUTY OCCUPATION CODE

OFFICE SYMBOL

AUDIOMETER

TYPE
1-MANUAL
2-SELF-RECORDING (AUTOMATIC)
3-MICROPROCESSOR

MODEL

MANUFACTURER

SERIAL NUMBER

LAST ELECTROACOUSTIC CALIB DATE (YEAR, MONTH, DAY)

PERSONAL HEARING PROTECTION

TYPE USED
1 SINGLE FLANGE (VENTED)
2 TRIPLE FLANGE
3 HAND FORMED EARPLUGS
4 EAR CANAL CAPS
5 NOISE MUFFS
6 OTHER

EARPLUGS ISSUED
1-NO
2-YES
3-PREVIOUSLY ISSUED

SIZE EARPLUGS (R, L)
1 XS
2 S
3 M
4 L
5 XL

DOUBLE PROTECTION USED
1-NO
2-YES

GLASSES WORN (HEARING PROTECTION)
1-NO
2-YES

FREQUENCY GLASSES WORN
1-ALWAYS
2 SELDOM
3-N/A

REMARKS

CONTENTS REVIEWED AND VALIDATED BY

NAME OF REVIEWER (Signature)

SERVICE DUTY OCCUPATION CODE

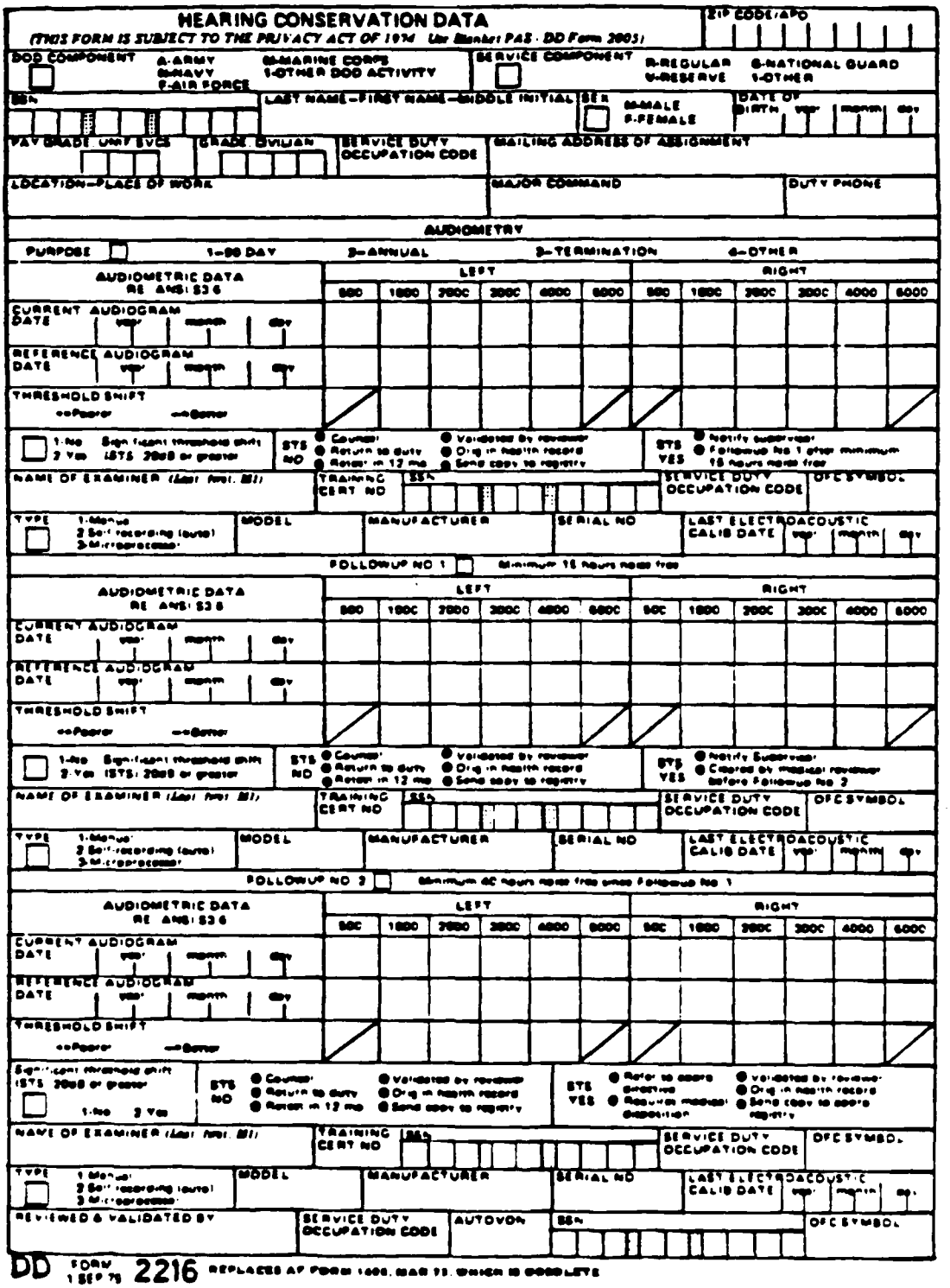
AUTOVON

SSN

OFFICE SYMBOL

DD FORM 1 SEP 79 2215 REPLACES AF FORM 1461, MAR 75 WHICH IS OBSOLETE

Figure 7-4: Reference Audiogram - DD Form 2215.



HEARING CONSERVATION DATA

(THIS FORM IS SUBJECT TO THE PRIVACY ACT OF 1974 Use Blanket PAS - DD Form 2005)

ZIP CODE/APO

DOD COMPONENT: A-ARMY; N-NAVY; P-AIR FORCE; M-MARINE CORPS; 1-OTHER DOD ACTIVITY

SERVICE COMPONENT: R-REGULAR; V-RESERVE; G-NATIONAL GUARD; 1-OTHER

SSN | LAST NAME-FIRST NAME-MIDDLE INITIAL | SEX: M-MALE F-FEMALE | DATE OF BIRTH (year, month, day)

PAY GRADE UNIFORMED SVCS | GRADE CIVILIAN | SERVICE DUTY OCCUPATION CODE | MAILING ADDRESS OF ASSIGNMENT

LOCATION-PLACE OF WORK | MAJOR COMMAND | DUTY PHONE

AUDIOMETRY

PURPOSE: 1-90 DAY; 2-ANNUAL; 3-TERMINATION; 4-OTHER

AUDIOMETRIC DATA RE ANSI S3.6	LEFT 500	1000	2000	3000	4000	6000	RIGHT 500	1000	2000	3000	4000	6000
CURRENT AUDIOGRAM DATE (year, month, day)												
REFERENCE AUDIOGRAM DATE (year, month, day)												
THRESHOLD SHIFT --Poorer --Better												

1-No 2-Yes Significant threshold shift ISTS 2000 or greater

STS NO: Counsel; Return to duty; Refer in 12 mo; Validated by reviewer; Orig in health record; Send copy to registry

STS YES: Notify Supervisor; Followup No 1 after minimum 15 hours noise free

NAME OF EXAMINER (Last, First, MI) | TRAINING CERT NO | SSN | SERVICE DUTY OCCUPATION CODE | OFC SYMBOL

TYPE: 1-Manual; 2-Self recording (auto); 3-Microprocessor | MODEL | MANUFACTURER | SERIAL NO | LAST ELECTROACOUSTIC CALIB DATE (year, month, day)

FOLLOWUP NO 1 — Minimum 15 hours noise free

AUDIOMETRIC DATA RE ANSI S3.6	LEFT 500	1000	2000	3000	4000	6000	RIGHT 500	1000	2000	3000	4000	6000
CURRENT AUDIOGRAM DATE (year, month, day)												
REFERENCE AUDIOGRAM DATE (year, month, day)												
THRESHOLD SHIFT --Poorer --Better												

1-No 2-Yes Significant threshold shift ISTS 2000 or greater

STS NO: Counsel; Return to duty; Refer in 12 mo; Validated by reviewer; Orig in health record; Send copy to registry

STS YES: Notify Supervisor; Counsel by medical reviewer before Followup No 2

NAME OF EXAMINER (Last, First, MI) | TRAINING CERT NO | SSN | SERVICE DUTY OCCUPATION CODE | OFC SYMBOL

TYPE: 1-Manual; 2-Self recording (auto); 3-Microprocessor | MODEL | MANUFACTURER | SERIAL NO | LAST ELECTROACOUSTIC CALIB DATE (year, month, day)

FOLLOWUP NO 2 — Minimum 40 hours noise free since Followup No 1

AUDIOMETRIC DATA RE ANSI S3.6	LEFT 500	1000	2000	3000	4000	6000	RIGHT 500	1000	2000	3000	4000	6000
CURRENT AUDIOGRAM DATE (year, month, day)												
REFERENCE AUDIOGRAM DATE (year, month, day)												
THRESHOLD SHIFT --Poorer --Better												

Significant threshold shift ISTS 2000 or greater 1-No 2-Yes

STS NO: Counsel; Return to duty; Refer in 12 mo; Validated by reviewer; Orig in health record; Send copy to registry

STS YES: Refer to audiologist; Validated by reviewer; Orig in health record; Request medical disposition; Send copy to registry

NAME OF EXAMINER (Last, First, MI) | TRAINING CERT NO | SSN | SERVICE DUTY OCCUPATION CODE | OFC SYMBOL

TYPE: 1-Manual; 2-Self recording (auto); 3-Microprocessor | MODEL | MANUFACTURER | SERIAL NO | LAST ELECTROACOUSTIC CALIB DATE (year, month, day)

REVIEWED & VALIDATED BY | SERVICE DUTY OCCUPATION CODE | AUTOVON | SSN | OFC SYMBOL

DD FORM 1 SEP 75 2216 REPLACES AF FORM 1408, MAR 73, WHICH IS OBSOLETE

Figure 7-5: Hearing Conservation Data - DD Form 2216.

frequency-specific shifts in hearing thresholds. Upon observation of a consequential threshold shift (currently defined as a shift greater than 15 dB), audiometric examinations are repeated at intervals ranging from 15 h to several months. Each repeat examination produces a record forwarded to Brooks Air Force Base, so that a single individual may contribute data to the Registry several times within a year.

There is no practical or cost-effective way of verifying the absolute hearing level or threshold shift information contained on reporting forms. While mental arithmetic and manual transcription are well known error sources, there is little reason to believe that such errors are likely to be systematic in the present case. Instead, the general effects of incorrect spellings of names, incorrect arithmetic, partial or illegible entries of identification or other numeric data, etc. are to reduce the overall quality of information in the database by reducing the total number of cases and by introducing error variance. Given the magnitude of the Registry, these influences are negligible for most purposes.

In the past audiograms were given to personnel upon assignment to a noisy area and at yearly intervals thereafter. In practice, there was little validation that all affected personnel did, in fact, receive their tests. There have also been problems in the past with improper training of the Unit Health Monitor, the person responsible for maintaining the roster of people requiring hearing tests.

A new HCDR procedure, with improved batch control checks, is expected to improve compliance in this area. Audiometric tests will be administered to groups of personnel (e.g., an entire machine shop), and the batch will be matched with the personnel file maintained by the Military Personnel Center at Randolph AFB. Personnel will automatically be included in the hearing conservation roster as they are assigned to new work areas. The automated registry will identify individuals who did not take their tests, as well as individuals who took the test but are not coded as requiring testing.

The official medical record is the primary source of individual health information within the military. Personnel have access to their own records, however, and items may be missing from the file through loss, misfiling or removal. The Occupational and Environmental Health Laboratory database is expected to be able to supply missing items pertaining to the hearing conservation program.

Despite the better control that the new system will provide, the success of the hearing conservation program will continue to depend on the conscientiousness and cooperation of management as well as responsible action on the part of the workers. Compliance and accuracy will remain local issues, but future records will be less likely to be lost, incomplete or contaminated.

The development of software to capture and access both current and historic information has been going on for several years. Total investment in design and implementation of the new HCDR system through December 1988 is on the order of 10 person-years.

As manual entry of records is reduced by new equipment, approximately 4 calendar years will be required to review and transfer existing records to the new system. The staff available for this work at the time of this writing includes 5 data entry personnel, 1 system analyst and 2 programmers. With 4 million records to be transferred, data entry personnel will be able to spend less than 10 s per record to process 1 million entries per year. Even at lower processing rates, any review of the data would be cursory at best.

7.2.2 Audiometric Data Collected by School of Aerospace Medicine

The School of Aerospace Medicine collects a much smaller sample of audiometric data than that routinely collected by HCDR, as part of clinical evaluations of hearing sensitivity of flight crews. Approximately 350 Air Force military personnel who have been denied flight status for medical reasons appear annually at Brooks Air Force Base for more thorough physical evaluations, including audiometric examinations. It is this information on which the recently reported research of Kent, Von Gierke, and Tolan (1986) is based.

These audiograms are not entered into the Registry, but remain part of individual medical records. The information can in principle also be linked with demographic and other information from the Military Personnel Center. Both the medical record and the Registry can, with certain constraints, be linked with information maintained by the Office of Medical Support of the Surgeon General's office.

One such source of information is the Automated Inpatient Database, which contains information about dates of entry, diagnoses, and therapies for active duty and retired military personnel (but not dependents) in DoD hospitals. Data have been collected in this database in present form since 1980. Approximately half a million inpatient records are added annually to the database, of which about 140,000 are from active duty personnel. Data on retired personnel may not be immediately available if retirees were seen in Veteran's Administration hospitals.[22]

Quality control for data in the Automated Inpatient Database is good from point of entry onward. Extensive and consistent information is available for over 8 million inpatient years in record formats providing demographic and clinical information, as well as procedural and health

[22]Although identifiers (Social Security number) allow linkage of data from one federal agency to another, privacy protections apply to these records as they do to all medical records.

care provider details. Access to the contents of the database is via special purpose software written in COBOL and FORTRAN. Manipulations of information in the database are said to require the services of a small number of programmers who are familiar with the database, since no efforts have yet been made to incorporate the files into a standard database management system.

7.2.3 Database Maintained by Cardiovascular Unit at the School of Aerospace Medicine

Another Air Force database of interest is one maintained by the Cardiovascular Unit at SAM (School of Aerospace of Medicine), in which extensive medical data are kept for military personnel who undergo thorough cardiovascular evaluations. Although this database includes information on many of the accepted risk factors for myocardial infarction and angina (except perhaps for cigarette smoking), it is not clear that data on potential confounders for hypertension such as dietary use of sodium, alcohol use, or psychological stress have been documented consistently. Recent files are said to be more complete. An even more critical problem, however, is the selective nature of the sample of aircrew personnel who actually receive these cardiovascular evaluations. Since not all individuals, including pilots, who present with overt hypertension or overt cardiovascular disease are referred to SAM, detailed health outcome data may be sparse or difficult to locate for most of a defined study group.

7.2.4 PHOENIX Database

PHOENIX, an acronym for "Promoting Healthy Occupational Environments through Information Exchange," is an occupational health surveillance database management system that the Air Force is developing specifically to comply with AFR 161-35 (April, 1982). The goal of the system is to provide the Air Force with a tool for monitoring occupational exposure and health of its uniformed and civilian employees. PHOENIX is analogous to the NOHIMS (Navy) and OHMIS (Army) database management systems (v.i.). PHOENIX is not yet operational. The system has undergone some testing at Hill Air Force Base, but as of the time of this writing development work is in abeyance.

7.3 Databases Maintained by the U.S. Navy Environmental Health Center

7.3.1 HECMIS - Hearing Conservation Management Information System

HECMIS is a system intended to assist Navy hearing conservation personnel to assess the adequacy of their programs. This system is currently in transition from a standalone database to a module of a larger occupational health record system known as NOHIMS (Navy Occupational Health Information Management System). NOHIMS is analogous to the Air Force's PHOENIX and the Army's OHMIS (v.i.) systems.

HECMIS was originally developed by the Naval Aerospace Medical Research Laboratory in Pensacola, FL. It contained approximately 875,000 audiometric records of active duty and civilian employees of the Navy and Marine Corps for the period beginning late in 1982 and ending in 1986. These records were stored on government-owned detachable disk media located at a contractor's facility in Elmsford, NY.

The data were originally accessible within a 1960s era medical information management system (MUMPS) for Digital Equipment Corporation computers, via dial-up telephone from a single terminal located at the Navy Environmental Health Center (NEHC) at Norfolk Naval Base. These 5 disk packs had been moved to Norfolk for storage at the time of this writing. The logical record layout follows the DD 2215 and 2216 format (Figures 7-4 and 7-5), but the data are not presently hosted on any computer system.

HECMIS will continue to be implemented on MUMPS operating on a collection of Digital Equipment VAX computers at various Naval installations, at least for the next five years. The information is stored at the site at which it is collected, without central control or followup. Development efforts at NARDAC (Navy Regional Data Automation Center) focus strictly on entering and retrieving information. There are no efforts presently underway to integrate audiograms into a hearing conservation program, or for tracking and evaluating followup audiograms.

It is estimated that a network that would allow the collection of audiometric information across the various VAX systems for consolidated reporting and analysis will not be available until 1992 at the earliest. While this network is a prerequisite for NEHC to upload information from individual sites to their local host computer, it is not clear how queries across the systems will be done. Nor do capabilities for checking data integrity or analyzing trends appear to have been central to the design of HECMIS and AIMS, the Audiology Information Management System component of NOHIMS (v.i.).

Linkage from HECMIS into NOHIMS is expected to be accomplished via exchange of

diskettes, although a Digital Data Network link may eventually be purchased. Plans for disposition of historical data have not been included in current development.

The individual records in HECMIS are simply reports of annual audiometric examinations of uniformed and civilian Navy personnel, as recorded on DoD forms 2215 and 2216 (Figures 7-4 and 7-5). As may be seen in Figures 7-2 and 7-3, these forms are descendants of Air Force Form 1490 and 1491 for reference and annual audiograms. HECMIS contains no noise exposure information.

The earliest audiometric records contained in HECMIS date from 1982. DD Forms 2215 and 2216 (for reference and annual audiometric examinations, respectively) were first published in 1979, and there was no mandate within the Navy for their storage until several years later. Routine receipt of DD Forms 2215 and 2216 did not begin at the Naval Environmental Health Center until 1983, in accordance with OPNAVINST 5100.23B, dated 31 August 1983. Many of the early records contained illegible or incomplete information, such as missing occupational specialty codes. In-house studies of the quality and reliability of audiometric data in HECMIS have also revealed some difficulties of the sort noted earlier by Gasaway and Sutherland (1976).

The demographic characteristics of the population that contributes these audiograms to HECMIS are not fully specifiable. It is the responsibility of each naval command to determine which personnel require medical surveillance, including audiometric examinations. Navy directives provide guidance as to which personnel should be considered at risk, but HECMIS clearly contains audiometric information for only a portion of the work force exposed to occupational noise sources. It is possible if not likely that only some of the personnel performing similar duties under similar noise exposure conditions in different commands receive annual audiometric examinations.

Even though there are no consistent Navy-wide procedures requiring audiometric examinations for personnel in each of the Navy's four-digit occupational specialty codes ("NEC" codes for Navy Enlisted Classifications and "NOBC" codes for Navy Officer Billet Classifications), the total number of audiograms recorded is enormous. Between a half and two thirds of active duty uniformed Navy personnel receive annual audiograms, while a third to a half of Navy civilian employees receive annual audiograms. Reports of audiometric examinations are presently accumulating at the Navy Environmental Center at the rate of about half a million per year. About half of the audiograms currently stored are reference audiograms on Form 2215.

Air Force and Navy policy concerning interpretation of hearing loss and followup audiometry differ in several details. It is rare for an individual to contribute more than one audiogram per year to HECMIS, since there are no requirements for reporting results of followup (e.g., 90 day) audiometric examinations to the Navy Environmental Center. Navy policy also

considers Noise Induced Hearing Loss of 15 dB to be significant, whereas a 20 dB criterion for significance is listed on Form 2216.

The population represented by audiograms in HECMIS is likely to differ from the U.S. population as a whole, and also from the population exposed to aircraft noise near MOAs and MTRs. It is predominantly a population of young adult white males. As a whole, only 10% of naval personnel are female, 11% are black, and 1% are over 43 years of age. White males between 20 and 30 years of age contribute most of the of the data to the HECMIS database. Career paths of naval personnel often remove civilian employees from exposure to heavy industrial noise after 5 to 10 years, while career paths of enlisted military personnel do not often last more than a decade.

7.3.2 NOHIMS - Navy Occupational Health Information Management System

According to Helmkamp and Seidman (1988),

> "The Navy's Occupational Health Information Management System (NOHIMS), developed at the Naval Health Research Center in San Diego, California, is an information system designed to coordinate the components of the Navy's Occupational Safety and Health (NAVOSH) Program to meet the requirements of the OSHA Act of 1970."

NOHIMS is currently in limited distribution, and will not be ready for Navy-wide use for several more years. The Navy intends to incorporate audiometric information in HECMIS (v.s.) into NOHIMS within the next few years. The acronym for the project to integrate HECMIS data into NOHIMS is AIMS (Audiology Information Management System). While some other parts of NOHIMS can track required checkups, audiometric followups cannot presently be tracked.

The emphasis in NOHIMS is on civilian rather than military personnel, since Workman's Compensation claims for hearing damage, which currently cost the Navy about $10 million per year, are an important aspect of the motivation for developing this software. It will be another year before a decision is made about extending coverage to military personnel.

7.3.3 SAMS - Shipboard Automated Medical Information System

SAMS is a medical record keeping system designed for operation on a Zenith Z-248 personal computer. Deployment of SAMS is just beginning. SAMS will incorporate basic audiometric information about the crew of ships, many of whom work in hazardous noise exposure conditions aboard ships built before 1975. Although SAMS will at first contain only prospective data, it may eventually provide a source of information for retrospective studies.

7.4 Databases Maintained by the U.S. Army Environmental Hygiene Agency

7.4.1 HEARS - Hearing Evaluation Automated Registry System

HEARS was developed by the Environmental Hygiene Agency at Edgewood Arsenal in Aberdeen, MD. The audiometric data-capture features of HEARS are the most advanced of those of any of the armed services. Fully automated, microprocessor based administration of pure tone audiometry to multiple personnel and all-electronic record production are currently supported in the field.

Considerable backlog (nearly 1,000 diskettes produced by HEARS, containing tens of thousands of audiograms) of data entry into an Amdahl mainframe computer at Ft. Detrick existed when the field system became operational. This backlog has now been worked off. The database contains approximately 750,000 records (both DD 2215 and 2216). In principle, all uniformed Army personnel should receive annual audiograms. In practice, records of only about 280,000 personnel per year, many of these reference audiograms, are currently being taken in HEARS.

The field system is implemented on Intel 8088 CPU technology Wang PCs with 10 Mb hard disks. For some large installations 20 Mb disks are required. The software consists of database management software (MicroFOCUS which is to be migrated to dBXL) and the control programs for the audiometric equipment. Since the interface to the audiometer is device specific a more general interface is scheduled for development to permit use of other vendors' equipment without extensive software revisions. The current system represents approximately 6 person-years of development effort.

The central site supplies field sites with basic information, keyed to Social Security numbers, for personnel at the base. This information includes major command, name, address, gender, birthdate, and a reference audiogram (DD 2215) when available. The database contains a UIC code, but the assignment process and significance for noise exposure are not known.

On entry of a DD 2216 form, the software compares the 2215 and 2216 and alerts the technician if something requires further attention. After audiograms are taken, diskettes with up-to-date information are sent back to the central site. These data are uploaded into the mainframe on a quarterly basis, with plans to update monthly. The Army has no machine readable information more than 5 years old.

The system will feed data into CHCS, the Composite Health Care System in either ASCII or EBCDIC format. CHCS is an Army medical records system to which HEARS will contribute information. HEARS will not be accessible for nonclinical (i.e., research) purposes.

Although the Army, like the Navy, leaves the decision about uniformed personnel and civilian employees requiring audiograms to local commands, there are, nevertheless, strong self-selection biases in the database. As seen in Figure 7-6, the database contains information primarily about the hearing of young personnel with relatively short time in service (associated with the lower pay grades.) However, as seen in Figure 7-7, it is the older personnel, associated with higher pay grades, who have the greater prevalence of hearing damage.

In other words, precisely those personnel who have the greatest cumulative exposures to hazardous noise, and hence the greatest potential hearing damage and extra-auditory effects of exposure, are the ones who are most under-represented in the database. Older personnel have the strongest incentives--not to mention the greatest resources--for avoiding annual audiometric examinations which might disqualify them for desirable duty or might even result in their separation from the service. This strong self-selection bias creates severe sampling problems for research conducted on HEARS records.

The system is implemented and managed by the Directorate of Information Management, U. S. Army Information Systems Command. Current software development and hardware maintenance budget is $450,000 per year. The total cost for HEARS over the next 10 years is estimated at $10,000,000, much of it for hardware.

7.4.2 OHMIS - Occupational Health Management Information System

OHMIS was conceived in 1973 as part of the Army's response to Executive Order 12196, which directed that the armed services comply with the Occupational Safety and Health Act of 1970 (OSHA), Public Law 91-596. It was not, however, until 1983 that funds were allocated for system development work. A presidential directive was also issued in the same year to reduce Workman's Compensation claims by 3% per year. A further spur to rapid development of OHMIS is that the Department of Labor has begun charging back expenses for occupationally related illnesses and injuries of employees of the armed services to the individual services. The Army is beginning to redistribute these charges to local commands to provide even greater incentives for commanders to reinforce compliance with safe work practices such as wearing of hearing protection.

OHMIS consists of three major modules: HEARS, a Medical Information Module (MIM), and a Health Hazard Information Module (HHIM). The three modules are not expected to be integrated into a fully operational system for several more years, but portions of the system are already deployed. The first phase of MIM, for example, was operational at the time of this writing on 145 WANG personal computers at Army installations in North America, Europe, and Asia. Two other development phases for MIM are currently planned, however, before MIM is complete. Limited deployment of HHIM began at the end of FY 1988, but routine utilization of the system awaits further software development.

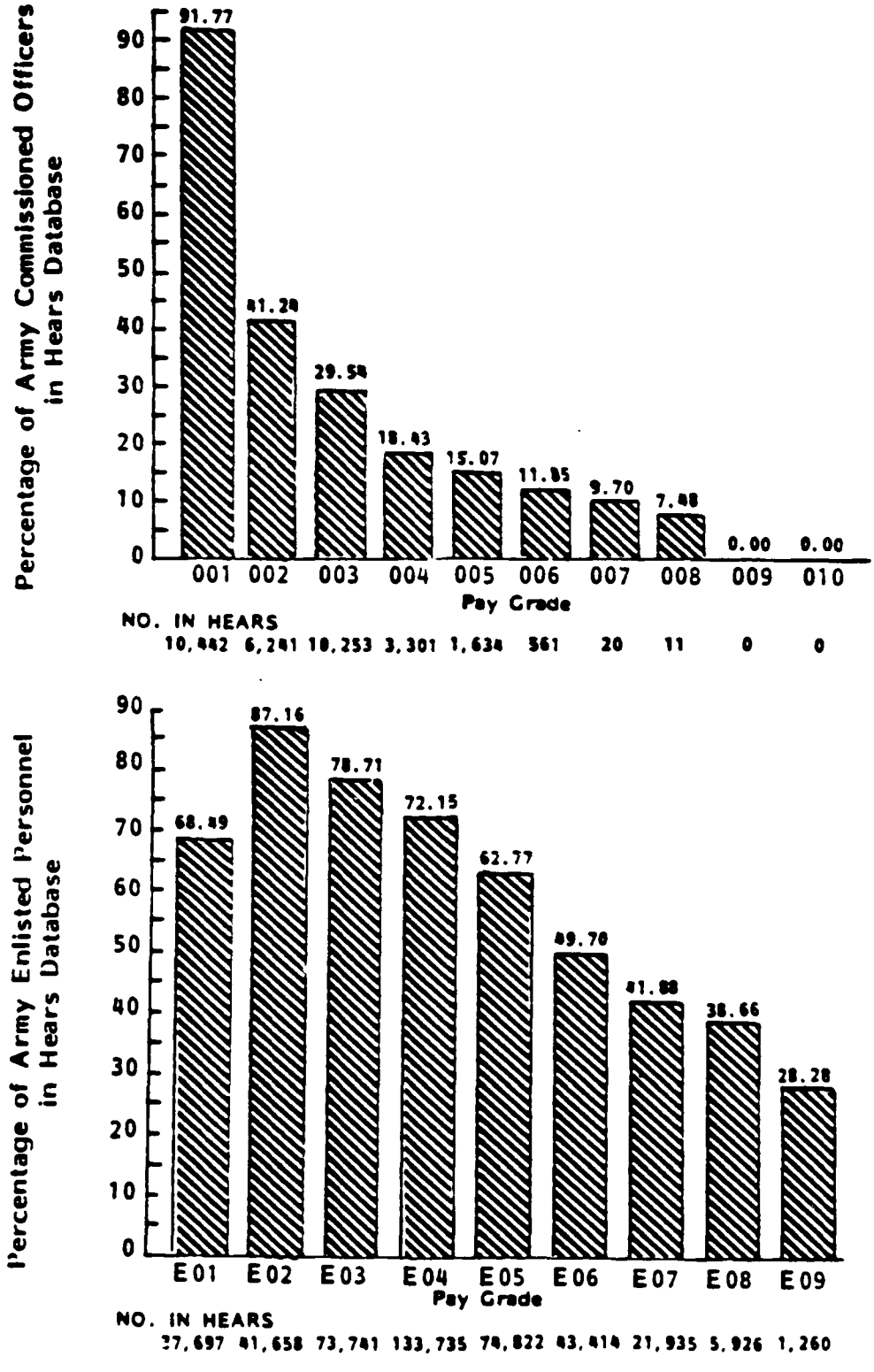


Figure 7-6: Percentages of Commissioned Officers and Enlisted Personnel by Pay Grade in HEARS.

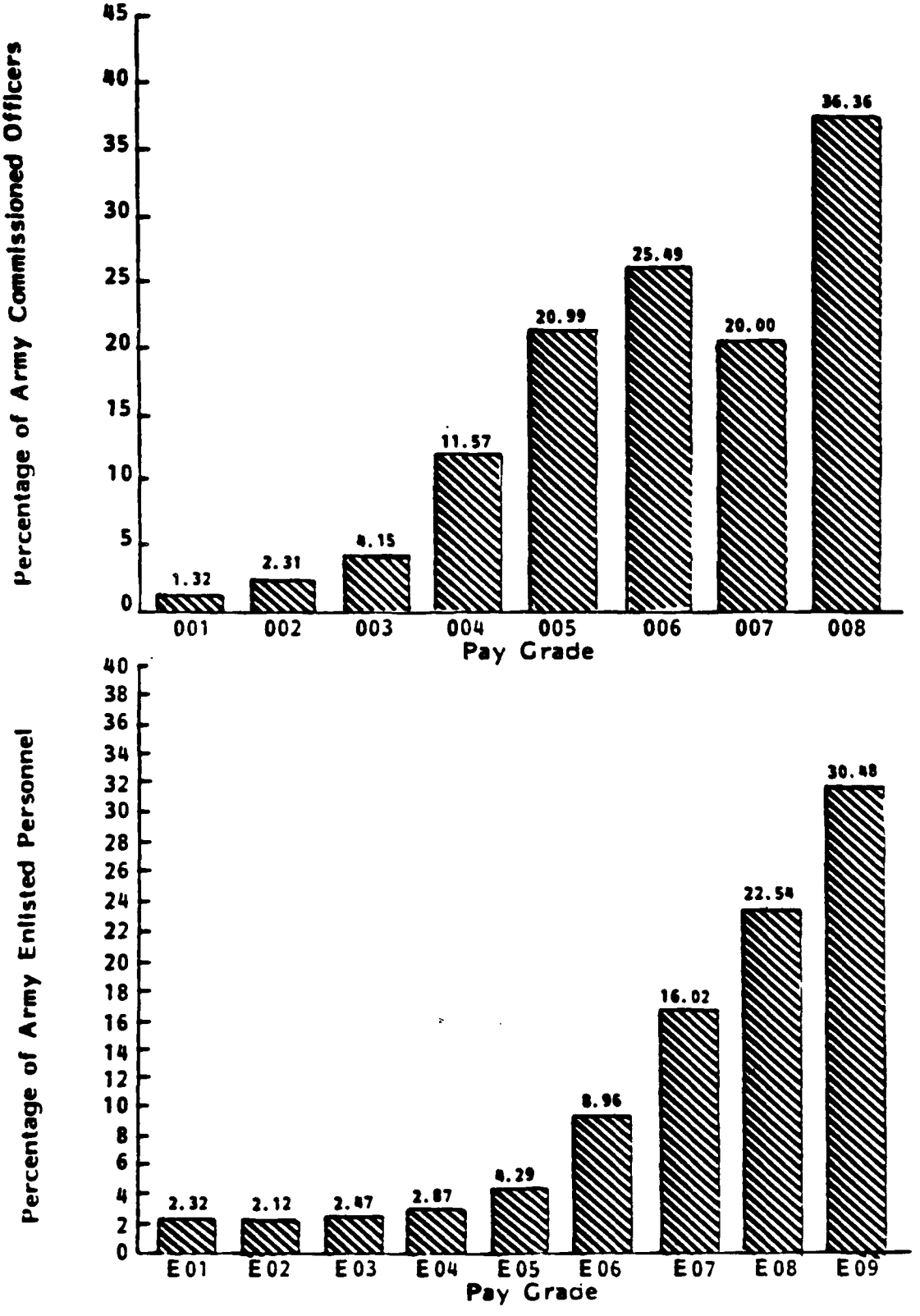


Figure 7-7: Prevalence of Significant Hearing Loss in Commissioned and Enlisted Personnel by Pay Grade.

The medical and health hazard modules deal primarily with the records of personnel exposed to various occupational risks in heavy industrial activities. Many (if not most) of these personnel are civilian employees; many (if not most) of these are young adult males with few years of job-related exposure to noise.

Since OHMIS was designed for purposes other than epidemiologic research, it contains few provisions for tracking individual occupational and exposure histories over time, or for linking such information with health outcomes.

7.4.3 Other Databases

The Army also maintains a number of other health-related databases through which individual records could conceivably be linked via Social Security number to audiometric data in HEARS. These databases include an IPDS (Individual Patient Data System) managed by Patient Administration Systems and Biostatistics Activity at Ft. Sam Houston in San Antonio, TX, and a Hospital Discharge database that includes ICD (International Classification of Disease) discharge diagnoses.

7.5 Databases Other Than DoD

7.5.1 Other Government Databases

Non-DoD agencies of the U.S. government also maintain numerous health-related databases. Perhaps the most notable of these agencies are the Centers for Disease Control in Atlanta, GA, and the National Center for Health Statistics, an agency of the Office of Health Research, Statistics and Technology of the U.S. Public Health Service (U.S. Department of Health and Human Services). The latter agency maintains over a dozen national vital statistics databases, including natality, morbidity, and mortality, as well as information on health resource utilization, such as a National Hospital Discharge Survey, a National Ambulatory Medical Care Survey, and a National Nursing Home Survey. For reasons of confidentiality, no information is distributed by these agencies that could conceivably be traced to individuals. Although tapes are available for epidemiologic analysis of individual patient records without identifiers, there is no noise exposure data and no way of linking other data sets to these files. Also, because of the sampling frame, inferences cannot be drawn for given geographical areas. Thus, none of the databases maintained by CDC or NCHS is useful for present purposes.

7.5.2 Private Databases

Industrial databases offer few opportunities for applications in federally-sponsored health research. The major studies on hearing loss industry were done more than 20 years ago (e.g., Baughn, 1966), before the advent of large scale organized electronic databases. Although more current health records exist, their utility for present purposes is limited for several reasons.

- Data collected by private companies are likely to be considered proprietary, with strong protections for both employee and commercial rights.
- There are strong economic incentives for industry to under report hearing damage. For example, OSHA has fined at least one major automobile manufacturer for failing to record adequately and report work-related injuries and illnesses (Noise Regulation Report, 1989).
- Databases would most likely be available only for retrospective study, if at all.
- Exposure would be limited to occupational noise, not directly applicable to the kind of noise produced by military aircraft operations.

7.6 Summary of Utility of Audiometric Databases for Air Force Purposes

The preceding sections have discussed the suitability of databases of audiometric information in the context of their suitability for use in epidemiologic studies of extra-auditory health consequences of noise exposure. The nature of the information contained in these databases would impose a number of constraints on the sorts of hypotheses about nonauditory health that could be explored in epidemiologic studies. A large number of arguable assumptions would have to be made about the use of hearing damage as a surrogate for noise exposure, about the similarity of people represented in the databases to residential populations, about the similarity of occupational and residential noise exposure, among other issues.

More specifically, some assumptions implicit in drawing inferences about adverse effects of noise exposure on health from an epidemiologic study based on these databases are the following:

1. Hearing threshold shifts as measured by pure tone audiometry are directly attributable to occupational noise exposure and may therefore serve as reliable indices of personal exposure;
2. Noise-induced morbidity and/or mortality have relatively short latency periods (on the order of 5 years or less) and produce acute rather than chronic effects;
3. Such adverse health consequences may be observed in a predominantly young and healthy population.

In the aggregate, these assumptions pose serious logical obstacles to the conduct of meaningful epidemiologic research. For example, unless it is possible to obtain a credible estimate of levels of exposure to noise other than hearing threshold shifts in both a study and comparison group, an attempt at a large cohort analysis or a large case-control study is not warranted. Based on current knowledge, hearing loss and permanent threshold shifts in hearing levels should be treated as indicators of a high risk group rather than a good surrogate exposure measure for noise exposure. The risk factors for NIHL are not clearly delineated and the relationship between hearing loss and cardiovascular disease/health outcomes is far from clear. Inferences from studies using hearing loss as a surrogate for noise exposure must be considered tentative until studies are conducted to determine that hearing loss is indeed a good surrogate measure.

Accurate exposure characterization is critical to reducing misclassification bias in epidemiologic studies. Misclassification of exposure status of individuals inevitably reduces the power of an epidemiologic study to detect associations--most particularly weak associations--between specific exposures and health outcomes. The ability to develop individual rather than group exposures also strongly influences the accuracy of identifying high risk groups and avoiding dilution effects. In general any dilution effects translate into a decreased ability to detect a true etiologic association between noise and a health outcome, if one indeed exists.

There is no practical means of retrospectively determining detailed noise exposure of individuals whose audiograms are included in the different services' databases. Individual occupational codes are not reliable guides to noise exposure, since it is not uncommon for uniformed personnel and civilian employees of the armed services to work at jobs other than those indicated by their specialty codes.

While it is possible to make reasonable estimates of Air Force flight crew members' cumulative noise exposure using hours flown, aircraft type, etc., the Personnel Database containing this information is kept for officers only and the data collected are not consistent over time. No such distinction by flight status is made in the Navy's databases, and there is reason to believe that Army helicopter pilots may be poorly represented in audiometric databases (Ohlin, 1988).

Code definitions and record layouts in the Air Force databases would require extensive manipulation of some 30 generic types of files to construct these individual noise exposures. There is no guarantee, however, that complete data would be available for a defined group of individuals even if the effort were undertaken. It is also questionable whether a measure of exposure better than the individual's Air Force Specialty Code could ultimately be derived since exposure to noise in the cockpit depends on numerous factors (Kent, et al., 1986).

DoD personnel who actually do work under conditions that expose them to a consequential

risk of hearing damage are provided with hearing conservation equipment (earmuffs, earplugs, acoustic enclosures, etc.) that is currently intended to keep their personal 8-h equivalent level noise dose under 85 dBA. Thus, there is no certain way to associate personal noise dose with audiometric data. One can at best assume that if individuals exhibit large shifts in hearing threshold levels, their threshold shifts are attributable at least in part to occupational noise exposure.

Thus, the nature of the audiometric databases does not offer a clear design strategy because of the difficulty in using any one of them (1) to identify complete cohorts for defined periods of workers who were and were not exposed to aircraft noise and for whom complete health information is obtainable; or (2) to identify representative groups of "cases" (of hypertension/cardiovascular disease) and comparable controls for whom complete exposure information is available. Unless the population under study is clearly defined and exclusions documented, any conclusions would be subject to criticisms of biases of the researcher in selecting the sampling frame.

Furthermore, there are also practical constraints to the design of studies that rely on information in DoD audiometric databases for Air Force purposes, including considerable costs of accessing the information and the fact that the databases are still under development. Some of the practical obstacles to use of the armed services' audiometric databases are likely to be ameliorated within a period of 3 to 5 years as automated data entry and better quality control procedures are more fully deployed, and as experience is gained in their operation. Barring unexpected changes in the design of these databases, however, tracking individuals identifiable in audiometric databases over time through multiple unrelated sets of health records will always be difficult, due in large part to privacy considerations.

7.7 Feasibility and Cost of Extracting Information from Audiometric Databases

The investigations described above indicate that there is no consistent, computer accessible audiometric information available in DoD databases more than about 5 years old. Much of the earlier data is inaccessible for practical purposes: either destroyed, unavailable in computer-compatible format, or of dubious quality due to unreliability of audiometric measurement.

The quality of the audiometric measurements in DoD databases is improving as Air Force and Army audiometric data capture systems become increasingly automated, with less margin for error, better batch controls and more comprehensive data checking at the time of capture. Several years are probably needed before the Navy achieves a corresponding level of sophistication in audiometric data capture.

None of the presently available databases contains information about workplace noise exposure. Exposure can be crudely estimated from job codes, but the correlation between codes and individual dosage is too tenuous for research purposes. Only the USAF HCDR system will attempt to include a direct measurement of workplace (only) noise exposure. HCDR is still under development, and is not expected to be operational for another 5 years.

All of the computerized databases investigated have Social Security number keys. Audiometric information from individuals in these databases can therefore be matched, at least in principle, with information in other health-related databases which also contain Social Security numbers.

On the assumption that privacy constraints and technical considerations involved in linking databases and gaining access to medical records can be resolved, it is possible to estimate the total amount of information that would be available for a comprehensive epidemiologic study. Within the next few years, the three services will have accumulated an estimated 10 to 15 million audiograms, representing roughly 25 GBytes (gigabytes) of information. A similar amount of information (if not more) is probably contained in the medical records with which this information must be matched.

Perhaps the simplest way to develop rough estimates of the costs of dealing with quantities of information on the order of 50 GBytes can be made by assuming that manipulation of all of this information will require a dedicated mainframe computer[23]. Although these estimates do not directly consider some reduction in bulk of information possible through elaborate sampling plans, they are still instructive.

The analysis required to construct the specification of a workable audiometric database is estimated by both Army and Air Force personnel on the basis of actual experience as approximately three person-years, with an equal amount of programming and implementation effort (Patrick, 1988; Schlieffer, 1988). One can safely assume that once the data elements to be incorporated in the databases needed for an epidemiologic study have been determined, greater effort will be required than for the audiometric database. Rather than merely capturing data from a known single source, the research database must be constructed from samples drawn from many different sources. Each of these sources has its own peculiarities, and each requires its own detailed analysis of information available, controls and checks, reliability and credibility, etc.

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[23]None of the computer systems currently used for managing audiometric databases has sufficient excess capacity to perform analyses of the kind contemplated, even for the information normally hosted. Therefore each computer installation would have to be upgraded if epidemiologic analyses were to be locally conducted. Since such upgrades come in finite increments, the sum of all additional capacity required by Army, Navy, and Air Force computers to perform database matches would almost certainly exceed the cost of purchasing a machine dedicated to such a project.

The Air Force and Army experience can serve as a guide in this respect. A conservative estimate of the effort required to link the 3 major audiometric databases with a half-dozen major medical information systems is 40 person-years. A staff of 3 system analysts, 2 statisticians, 6 programmers, 2 clerks and a project manager would probably require 2 to 3 calendar years to construct such a comprehensive database. This estimate considers only the technical difficulties of linking the databases and constructing samples of the intersection of entries that occur in both medical records and audiometric databases.

As in the case of audiometric databases, two issues must be considered: (1) the technical issue of linking the information, and (2) the statistical issue of data validity. The technical issues are quite complex since the databases were developed for purposes other then epidemiologic study. To link these databases requires common database systems and common, unique keys. Since this is not presently the case, it would be a formidable task to convert data into a common format. Because all systems are fully operational production systems, every newly-developed tool must be subjected to a full quality assurance cycle so as not to endanger the production environment. A general rule in the data processing industry is that building such conversion tools requires 2-3 times the effort of developing the systems themselves.

Connecting each of the (Army, Air Force, and Navy) audiometric databases to about 6 medical databases was considered to be more complex by an order of magnitude than construction of the initial database. It would be more efficient to have a single agency perform this work rather than having each organization work on its own. Considerable work, including development of software tools, will be required to test the validity of the data. Since the databases were constructed for other purposes, there is no assurance that the information needed is available nor that, if it is available, it is of acceptable quality. This analysis represents a significant level of effort over and above the task of linking the information.

While no provision was made to account for legal challenges posed by privacy laws which specifically prohibit transfer of medical records, management time to coordinate efforts among agencies was considered in developing staffing estimates.

Rather than considering the task as one of linking 3 audiometric databases to 6 medical databases, representing 18 connections, it is more efficient to view the task as one of connecting 6 medical databases to a combined audiometric database. This would result in an 8-step aggregation, each step estimated by personnel interviewed to be of a level of complexity comparable to projects thus far undertaken. Since each of these steps takes 2-3 years of analysis and equal amounts time for programming, consensus among military data processing professionals was that the required effort would be on the order of 40 person-years.

Detailed estimates of the total costs for assembling the resources needed to support an epidemiologic study of the sort under consideration cannot be developed without consideration

of many ancillary matters. Order of magnitude costs can be estimated however, as follows. The cost of a computer system capable of efficiently manipulating the quantities of information involved is on the order of $10 million. Approximately 20 person years of support would also be required for the computer system and host organization support. Using $75,000 as an average cost per person year for the clerical through professional personnel required, the cost of procuring 60 person-years of labor (40 for database design, 20 for computer support) would be on the order of $4.5 million. Thus, the order of magnitude of the total costs would be $15 million.

The computer support costs are derived from the need for a very large disk farm to store and manipulate the data. The size of this effort is such that it cannot be accommodated in excess capacity of current machines. Maintaining a large mainframe computer (operations, systems programming, hardware maintenance, and telecommunications) was estimated to require a 7-person crew for the 3-year project.

This estimate does not include any analysis effort required after construction of the database for the actual conduct of an epidemiologic study. (As noted earlier, there are grounds for reservations about the utility of the information contained in the databases for present purposes.) The estimate of the duration of the effort is based on the assumption that all time is spent resolving technical problems without delays caused by administrative or other nontechnical difficulties.

This estimate also does not include the work required to clean up any databases. The extent to which clean up may be necessary would become apparent only after exploratory analyses and consistency checks had been performed to assess the quality of the data. For example, the extent to which information about individuals in legally accessible health databases intersects information about individuals in the audiometric databases is unknown. The population of personnel for whom information is available (predominantly young personnel in good general health) in audiometric databases is likely to be quite different from many of the health-related databases (e.g., relatively older personnel sufficiently ill to be hospitalized).

The Army database is currently complete, in the sense that 5 years of audiometric information is loaded into a mainframe computer. Over the next 5 years the Air Force's audiometric database will be complete as well, and will provide at least preliminary information about occupational noise exposure. The Navy's audiometric data will continue to be highly fragmented for the next 5 years. The technology to link the Navy's computers will not be in place for at least 3 years. Some 20 Navy sites have to be equipped with appropriate computers, and a national telecommunications network has to be installed before the backbone is in place. Little detailed consideration has been given thus far to logically linking the Navy's databases.

Some of the effort required to consolidate audiometric measurements and increase their

accessibility for research purposes will take place without outside intervention within the next several years. This consolidation will reduce the effort required for database analyses by perhaps five person-years, since most of the effort remains in correlating and extracting data from the health databases. New health databases under development (PHOENIX, OHMIS, NOHIMS) will ultimately be able to capture audiometric information. The difficulties that the three services have experienced in developing this capability are indicative of the complexity of the task.

It should also be recognized that the $15 million order-of-magnitude estimate would not suffice to fund development of a production system that could be used to conduct ongoing analyses over time. In the next 5 years (or perhaps longer if current development schedules prove to be more optimistic than can be realized in practice) DoD's audiometric databases will be accumulating more of the type of information needed on an on-going basis. A separate research project of the sort under discussion could well detract from the orderly development of PHOENIX and similar systems, since the support resources from the organizations maintaining or developing the databases represents roughly a third of the total effort.

Thus, although epidemiologic studies based on DoD audiometric and health databases are currently premature, it is likely that in about 5 years the types of analyses contemplated could be conducted without hindering ongoing development work. It is also likely that if the new health database systems are appropriately designed, the effort required to extract the data would be on the order of several person-years. The only staff required would be a biostatistician and/or epidemiologist and a programmer/analyst who could use then-available database tools for constructing defensible samples and matching records among databases.

8. Evaluating Designs for Epidemiologic Study of Potential Health Consequences of Aircraft Noise Exposure

The preceding chapters have described the advantages and disadvantages of several types of epidemiologic studies of the health consequences of aircraft noise exposure. Most of the study types considered have one or more serious flaws that preclude an unconditional recommendation in favor of immediate implementation. Given enough time and other resources, however, it is also apparent that some types of epidemiologic study could be undertaken to accomplish limited goals. However, since research resources are themselves limited and the needs of Air Force environmental planners are pressing, a rationale for prioritizing study designs is needed. This chapter develops such a rationale, and then applies it to the study designs described in Chapters 5 to 7. Volume I contains a strategic plan for implementing the rationale developed in this chapter.

8.1 Prioritizing Study Designs

The utility of potential epidemiologic studies may be ranked by the degree to which they satisfy a primary goal of the NSBIT program: creation of information and tools that Air Force environmental planners can defensibly use to improve their assessments of the effects of aircraft noise on health, especially near MOAs and MTRs. This goal in turn implies (1) that the circumstances of noise exposure of greatest interest are those characteristic of residential exposure to aircraft operations near MOAs and on MTRs (sporadic and intermittent exposure to relatively small numbers of low altitude, high speed flyovers and sonic booms); (2) that the population of greatest interest is a low density rural residential population; and (3) that studies must be capable of yielding quantitative information about causal relationships.

8.2 Epidemiologic Criteria for Evaluating Desirability of Study Designs

8.2.1 Study Designs of Greatest Utility

Study designs most likely to produce information useful to environmental planners have the following characteristics:

- they focus on the individual as the unit of analysis and include statistical controls for confounding variables;
- they permit causal inferences about the influences of aircraft noise exposure on specific cardiovascular health outcomes;
- they involve direct physical measurement of individual noise exposure; or in the absence of individual exposure, well-documented place estimates of exposure for individuals;
- they are of a duration comparable to or longer than the latent and induction periods of diseases of interest;
- they support development of quantitative dosage-effect relationships;
- they investigate effects of the types of residential aircraft noise exposure that actually occur near MOAs and MTRs;
- they examine effects that occur in individuals residing in low population density rural areas; and
- they are powerful enough in terms of sample size and absence of bias to be highly likely to find a consequential effect if one exists, and to have very low probabilities of producing spurious findings.

8.2.2 Study Designs of Lesser Utility

Study designs less likely to produce information useful to environmental planners have the following characteristics:

- they focus on effects of occupational or other non-aircraft exposure;
- they do not permit causal inferences about the influences of such noise exposure on specified health outcomes;
- they do not support development of quantitative dosage-effect relationships;
- they do not directly measure noise exposure, but rely instead on estimate, assumption, or measurement of surrogate variables.
- they examine effects that occur in groups rather than individuals residing in places other than low population density rural areas, or in occupational rather than in residential settings; and
- they have too little power to uncover a consequential effect, even if it exists.

8.2.3 NonTechnical Criteria for Judging Utility of Study Designs to Air Force

Nontechnical factors must also be considered in reaching decisions about implementation of any epidemiologic studies of health effects of aircraft noise exposure. Such factors might include the time and other resources required for the conduct of a study, its likelihood of uncovering a consequential effect, its potential controversiality, and so forth. Note is therefore taken in Tables 8-1 through 8-3 of aspects of studies other than strict technical feasibility.

8.3 Application of Criteria to Potential Studies

The criteria noted in Section 8.2 may be used to rank the various study designs described in the previous chapters of this report. This ranking assigns the highest priority to study designs that can yield definitive information about the health consequences of the types of aircraft noise exposure experienced near MOAs and MTRs on the type of populations residing near them. It assigns the lowest priority to study designs that cannot yield definitive information, that do not involve exposure to the types of aircraft noise of greatest interest, and do not involve low density residential populations.

Tables 8-1 through 8-3 apply these criteria to the study designs discussed in Chapters 5, 6, and 7. The tables evaluate each of the study designs on technical and practical criteria judged in relation to what can be expected from nonexperimental epidemiological research. Categories for the ratings assigned in the columns of Tables 8-1 through 8-3 are as follows:

- *Unit of Analysis*: Studies based on individuals rather than groups are more likely to produce persuasive epidemiologic evidence.
- *Statistical Controls*: Study designs rated "good" permit adjustments for potential confounders such as cholesterol, and take into account such mediating variables as genetic predisposition.
- *Possibility of Causal Inference*: Designs which permit causal inference provide the greatest confidence that a causal chain from noise exposure to health effects can be inferred from an apparent relationship between noise and health.
- *Quantitative Dosage-Effect Relationship*: Accurate measurement of varying levels of exposure allows development of a numeric relationship between dosage and response.
- *Measurement of Individual Noise Exposure*: A rating of "yes" means that accurate inferences of individual exposure are possible.
- *Exposure Duration*: A "short" exposure duration is less than a year; a "moderate" exposure duration is between 1 and 5 years; and a "long" exposure duration study is one greater than 5 years.

Table 8-1: Ability of Community-Based Epidemiologic Studies Near MOAs/MTRs to Meet Criteria.

Ability to Meet Technical Criteria					
Study Design	*Individual Unit of Analysis*	*Quality of Statistical Controls*	*Causal Inference Possible*	*Quantitative Dosage-Response Relationship*	*Measurmnt. of Individual Noise Exposure*
Prospective Cohort	Yes	Good	Yes	Yes	Possibly
Retrospective Cohort	Yes	Intermediate	Yes	Doubtful	No
Retrospective Case-Control	Yes	Poor	Yes	Doubtful	No

Ability to Meet Practical Criteria					
Study Design	*Exposure Duration*	*Study Duration*	*Resources Required*	*Power*	*Potential Controversiality*
Prospective Cohort	Moderate	Long	High	High	High
Retrospective Cohort	Long	Moderate	Moderate	Low to Moderate	Moderate
Retrospective Case-Control	Short	Moderate	Moderate	Low	Moderate

Table 8-2: Ability of Community-Based Epidemiologic Studies in the Vicinity of Airports to Meet Criteria.

Ability to Meet Technical Criteria					
Study Design	*Individual Unit of Analysis*	*Quality of Statistical Controls*	*Causal Inference Possible*	*Quantitative Dosage-Response Relationship*	*Measurmnt. of Individual Noise Exposure*
Prospective Cohort	Yes	Good	Yes	Yes	Possibly
Retrospective Cohort	Yes	Intermediate	Yes	Possibly	No
Retrospective Case-Control	Yes	Poor	Yes	Possibly	No
Ecologic Time Series	No	Very Poor	No	Possibly	No

Ability to Meet Practical Criteria					
Study Design	*Exposure Duration*	*Study Duration*	*Resources Required*	*Power*	*Potential Controversiality*
Prospective Cohort	Long	High	Moderate to Long	High	Moderate
Retrospective Cohort	Moderate to Long	Moderate	High	Moderate	Low
Retrospective Case-Control	Moderate	Moderate	High	Moderate	Low
Ecologic Time Series	Moderate to Long	Short	Low	Low	Low

Table 8-3: Ability of Surrogate Variable Studies of Health Effects of Occupational Noise Exposure to Meet Criteria.

Ability to Meet Technical Criteria					
Study Design	*Individual Unit of Analysis*	*Quality of Statistical Controls*	*Causal Inference Possible*	*Quantitative Dosage-Response Relationship*	*Measurmnt. of Individual Noise Exposure*
Prospective Cohort	Yes	Good	Yes	Possibly	Surrogate Only
Retrospective Cohort	Yes	Intermediate	Yes	Possibly	Surrogate Only
Retrospective Case-Control	Yes	Intermediate	Yes	Possibly	Surrogate Only

Ability to Meet Practical Criteria					
Study Design	*Exposure Duration*	*Study Duration*	*Resources Required*	*Power*	*Potential Controversiality*
Prospective Cohort	Short to Moderate	Long	High	High	Moderate
Retrospective Cohort	Moderate to Long*	Moderate	High	Moderate	Low
Retrospective Case-Control	Short to Moderate*	Moderate	Intermediate	Moderate to Low	Low

*Limited by duration, reliability and availability of records

- *Study Duration*: A "short" duration study is one that can be completed within 2 years; a "moderate" duration study is one that requires 2 to 4 years; and a "long" duration study is one that requires 5 or more years to complete.
- *Resources Required*: Rough estimates of cost are as follows: "low cost" is less than $100,000, "intermediate cost" is between $100,000 and $1,000,000; and "high cost" is more than $1,000,000.
- *Power*: This rating refers to the likelihood of finding a consequential effect. A "high" rating means that the power of the study, in terms of level of noise exposure, sample size, and statistical controls over sources of bias, is great enough that if an effect exists, the study is capable of finding it.
- *Potential Controversiality*: Studies with a rating of "high" are likely to be hampered by lack of cooperation, or may entail considerable risk of adverse publicity.

8.4 Implications of Ranking of Study Designs

8.4.1 Implications for Community Based Studies

It follows from information summarized in Tables 8-1 and 8-2 that most of the studies described in Chapters 5 and 6 of potential health hazards of residential aircraft noise exposure are unlikely to advance the Air Force's primary goals.

First, with the exception of a prospective study at a domestic site discussed in Chapter 6 or at an overseas site, none of the study designs identified in Chapter 5 is capable of yielding definitive evidence of an association between aircraft noise exposure and manifestations of cardiovascular disease. A cohort cannot be identified for a retrospective cohort study in Oklahoma City, and a case-control design could not be undertaken without introducing unacceptable selection bias and risks of missing most of the 1964 population potentially susceptible to the effects of exposure. Findings from either design would be essentially uninterpretable due to generally poor study validity.

Second, the inherently place-oriented noise exposure estimates that could be developed for any of the study designs discussed in Chapter 5 (except for a prospective study) do not apply to any given individual. This lack of individual exposure information forces the level of analysis of any of the studies described in Chapter 5 to be the group, not the individual. As a result, large misclassification errors in noise exposure are unavoidable, seriously compromising study validity.

The accuracy of reconstructed noise exposure is marginal for both sonic booms and

subsonic flyovers. In the case of Oklahoma City, the only available quantitative information about the levels of exposure is in the form of place-oriented probability distributions of peak overpressures. Although it may be possible to distinguish geographic areas with greater and lesser degrees of sonic boom exposure from such distributions, individual exposure levels cannot be determined with useful precision.

Third, in the subsonic noise case for which it is possible to reconstruct aircraft noise exposure with adequate precision (e.g., at a civil airport), the nature of the aircraft noise exposure differs markedly from that produced in the vicinity of MTRs and MOAs. Numbers of operations at major civil airports are typically one or two orders of magnitude greater than the numbers of operations on MTRs. Individual noise events in neighborhoods near such airports with exposure levels comparable to those near MTRs are thus are often lower in peak level. Noise intrusions at civil airports are also highly predictable; ambient noise levels at major civil airports in urban areas are typically higher than in rural areas; and the onset times of individual events at civil airports are longer than those of low altitude, high speed flyovers heard near MTRs.

Except for prospective studies, circumstances of exposure and of exposed populations at major civil airports permit only ecologic analyses which produce geographic associations. The two problems presented by ecologic study in the vicinity of civil airports are:

1. A geographic association is the weakest form of epidemiologic evidence because of the lack of information about the joint distribution of the study exposure and disease within each group. Thus, even if a geographic association were found between aircraft noise exposure and some measure of the prevalence of cardiovascular disease, the Air Force would be no further ahead in its understanding of potential adverse health consequences of aircraft noise exposure than it is at present. The literature already contains ample studies suggesting geographic associations between aircraft noise and admissions to hospitals, low birth weight, teratism, and even death by cirrhosis of the liver.
2. Prevalence rates of cardiovascular disease vary from one locale to another for a large number of known and unknown reasons. It is unlikely that comparison areas for aircraft noise exposed residential neighborhoods can be found that fully control for the known, let alone the unknown, reasons. Because of this inability to control for confounders and other selection biases, overestimation of the effect of noise *per se* is more likely to occur than underestimation. A spurious geographical association uncovered in an Air Force-sponsored study would greatly complicate the efforts of Air Force environmental planners.

Thus, while it is possible to quantify one type of subsonic aircraft noise sufficiently well to support a potential study of geographic association, the type of exposure that can be well quantified is quite different from the type of aircraft noise experienced by people living near MTRs and MOAs.

Prospective epidemiologic studies in the vicinity of low-altitude busy MTRs and/or MOAs

are best suited to providing information of use to Air Force environmental planners. Chapter 6 describes the results of a feasibility analysis to determine whether it is reasonable to attempt such a study. Among the elements required for a feasible study are:

- a sampling plan that includes several communities which are in the vicinity of MTRs as well as MOAs and supersonic ranges;
- further information about catchment areas and sizes for health/medical care facilities most likely to be used by selected communities,
- willingness of medical personnel to cooperate in long term epidemiologic study; and
- direct measurement of noise exposure in communities selected to represent exposed as well as unexposed populations, preferably on an ongoing basis throughout the duration of the study.
- willingness of communities to cooperate over a long period of time with monitoring procedures which offer no direct benefits to participants and are often inconvenient and time consuming.

8.4.2 Implications for Database Studies

The information summarized in Table 8-3 makes it clear that studies involving existing audiometric databases are unlikely to yield information directly useful to Air Force environmental planners about the effects of residential aircraft noise exposure near MOAs, MTRs, and airbases. While the quality and accessibility of information in these databases is rapidly improving, their utility for studies of effects of residential noise exposure on nonauditory health is unlikely to improve without changes in database design and management. The audiometric database system, designed primarily for health monitoring and clinical followup purposes, contains basic hearing loss and threshold shift information which could potentially be linked with other health data systems.

Once linkage of the selected systems is accomplished, two studies that would be useful in the long run to Air Force environmental planners could be conducted prospectively. One study could explore the range of noise conditions and personal characteristics under which sensorineural hearing loss can serve as a valid indicator (surrogate) of noise exposure. A second study could address adverse health consequences of noise exposure as measured by hearing loss, albeit of occupational noise exposure, which may affect the cardiovascular system in different way from super- or subsonic aircraft noise. Use of occupationally based noise effects data in environmental planning of residential aircraft noise exposure probably represents a "worst case scenario." Much of the data currently available to environmental planners for estimating the effects of residential aircraft noise exposure is derived from cross-sectional occupational studies. Information from prospective or retrospective cohort occupational studies would be more appropriate for drawing inferences than these cross-sectional data since information from cohort studies derives from a temporal sequence of cause preceding effect.

8.5 Recommendation for Continued Effort

Residential aircraft noise exposure is only one of many sources of individual noise exposure, and not necessarily a dominant source. Effects of noise on nonauditory health are indirect at best: a link between noise exposure and cardiovascular damage, if it exists, is almost certainly modified by genetic, psychosocial, and other factors. The most plausible mechanism for production of disease from residential aircraft noise exposure is via an interaction of genetic influences and psychological states such as annoyance, which can arguably cause physiological stress capable of influencing cardiovascular function to an unknown degree by poorly understood means. A further complication is that it is difficult to distinguish the effects of stress associated with aircraft noise exposure from that produced by other forms of noise exposure and from stress produced by factors other than noise.

Studies of aircraft noise effects on health that have already been conducted and most of those which could ethically and practically be conducted tend to produce inconsistent results for all of these reasons. Compelling arguments can be made on logical grounds against Air Force sponsorship of additional studies that cannot adequately adjust for factors other than residential aircraft noise exposure which may also be capable of causing disease. It is not intended to leave the impression that most methods to investigate aircraft noise effects on health are so complicated and vulnerable to bias as to be thought dismally hopeless. For all their limitations, well designed and monitored epidemiologic studies are needed, if for no other reason than to provide an estimate of the magnitude of the risk to human health posed by aircraft noise.

Given the lack of evidence for strong, stable effects of residential aircraft noise exposure on nonauditory health, only one of the study designs evaluated is capable of producing results directly useful to environmental planners. This design is a prospective cohort study of the type that might be possible to conduct in the vicinity of low-altitude, high use MTRs and/or MOAs.

More limited goals could be served by epidemiologic studies of existing databases of audiometric and health information. Such studies could confirm the possibility of an association between exposure to high noise levels of any origin and adverse health effects, prior to embarking on a prospective study of residential aircraft noise. Postponing a prospective study that directly addresses the issues at the core of the Air Force's interests in favor of the more indirect approach described in the preceding paragraph may not be the more prudent approach, however, since potential hazards of residential aircraft noise exposure (if any) should be identified as soon as possible.

Glossary

Terms in this Glossary are defined in the sense in which they are used in this report, not necessarily in their broadest sense.

Ambient noise distribution: A distribution of sound pressure levels observed for some duration at some location (see Appendix A for greater detail).

Atheroma: The fatty degeneration of the walls of the arteries in arteriosclerosis.

A-weighted sound level: A single number index of a broadband sound that has been subjected to the A-weighting network (q.v.).

A-weighting network: A frequency-equalizing function intended to approximate the sensitivity of human hearing to sounds of moderate sound pressure level.

Case-control study: An epidemiologic study design in which subjects are selected on the basis of the presence or absence of disease and then studied retrospectively for exposure.

Category measurement scale: An absolute judgment scale in which mutually exclusive labels are used to describe subjective intensity.

CHABA: Committee on Hearing, Bioacoustics and Biomechanics of the National Research Council of the National Academy of Science.

CHD: Coronary Heart Disease.

C-weighted sound level: A single number index of a broadband sound that has been subjected to the C-weighting network.

Cohort study: Study of groups of individuals (cohorts) from the same referent population who are classified on the basis of whether or not they receive exposure, and are followed to see whether or not they develop disease. Prospective cohort studies follow subjects forward in time; retrospective cohort studies follow subjects from exposure to outcome through historical records.

Confounder: A potential causal agent of disease that has not been controlled and, therefore, cannot be isolated from the presumed causal agent (exposure).

Covariate: Variable for which statistical control or adjustment has been made.

Day-Night Average Sound Level: A 24-h energy average sound level with a 10 dB adjustment for night (10 PM - 7 AM) time.

dB: Abbreviation for decibel.

dBA: Symbol for A-weighted sound level; use of alternative symbol, dB(A) is deprecated.

DBP: Abbreviation for diastolic blood pressure, the maximum pressure that can be applied before the heartbeat becomes clearly audible.

decibel: The unit for expressing the product of a constant (usually 10 or 20) and the logarithm to the base 10 of the ratio of a quantity of interest to a reference quantity.

Distant noise process: One component of an ambient noise distribution with a relatively low mean and variance attributable to noise sources distant from any given measurement point.

DNL: Abbreviation for Day-Night Average Sound Level.

Dosimetry: Measurement of individual noise doses in real time.

Ecologic Study: An epidemiologic research design in which the group rather than the individual is the unit of analysis.

Ecologic fallacy: Application of inferences about the causality of diseases in groups to individual members of groups; the assumption that outdoor noise measurements represent individual noise exposure.

Equivalent level: The averaged sound pressure level for a specified duration.

Etiologic Study Designs: Designs which permit inferences of causality of health effects from exposure conditions.

Factor analysis: A statistical technique for grouping variables that are highly intercorrelated, on the assumption that intercorrelated variables represent some underlying, unmeasured, single variable.

Geographic association: A difference between prevalence rates of a disease in distinct geographic areas that does not imply a causal relationship between exposure conditions and the disease.

HMO: Health Maintenance Organization

Incident Cases: New cases of disease occurring during a defined time period, usually after the start of a study.

Induction time: The period of time from causal action until disease is initiated.

Insertion loss (of a residential structure): A frequency-specific reduction in signal energy undergone by an outdoor sound in propagating through the walls of a building; in general, low frequency energy is attenuated much less than high frequency energy. Reductions in signal energy of approximately 15-25 dBA are common, varying with window openings.

Ischemia: A suppression of the flow of blood to an organ or tissue.

Intervening variable: A variable that mediates (accentuates or minimizes) the effect of exposure on health outcomes.

Latency period: The time interval between disease occurrence and detection; frequently used interchangeably with induction period to indicate the interval between first exposure and first appearance of symptoms.

L_{Cdn}: Symbol for C-weighted day-night average sound level.

L_{dn}: Symbol for Day-Night Average Sound Level.

L_{eq}: Symbolic representation of Equivalent Levels. Logarithmic sum over time periods of Sound Exposure Levels (SELs).

Log-linear analysis: A statistical technique for assessing associations among categorical (noncontinuous) variables.

Mantel-Haenszel statistic: A technique for controlling for confounders by stratifying the data into subgroups defined by several covariables. The statistic gives a consistent estimate of the adjusted odds ratio or estimate of relative risk.

Misclassification: Incorrectly labeling of individuals with respect to disease or exposure status.

MOA: Military Operating Area.

MTR: Military Training Route.

Multiple-correlation analysis: (see Regression)

NIHL: Noise Induced Hearing Loss

nm: Nautical mile (6076 feet)

NNI: Noise and Number Index, a noise metric based on a combination of the average Perceived Noise Level and the number of noise events (e.g., aircraft flyovers) heard within a specified period.

Normotensive: Absence of hypo- or hypertension.

Odds Ratio: A measure of association which closely approximates the relative risk for rare diseases. The Odds Ratio in cohort studies is the ratio of the odds of disease given the exposure to the odds of disease given the absence of exposure. In case-control studies, the Odds Ratio is the ratio of the odds of exposure given the disease to the odds of exposure given no disease.

Perceived noise level: A measure of the apparent noisiness of sounds calculated from full or one-third octave band sound pressure levels.

PNdB: The unit of measurement of Perceived Noise Level (PNL).

PNL: Perceived Noise Level.

Power: In statistics, the probability of declaring two populations different when in fact they are different.

Prevalence: Cases of all disease existing at any defined point or period of time.

psf: Pounds per square foot.

Regression analysis: A statistical technique for assessing the correlation between one variable (the dependent variable) and a set of other variables (the independent variables).

Relative risk: The ratio of the probability of disease given the exposure to the probability of disease given the absence of exposure. In case-control studies the relative risk is estimated by the Odds Ratio (q.v.).

Response bias: The willingness to report the presence or absence of a condition independently of any substantive information on which to base a decision.

Risk ratio: See Relative risk.

SBP: Abbreviation for systolic blood pressure, the maximum pressure that can be applied before the heartbeat becomes inaudible.

SEL: Sound Exposure Level (in decibel notation, ten times the logarithm of the ratio of the observed sound pressure to an agreed upon reference level of 20 μPa).

Signal to Noise Ratio: The relative level (in dB) of some characteristic of a signal (e.g., its rms value) and the corresponding characteristic of a distribution of noise.

Sound pressure: A fluctuating pressure superimposed on the static pressure by the presence of sound.

Sound pressure level: In decibels, 20 times the logarithm to the base 10 of the ratio of the time-period, root-mean-square sound pressure, in a stated frequency band, to the standard reference sound pressure--20 micropascals (20 μ Pa).

Stenosis: Narrowing of a passage, such as an artery.

Teratism: Fetal malformation.

Teratogen: An agent causing fetal malformation.

TTS: Abbreviation for Temporary Threshold Shift.

Type I error: Declaring two populations different when in fact they are not different.

Type II error: Failing to declare two populations different when in fact they are different.

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Appendix A
Some Basics of Environmental Noise Exposure

This Appendix, adapted from Fidell et al. (1987), is intended to provide the reader with background information that may be helpful in understanding how noise doses can be quantified in epidemiologic studies.

Exposure, the product of level and duration, is a fundamental quantity of interest for assessing potential physiological effects of aircraft noise, principally because it is a convenient representation of the total acoustic energy produced by noise sources and potentially heard by people. Before the nature of community noise exposure can be understood, however, it is important to understand more general attributes of the acoustic environment.

A.1 The Outdoor Noise Environment

No environmental noises are heard in *isolation*. Even in sparsely populated areas, a long term, time varying distribution of noise levels attributable to wind, water, rustling vegetation, animal sounds, and other natural causes can be heard and measured. Noises of human origin are superimposed upon the natural ambient noise distribution in highly predictable ways. In areas inhabited by modern societies, noises of human origin are superimposed on each other as well. Galloway (1977) has shown that outdoor noise exposure grows directly with population density, as may be seen in Eq. A-1:

$$L_{dn} = 10 \log \rho + 22 \text{ dB} \qquad \text{Eq. A-1}$$

where: ρ is population density in people per square mile and L_{dn} (DNL) is the Day-Night Average Sound Level.

In short, people and their machines make noise; the more people there are per unit area, the more noise is produced.

The mean DNL in uninhabited areas is often on the order of 30-35 dB. In sparsely settled areas (ρ less than or equal to 100) DNL values of 35-40 dB are common; for rural areas (ρ about 500), the estimate is on the order of 50 dB; and in low density suburban areas (ρ about 2,500), the estimate is about 55 dB. In industrial society, transportation noise--both individual vehicle

passbys, and traffic on distant roads--is the major source of community noise exposure. DNL values in the 60-70 dB range are common in major urban areas, and values as high as 80-85 dB are possible in the vicinity of major noise sources such as airports.

A.2 The Indoor Noise Environment

A.2.1 Spatial Sampling of Individual Noise Exposure

Measurements of the noise to which inhabitants of residential neighborhoods are exposed are made by placing an outdoor microphone more or less centrally with respect to a group of homes. It is assumed that neighborhood noise exposure so measured is representative of that of all neighborhood residents. To believe that such outdoor noise measurements are useful reflections of the exposure of individuals indoors, one is forced to assume that most of the noise indoors, where people spend most of their time, comes from outdoors. It is worthwhile to examine this assumption in some detail.

A.2.2 Indoor vs. Outdoor Noise Exposure

Bishop (1973) has compared the statistics of simultaneous outdoor and indoor noise level measurements at a number of quite different locations. The results were expressed as hourly values for the centile levels L_1, L_{10}, L_{eq}, L_{50}, and L_{90}, for both the outdoor and indoor locations, together with the hourly variations of the outdoor-indoor difference in sound levels for each percentile (cf. Figures A-1 through A-3).

If the distribution of indoor noise levels were dominated by noises originating outdoors, one would expect that differences in levels of the indoor and outdoor distributions would remain nearly constant, even though the outdoor level might fluctuate widely. A constant difference could be attributed to the noise reduction afforded by the exterior walls of the building.

Instead, the figures show that this difference fluctuates widely over a range of as much as 30 dB. In fact, the fluctuation of the difference is typically far greater than that of either the outdoor or indoor level alone. Evidently, a large part of the noise in a house is generated indoors and is nearly independent of outdoor events. It is very doubtful, therefore, that an outdoor microphone can correctly characterize indoor noise exposure.

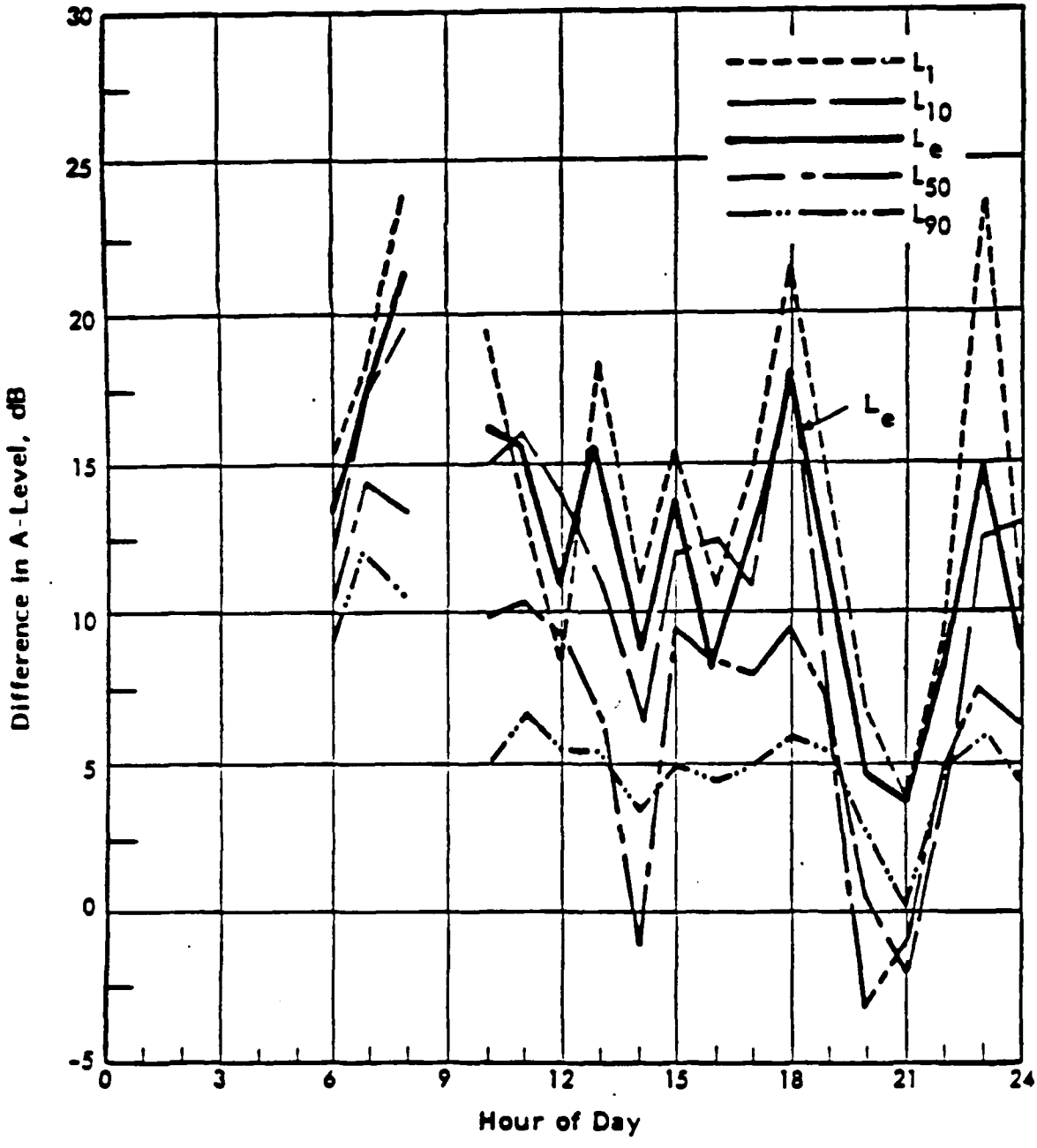

Figure A-1: Difference Between Outside and Inside Hourly Noise Levels at a Suburban Residential Site.

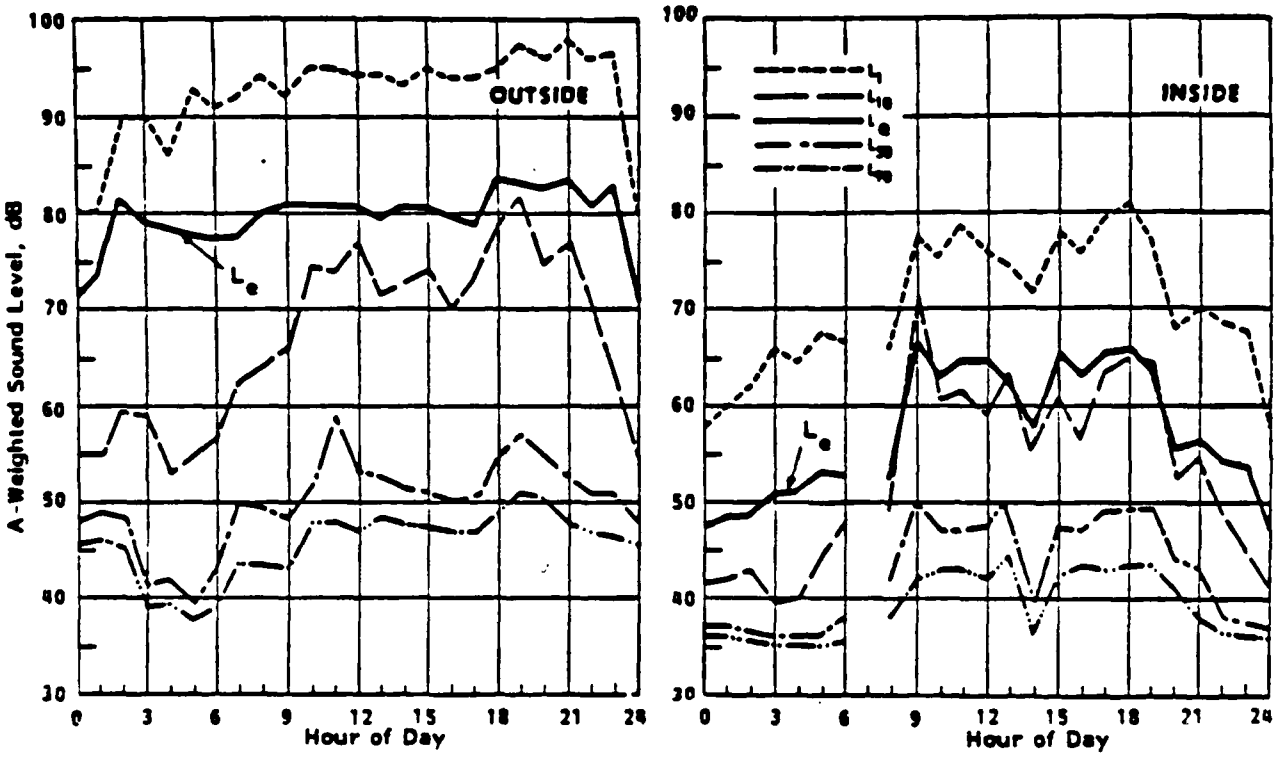

Figure A-2: Hourly Noise Level Time Pattern Outside and Inside a Residential Site Under a Landing Path at a Major Commercial Airport.

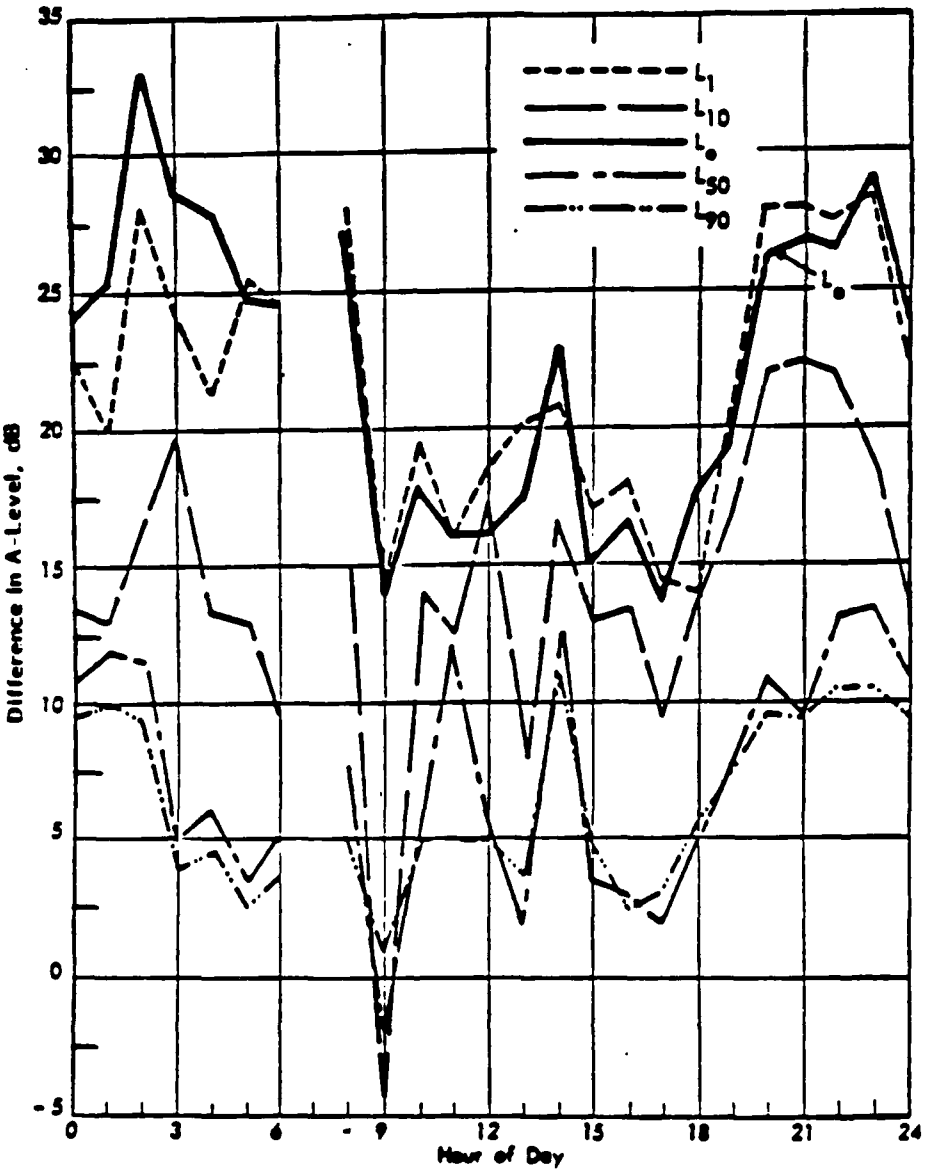

Figure A-3: Difference Between Outside and Inside Hourly Noise Levels at a Residential Site Under Paths Near a Major Commercial Airport.

A.2.3 Place vs. Personal Exposure Differences

This is not the end of the problem, however. It cannot be assumed that a microphone placed inside the house yields a better characterization of the occupants' noise exposure than an outdoor microphone. The results of a pilot study that compared noise exposure recorded by a fixed indoor microphone with the exposure recorded by a microphone mounted near the ear of a mobile occupant are illuminating.

The fixed microphone was placed in the middle of the living room, on the second floor of a three-floor house. The moving microphone was attached to the shoulder of the occupant, and transmitted its signal to a radio receiver in the living room. The signals from both microphones were recorded simultaneously on a two-channel tape recorder for two periods of about 45 min. During the first recording period, the activities of the occupant ranged over all three floors of the house and included typing, handwashing, clearing the table, pouring a drink, rinsing glasses, making beds and various other ordinary household chores. The second period included some vacuum-cleaning of the carpet in the living room.

A comparison of the levels recorded by the fixed and mobile microphones is shown in Figure A-4 for the first 45 min sample and Figure A-5 for the second. The cumulative distribution from the fixed microphone bears almost no relation to that from the moving microphone. In the first period, the L_{10} levels from the two microphones differed by about 17 dB, while the L_5 levels differed by 21 dB. Only for centile levels of 50 or more (i.e., the background noise levels) do the two distributions approach agreement.

During the second recording period, the predominant noise of the vacuum cleaner (in the same room with the fixed microphone) reduced the difference between the noise exposures recorded by the fixed and moving microphones, but a substantial difference remained.

The data shown in Figures A-4 and A-5 demonstrate that a fixed microphone, no matter where it is placed, gives a poor account of the actual noise exposure of active occupants of a dwelling.

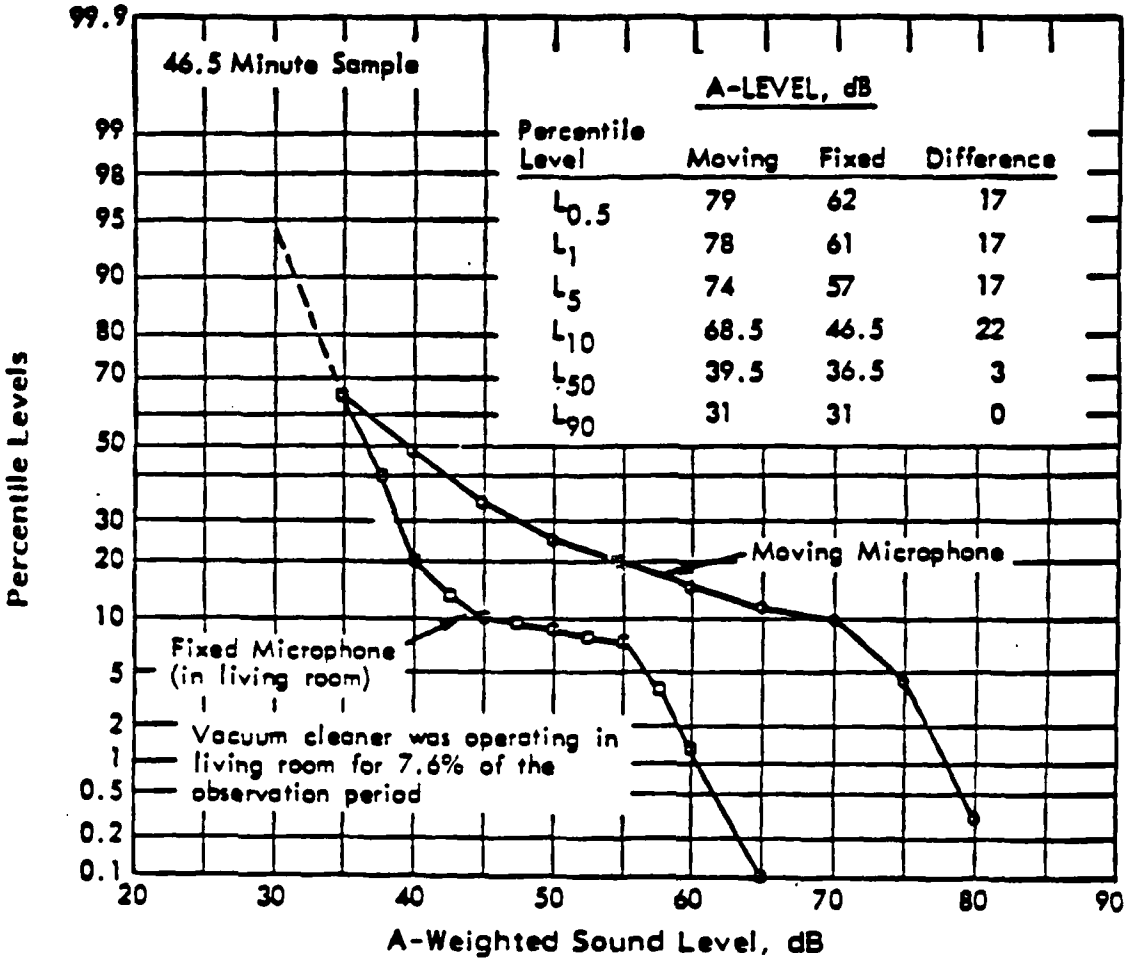

Figure A-4: Comparison of Cumulative Distribution of Levels for Fixed vs. Moving Indoor Microphones Without Vacuum Cleaner Noise.

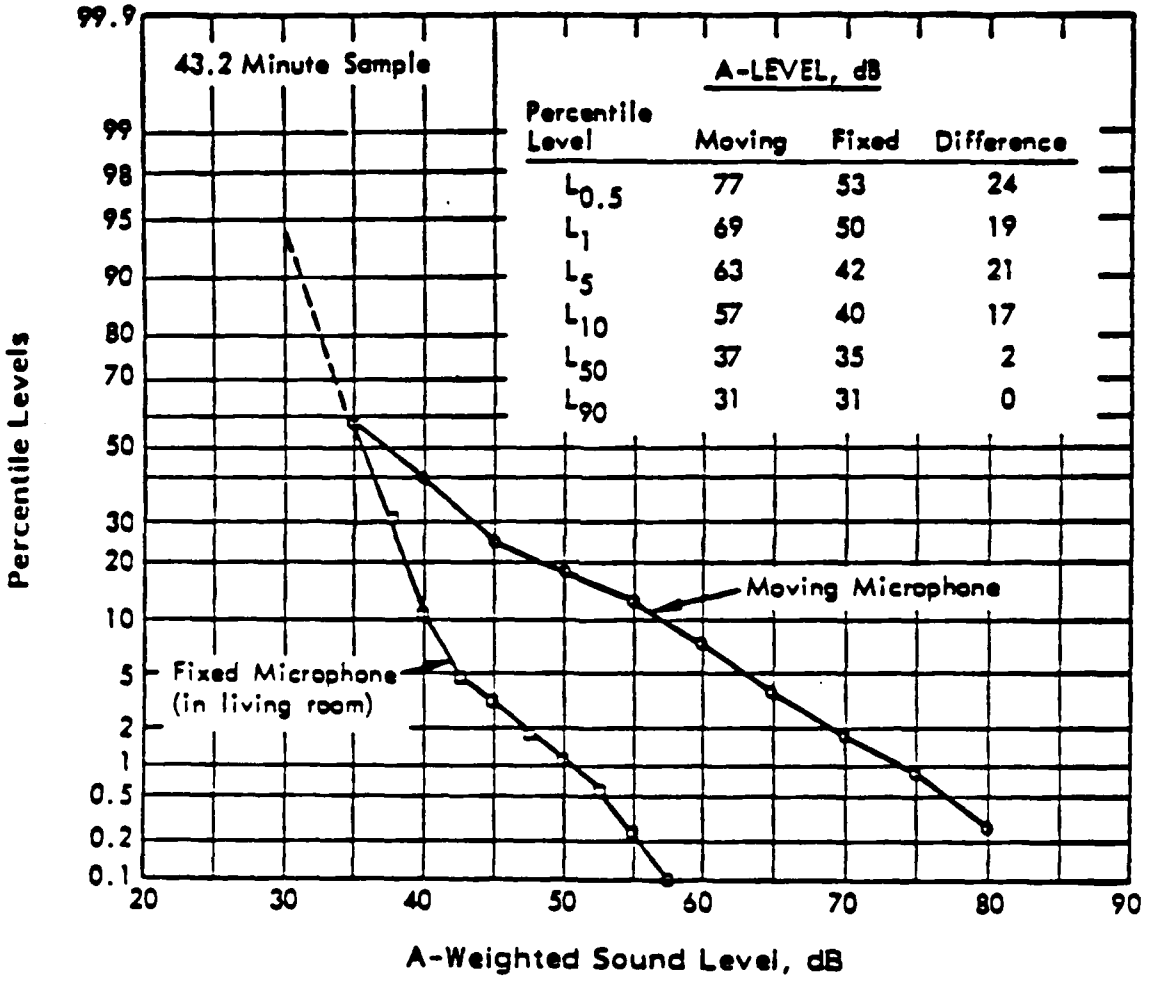

Figure A-5: Comparison of Cumulative Distribution of Levels for Fixed and Moving Indoor Microphones, with Vacuum Cleaner in Room.

A.3 The Relevance of Outdoor Noise to Indoor Exposure

Findings of this sort raise serious questions about the usefulness of outdoor measurements for the prediction of health effects. Similarly, such findings throw doubt on such once-popular noise ratings as the Traffic Noise Index (TNI) and the Noise Pollution Level (NPL), which make a great point of accounting for the variability (i.e., the level fluctuations) of the outdoor noise exposure. The major part of the noise variability that people actually hear may well be self-generated.

In short, the relationship between outdoor noise levels and individual noise exposure is not straightforward. The ecologic fallacy as applied to noise exposure is that outdoor noise exposure measurements in residential neighborhoods are representative of individual noise exposure. People are not stationary objects; they may spend considerable time away from home in much quieter or noisier noise environments; their homes attenuate outdoor noise levels by 10 to 20 dB (or more); and indoor noise environments are often higher in level than outdoor noise environments due to operation of discretionary noise sources. As a general rule, however, it is common for long-term outdoor noise measurements to overestimate indoor residential noise levels, especially in noisier neighborhoods.

An important exception to this rule of thumb is that of low frequency noise. For example, a sonic boom experienced indoors often seems louder than one heard outdoors, because dwellings and other structures transduce inaudible energy at very low frequencies into highly audible secondary emissions, especially rattling noises.

For all these reasons, there is no simple transform that can be used to estimate individual exposure from either indoor or outdoor noise exposure. Since it is impractical to make direct measurements of individual noise exposure on a large scale, it is doubtful that individual exposure will ever be known with great precision and generality. This rationale implies that a dosage-effect relationship for predicting health consequences of outdoor noise exposure is unlikely to yield precise predictions.

A.4 The Nature of Aircraft Noise Exposure

A.4.1 Noise Exposure in Airport Environs

Noise emissions of aircraft can be characterized in a variety of ways. The measures which produce the highest numeric values are the instantaneous peak level (the greatest sound pressure level attained at any time during a flyover, even if the duration of the peak is only a small fraction of a second), and the single event sound exposure level. The latter measure is a summation of all the sound energy occurring during the course of a noise event of arbitrary duration, normalized to a hypothetical one-s interval.

This normalization is accomplished by trading duration for energy, so that a factor of 10 in duration (e.g., the ratio of 10 s to 1 s) is expressed as an additional 10 dB of integrated level. Thus, the sound exposure level of an aircraft flyover longer than 1 s in duration is invariably greater numerically than the actual peak level of the flyover.

Calculation of sound exposure levels for single events provides a convenient means for comparing the total noise energy of flyovers of different durations. It also simplifies estimation of longer term exposure by permitting simple logarithmic manipulations of the noise energy in multiple flyovers to calculate measures such as 24-h equivalent levels, or even annual average DNL values.

According to some estimates, approximately a million people in the United States are exposed to aircraft noise-produced Day-Night Average Sound Levels in excess of 80 dB. These are mostly people living close to major airports, who experience both large numbers of flyovers (hundreds or more per day) and high sound exposure levels for individual flyovers. A great many more people, however, live in neighborhoods with exposure levels in the range of DNL = 70 dB or more. These are people who do not live directly beneath approach or departure flight paths, but within a few thousand meters of flight tracks. Their numbers are probably comparable to those who suffer similar levels of exposure due to their homes' proximity to major highways.

The point of these comparisons is to emphasize that high exposure levels are not generally produced solely by very high individual event levels, but rather by a combination of moderate individual event levels and large numbers of events. In the aggregate, the civil aircraft noise problem in residential areas is more a problem of multiple exposures to moderately high noise events (say, on the order of 70-80 dBA as heard indoors) than a problem of small numbers of exposures to very high level (say, in excess of 85 dBA indoors) individual events.

A.4.2 Noise Exposure Near MOAs and MTRs

Aircraft noise exposure in the vicinity of MOAs differs from that experienced in airport environs in a number of ways:

- Because MOAs are sited in areas of very low population density, the ambient noise environment in the vicinity of MOAs is generally far lower in level than that of airport environs. This exposure implies that individual aircraft noise intrusions are likely to be more audible, and for greater periods in the vicinity of MOAs than in the vicinity of airports.
- The numbers of aircraft operations in MOAs, and hence, cumulative noise exposure levels, are generally lower than in the vicinity of major civil airports.
- Exposure to aircraft noise in MOAs are generally much more intermittent than in airport environs. It is also much more unpredictable than in airport environs.
- Sonic booms are the most prominent aspect of aircraft noise exposure in the vicinity of MOAs in which supersonic operations are conducted.
- Sonic booms are impulsive in character, with durations of a few tenths of a second, but very high instantaneous sound pressure levels. Unlike aircraft noise produced during approaches to or departures from commercial airports, individual noise intrusions produced by sonic booms may vary greatly in level.
- Sonic booms, when heard, may be of sufficient level and unpredictability to startle people.
- Sonic booms experienced indoors can be subjectively louder than those experienced outdoors.
- In many cases, the long-term probability that a sonic boom will be audible on a given day within a relatively small area underneath airspace in which high altitude supersonic flight is conducted is very low--often on the order of 0.01. Nonetheless, persons living or working in areas near MOAs may occasionally hear as many as several sonic booms per day.
- The probability that more than one sonic boom will be audible on a given day at any one point on the ground underneath an area in which air combat maneuvers are practiced is yet lower--often vanishingly small.
- Infrequent carpet booms produced by aircraft operating along well defined flight paths may be audible periodically in certain areas.

Aircraft noise exposure in the vicinity of MTRs also differs from that experienced in airport environs in a number of ways:

- Residences directly beneath flight paths of aircraft operating at high speed and low altitude may be exposed to individual aircraft noise intrusions at considerably higher levels than those in airport environs.
- Onset times of individual noise intrusions may be considerably more rapid than

those characteristic of airport environs--potentially as rapid as 70 dB/s. Such onset times can be nearly as startling as those associated with sonic booms.

- Because communities in the vicinity of MTRs are generally exposed to much smaller numbers of aircraft operations than those in airport environs, cumulative exposure levels are generally considerably lower even though individual event levels may be much higher.

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Appendix B
Discussion of Exclusion of Reproductive and Mental Health Outcomes from Consideration

The discussion of the literature associating noise exposure with reproductive and mental health in this Appendix is provided to support the decisions made in Chapter 4 to focus on cardiovascular effects.

B.1 Reproductive Outcomes

The Committee on Hearing, Bioacoustics and Biomechanics (CHABA) of the National Research Council of the National Academy of Science reviewed the human and animal research on prenatal effects of exposure to high intensity noise at Air Force request in 1982. They found no conclusive evidence of detrimental effects of high intensity sound on the fetus, but suggested that until better information is available, pregnant women should avoid exposures to sound of 90 dB SPL and above. It has been postulated that before 26 weeks gestation, that is, before the auditory system is developed, high intensity noise may affect the fetus directly by mechanical movement or indirectly through the maternal endocrine and circulatory systems; after maturity of the inner ear and its connections to the central nervous system, high intensity sounds external to the mother may produce fetal responses mediated by the fetal auditory system.

The major influences of noise on the human fetus which have been studied are birthweight (prematurity) and teratism (birth defects). Rehm and Jansen (1978) retrospectively studied 1,452 birth records in three areas near Dusseldorf airport. They observed an increase in the low birthweight rate from 5.7% to 6.7% in areas farther and nearer to the airport, respectively. This difference was not statistically significant. Although data on additional variables such as age of mother and social class were collected, they apparently were not controlled in the analysis. The actual levels and durations of noise exposure were not known. Furthermore, the number of cases in each area, especially the area closer to the airport, was judged by the authors to be too small for adequate analysis.

Ando and Hattori (1977) measured the level of placental lactogen in the blood of 343 expectant mothers living near Osaka International Airport and 112 expectant mothers living in a district with little aircraft noise. They found the placental lactogen levels of women in the high-noise area to be lower than those in the low-noise area especially after 36 weeks gestation. Low lactogen levels were associated with lower birthweights for babies whose mothers lived in the noise area.

Knipschild et al. (1981) reported significantly more birthweights below 3,000 grams for single birth infants whose mothers lived in high levels of aircraft noise (L_{dn} = 65-75 dB) than in low noise areas (L_{dn} < 65 dB) in six villages near Amsterdam airport. The hospital birthweight data (which represented about 50% of the births) suggested a gradient effect of low birthweight, with increasing noise level when the high noise level was divided into L_{dn} = 65 to 70 dB and L_{dn} = 70 to 75 dB noise exposure groups.

This study is one of two to date that has controlled for age of mother, sex of infant and family income. Since the authors had previously found smoking not to be associated with aircraft noise, they argue that the relationship is probably not confounded by smoking. The extent to which these small differences in birthweight, if indeed real, are meaningful in terms of survival and growth and development of the infant is unclear. In the distributions of birthweights presented, there is only a 0.8% difference in number of births less than 2,500 grams between the high and low noise areas although the 69-gram difference in mean birthweight between the two noise groups is statistically significant ($p = .03$).

It is unclear why 3,000 grams was used as the defining level of low birthweight when the World Health Organization has set 2,500 grams at birth as a criterion for prematurity, and 2,000 grams is more frequently suggested as the criterion when survival of low birthweight infants is the issue (Alberman, 1984, p. 86). It has been demonstrated that a simple change from a definition of "2,500 grams or less" to one of "less than 2,500 grams" can significantly decrease the percentage of low birthweights reported because of the tendency to digit preference. Data from England and Wales showed that such a change in the criterion for low birthweight in 1978 decreased the percentage of low birthweight infants from 7.09 to 6.74 (Alberman, 1984, p. 87).

One study has been reported concerning both length of gestation and birthweight of infants of mothers who were exposed to aircraft noise while pregnant (Schell, 1981). A multiple correlation analysis, partialing out the effects of mother's height, age, weight, smoking habits and father's weight and education, revealed a statistically significant negative correlation between aircraft noise exposure level and gestation length for female, but not male infants. Correlations between noise exposure levels and birthweights were not statistically significant.

In 1978 Jones and Tauscher showed a higher incidence of reportable birth defects in census tracts partly or wholly within an idiosyncratically defined peak individual aircraft noise contour at the Los Angeles Airport than in the remainder of the county. As Bader (1978) points out, these birth certificate data are subject to such wide variation due to reporting practices that the findings are not meaningful. Although the data were analyzed separately for Black and White births, other environmental and genetic factors were not considered. Bader demonstrated, using similar birth certificate data, that the rates of birth defects in the cities near the Seattle-Tacoma airport were even lower than rates for the remaining areas. Edmonds et al. (1979) conducted a similar study for the Atlanta airport using data from the Metropolitan Atlanta Congenital Defects

Program, which employs multiple methods of case ascertainment. They found no differences in the rates of 17 categories of defects between the high (L_{dn} ≥65 dB) and low noise (L_{dn} < 65 dB) census tracts controlling for hospital of birth, socioeconomic status and race. A matched case-control study of all neural tube defects showed no statistically significant association between high noise area and neural tube anomalies.

Although the CHABA Working Group (National Research Council, 1982) recommends that searches be made of exiting sources of data on women who have been exposed to noise during pregnancy for the purpose of retrospective analysis, it is unlikely that data meeting the criteria specified by the group exist even in industry. Suggested criteria included careful recording of data and the inclusion of information such as period of exposure during pregnancy, daily duration of exposure, measured noise levels, and documentation of all environmental pollutants involved. Difficulties in acquiring such data are evident in three recent industrial studies examining the effects of environmental conditions, including noise exposures, on pregnancy outcomes.

Mamelle and colleagues (1984) reported a systematic increase in the rate of prematurity due to preterm birth with a rising fatigue index score based on presence or absence of specified aspects of posture, work on industrial machines, physical exertion, mental stress and adverse environment. Adverse environmental conditions were defined as exposure to at least two of three elements: noise, heat, and cold. Although a relative risk of 1.9 (with lower and upper bounds on the 95% confidence interval of 1.3 and 2.8) for preterm births for women working under adverse environmental conditions was observed, when the number of loud noises was considered alone, the risk of 1.6 (with lower and upper bounds on the 95% confidence interval of 0.9 and 2.9) was not statistically significant. In this study of 1928 working women, medical data were collected from obstetric records, but all other information including noise exposure was obtained from a 150 item questionnaire administered to the women immediately after delivery.

Recent data from a study by McDonald et al. (1988) indicate an association between noise and low birthweight (O/E 1.11, p =.02), but not with preterm births, in the health and manufacturing sectors of industry (as defined in the Standard Occupational and Industrial Classification of Statistics, Canada). Information on environmental conditions including noise and vibration for the 22,761 pregnant women in paid employment in Montreal 1982-84 who had single live births was obtained by interview after delivery as in the Mamelle et al. (1984) study. Approximately 50% of the women in manufacturing and 27% in health services reported "great" noise in the work environment. In addition to factors such as previous premature birth, parity, SES, ethnic status and pregnancy pathology that were controlled statistically in the Mamelle et al. study, cigarette smoking, height and alcohol consumption were included in the Canadian analyses.

McDonald and colleagues claim that subject and observer bias is not very probable after the

birth of a premature but usually healthy live infant; that their recording and classification of type of employment could not easily have been influenced by pregnancy outcome; and that confounding factors were adequately taken into account in the analyses. However, they question their findings, arguing that undefined factors such as those associated with selection for certain types of employment could explain the association between noise and low birthweight but not preterm delivery. It also seems plausible that physical stress and fatigue could induce premature labor, but would be less likely to influence birthweight, even though the mechanism for any association between noise and pregnancy outcome is obscure.

A matched-pair case control study of 284 women with preterm deliveries and 299 women with full-term low birthweight infants showing no difference in noise exposure between cases and controls adds little to the accumulated evidence of prenatal effects of exposure to high levels of noise (Hartikainen-Sorri et al., 1988). Only 3.5% of the women in this Finnish study were exposed to occupational noise greater than or equal to 81 dB $L_{eq(A)8h}$, making any risk estimation too imprecise to draw definitive conclusions.

The CHABA working group also recognized that special difficulties would be encountered in studies of aircraft noise if exposure to high levels of noise is usually infrequent, if individual exposure is difficult to ascertain, and if other stresses may exist should noise interfere with certain activities or serve as a reminder of the danger of aircraft accidents.

It seems unlikely that appropriate data exist for determining in any definitive manner, the effects of subsonic and supersonic aircraft noise on the infants of women exposed during pregnancy because of a myriad of difficulties and limitations, of which a few are listed below.

- Exposure to aircraft noise is difficult to measure because exposures may occur infrequently and because dose depends upon multiple physical and behavioral situations surrounding the individuals of interest. Furthermore, the determination of who is exposed must accommodate the biologic question of whether an external measure of noise, even by dosimeter, is adequate for describing fetal exposure. Multiple exposures to noise from several sources are likely to occur.
- In the study of birth defects and perhaps low birthweight, it is necessary to know, with some precision, the timing of exposure (such as age at first *in utero* exposure and age at peak exposure) with reference to embryologic development as well as duration of exposure. This information requires knowledge of gestational age; it is widely recognized that gestational ages are frequently unknown or erroneous--20-30% error rate--even when some of the newer assessment methods are used at birth. Low birthweight includes children born too early, i.e., who are truly premature, and infants who are small for gestational age, or both. Accumulating evidence suggests that these conditions may represent different etiologies (Berendes, 1987). This evidence may partially account for Schell's (1981) finding of a negative correlation between noise and gestational age but not for birthweight. Since states and hospitals within states vary as to definitions and recording of fetal deaths, there is marked variation in the reported proportion of live

births under 500 grams. Some states require only reporting of fetal deaths of more than 20 weeks gestation. Although some states with under-reporting of live births of 500 grams or less may report them as fetal deaths, this only complicates the problem of identifying low birthweight infants. Until data on gestational age and birthweight are readily available and/or readily obtainable for most births, retrospective studies utilizing birth certificate data are not likely to produce interpretable nor informative data.

- The incidence of low birthweight is relatively low in the United States (6.7% of all live births), the incidence of most malformations is very low (less than 1%), and the frequency of exposure to noise and many other potential etiologic agents such as drugs is also quite low. Thus, studies of many thousands of women are likely to be required to detect adverse effects.

- There is a great deal of variation around the country and from hospital to hospital in the identification and recording of congenital malformations at birth. Only detailed examination of each infant at birth and for the first 5 or 6 years of age is likely to identify many anomalies which present in ways too subtle to be uncovered at birth. Studies of the effects of noise as a teratogen should probably focus on specific malformations if Rosenberg et al. (1982, p. 1432) are correct when they state that "no teratogen has yet been identified that causes defects uniformly across the broad spectrum of malformations."

- *Perinatal epidemiologic* research in this country has only recently begun to identify and measure with reasonable precision some of the major factors which influence prenatal outcomes of birthweight and malformations. To be meaningful today, studies must not only take into account major risk factors such as maternal education, age, parity, maternal weight to height, medical problems, differences in smoking, alcohol and drug use, nutritional status, but must include birthweight of both mother and father since some of the observed differences may be genetic in nature. The complexities of the field suggest that until noise exposure, other possible etiologic factors and fetal outcomes can be adequately assessed and routinely recorded for large populations of women, retrospective analyses will not contribute much new insight to the effect of noise on fetal health.

- To elucidate the complex interrelationship of the many and varied factors influencing fetal development, it seems prudent to heed Leon Gordis' (1986, p. 544) warning that "if the epidemiologic findings from a study do not make biologic sense at the present time, they should be viewed with caution even if they are found to be statistically significant." It is not yet clear from appropriate animal models or from extant human studies just how noise may impact the developing fetus.

In summary, the rarity of the events and the role of genetics in the etiology of congenital anomalies make birth defects a health outcome of questionable import to the understanding of possible adverse effects from noise exposure (Kryter, 1985, p. 504).

B.2 Mental Health Outcomes

Support for the contention that aircraft noise exposure is positively linked to mental illness is at best equivocal. In fact, once findings of studies are put in proper perspective by considering study design, measurement of noise exposure and mental health status, and statistical evidence, there appears to be no good scientific basis for inferring an association between noise and onset of psychiatric problems.

Abbey-Wickrama and colleagues (1969) compared psychiatric hospital (Springfield) admission records of 1966-1968 from a maximum noise area around Heathrow Airport to admissions from a less noisy section of the Borough. The maximum noise area was defined as the area where sound levels were over 55 NNI or where the perceived noise level (expressed in units of PNdB) from approaching aircraft was above 100 PNdB. Although noise levels in the less noisy area were not given, the area was described as very noisy. Analysis of a small sample of the 1966 census from each area indicated that residents were fairly homogeneous as to age, sex, marital status, population density, migration and socioeconomic status. No attempt was made to statistically control for these variables since the exact distribution of characteristics in the 2 populations was unknown. Admissions were divided into first admissions for the problem and total person admissions. Some 14 of 42 statistical tests were significant. The authors concluded that total and first psychiatric admission rates were significantly higher in the maximum noise area than in the less noisy area; significant differences existed for females over 45 years of age and for females who were single, widowed, separated or divorced. No significant differences were noted for males and no information as to differences in time spent in the residences between males and females was provided. Among females the only diagnostic categories that revealed significant differences were neuroticism and organic mental illness.

After the demographic analysis and the use of a combined noise exposure index was challenged by Chowns (1970), Gattoni and Tarnopolsky (1973) conducted a similar study of inpatient admission rates to the same hospital for the years 1970-1972. The demographic analysis was refined by comparing the high and low noise zones on socioeconomic variables associated with psychiatric morbidity, by calculating 2-year referral rates per 1,000 population at risk, and by age-standardizing the data. The high noise zone was defined by taking the 50 NNI contour as the boundary between the high and lower noise areas. This reanalysis showed no statistically significant results.

In yet a third study of admissions to Springfield Hospital, Jenkins et al. (1979) produced results which conflicted with earlier findings, noting an increase in admission rates in the lower noise area. In this study, the investigators attempted to address the problems in design of the 2 earlier exploratory studies. Data were collected from the 3 hospitals serving the whole

catchment area; noise was defined by 4 levels (25-34, 35-44, 45-54 and 55+ NNI) and by two sets of contours, 1 for 1969-70 and 1 for 1971-72. The complete 1971 census was used and the population was divided into enumeration districts by age, sex, marital status (persons in old people's homes and other similar institutions were excluded). The lower noise-area population showed greater mobility, more one-person households, more unemployment and rented homes and fewer high occupations while the high area included only one subgroup considered vulnerable to admissions, those educationally qualified above the A standard. Population base rates were constructed in a manner similar to that of Gattoni and Tarnopolsky (1973). Data were not available for adjusting on demographic variables. There were probably some underlying differences in noise exposure, the populations at risk, the proportion of cases missed and hospital admission policies between the 2 studies. Nevertheless, the latter survey certainly represents the more scientifically sound study design--referring to a longer time with a more precisely defined population base, covering a larger geographical area and specifying more detailed noise levels.

Nine thousand persons admitted to the 3 large psychiatric hospitals just described were studied further in an effort to control for the differences in social and economic characteristics of persons in the different noise areas (Jenkins et al., 1981). Since no attribution could be made of some important variables (house tenancy, mobility, unemployment, occupation) to a particular hospital case, the population was stratified and specific rates were examined from districts of high/low home ownership etc., divided by noise exposure. Log-linear analysis was employed. The previously observed trends could all be attributed to non-noise characteristics of the population, suggesting that if any aircraft noise effect exists, it is subtle and interacts with many other variables.

It appears from the resulting followup of the Abbey-Wickrama et al. (1969) study of aircraft noise that the original finding of higher psychiatric admission rates from areas of high noise resulted from inadequate sampling and faulty analysis. Similarly, the study by Meecham and Smith (1977) reporting higher psychiatric admission rates from the areas of high noise close to the runways of the Los Angeles Airport, suffers from failure to control for any confounding variables.

Several large community surveys in Switzerland and in England have shown no relation between scores on mental health scales and noise exposure (cf. McLean and Tarnopolsky, 1977; Tarnopolsky, 1978; and Stansfeld et al., 1985). Grandjean et al. (1973) administered a 30-item-questionnaire intending to measure a "mental health status factor" in about 4,000 persons living around 3 Swiss airports. The questionnaire made no mention of aircraft noise as a possible cause of symptoms of ill health. Although not validated, the questionnaire was judged by Tarnopolsky to be similar in content to epidemiological instruments used to screen for psychiatric illness. No evidence was found of an association between the self- rated symptoms and noise exposure. The survey did show significant correlations between visits to doctors, use of hypnotics and

tranquilizers and noise exposure. It also showed a positive relation between psychiatric disorder and noise sensitivity and annoyance.

In a more systematic study of mental health effects occurring under normal urban noise, Tarnopolsky (1978) and Tarnopolsky et al. (1980) found no evidence of a relation between scores on a General Health Questionnaire (GHQ) nor the proportion of confirmed psychiatric cases and noise exposure. The GHQ had been validated against a standardized psychiatric interview conducted by a trained psychiatrist and consisted of some 30 questions selected on the basis of statistical analysis after testing in large samples. The community study consisted of about 6,000 persons exposed to different levels of aircraft and traffic noise in London. When road traffic noise was statistically controlled, there remained no association between aircraft noise and psychiatric disorder. Findings of an association between psychiatric disorder and noise sensitivity were consistent with the observations of Grandjean and colleagues.

Further analysis of the London household survey data has shown that while acute symptoms such as depression increase with noise exposure, most chronic symptoms are found in low noise conditions (Tarnopolsky et al., 1980). Use of medications, general practitioner services, hospital facilities and community services were no greater in the high noise than in the low noise area (Watkins et al., 1981). However, most symptoms and the use of drugs and services were related to increasing annoyance in both high and low noise areas. Since it had been suggested repeatedly that noise-sensitive individuals respond differently to noise than less sensitive individuals, this issue was examined in a subsample of 77 women from the high and low exposure areas defined in the West London Survey. Noise sensitive women exhibited significantly more psychiatric symptoms than women with less noise sensitivity (Stansfeld, et al. 1985). Stansfeld and colleagues utilized several measures of noise sensitivity including the McKennell Noise Sensitivity Index and the General Noise Questionnaire which define sensitivity by responses to questions about annoyance of common noises.

These studies suggest there are very complex interactions between psychiatric disorders, sensitivity to noise, and noise annoyance. However, they provide no evidence that noise leads to psychological morbidity in the general population nor in the so-called noise sensitive individual. No study to date has satisfactorily controlled for the effects of the many variables that are known to influence the relationship between exposure to noise and admission to hospitals. The process of moving from the community to a psychiatric inpatient facility has also been shown to be affected by many different psychosocial factors not directly related to disease--factors which are likely to be more powerful than noise in determining hospital admissions. Of interest in drawing causal inferences is the fact that admissions to a psychiatric hospital, even first admissions, do not necessarily reflect incidence of mental disorders.

To study the association between noise and psychiatric disorders in the least ambiguous fashion, more weight should be put on studies using samples representative of the community.

Community based studies are especially difficult because there are few valid and reliable means for assessing mental health states and psychiatric disorders. Thus, there is the potential for large misclassification error in any study. For these reasons and because the evidence of a possible association between aircraft noise exposure and psychiatric disorders is so very weak, mental health outcomes are not considered further in this feasibility study.

References for Appendix B

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Appendix C
ORNL Database

C.1 Ranking Scheme using ORNL Database

Section 6.1.2 contains tables showing rankings of military airspaces in terms of exposure, a weighted product of population and noise. In each of these tables, population and noise values were obtained from the ORNL database. Military airspaces were ranked in terms of several population and noise criteria. The following shows, in detail, the database files and fields used in those computations.

Columns in Tables C-1 and C-2 show rank ordering of MTRs and computed values on which ranks are based, according to the following scheme.

Population rankings:

- Total ranks routes on the TOTALPOP[24] field of the ROUTE.DBF file, the total population overflown.
- Weekend uses the ROUTE.DBF file and ranks routes on TOTALPOP overflown on weekends, for those routes which are available for scheduling on weekends (WEEKENDS equal to T).
- Nighttime uses the ROUTE.DBF file and ranks routes on TOTALPOP overflown at night (ENDTIME not equal to SS).
- Low Level ranks routes on LOWLEVPOP from the ROUTELOW.DBF file, the population overflown under low altitude portions of the route.
- Duplicated Population ranks routes on POPDUP from the ROUTE.DBF file, the population under portions of the route which duplicates itself.
- Low Level Duplicated ranks routes on LOWPOPDUP from the ROUTELOW.DBF file.

Noise rankings:

- Number of Sorties ranks the average number of sorties for each route using the NUMFLIGHTS field from the ROUTECRA.DBF file, with PLANETYPE equal to TOTAL.
- Low Altitude Noise ranks the noisiness of routes in terms of the sum over each route

[24]Capitalized abbreviations follow those used in the ORNL database

of the triple product of the number of each PLANETYPE (linking the ROUTECRA.DBF and AIRCRAFT.DBF files) times the SEL-AT-400 (the Sound Exposure Level measured in dB at 400' from the AIRCRAFT.DBF file) times NUMFLIGHTS (number of sorties from the ROUTECRA.DBF files) for those routes with MINAGL (minimum ground level altitude in feet from the ROUTE.DBF file) greater than 200 but less than 600. For those routes with MINAGL equal to or less than 200, SEL-AT-200 (from the AIRCRAFT.DBF file) is used instead.

- Low Altitude Night Noise is the same as the previous column, except that MINAGL-PM (minimum ground level altitude in feet during nighttime hours from the ROUTE.DBF file) is used instead of MINAGL.

Tables C-3, C-4 and C-5 show the same columns, with computed values entered rather than rankings. Columns in Table C-5 follow the same format as those in Table 6-1 in Section 6.1.2.

Tables C-6 and C-7 show orderings and computed values for MOAs according to the following scheme:

Population rankings:

- Nonrural ranks areas on nonrural population (TOTALPOP minus RURPOP, both from the MOA.DBF file) beneath each MOA. Nonrural is defined as more than 500 people per square mile.
- Weekend ranks areas on TOTALPOP overflown on weekends, for those areas which are available for scheduling on weekends (WEEKENDS, from the MOA.DBF file, equal to T).
- Nighttime ranks areas on TOTALPOP overflown at night (ENDTIME, from the MOA.DBF file, not equal to SS).

Noise rankings:

- Number of Sorties ranks the average number of sorties for each route using the NUMFLIGHTS field from the MOACRAFT.DBF file, describing the average number of sorties scheduled, with PLANETYPE equal to TOTAL.
- Low Altitude Noise ranks the noisiness of routes in terms of the sum over each route of the triple product of the number of each PLANETYPE (linking the MOACRAFT.DBF and AIRCRAFT.DBF files) times the SEL-AT-400 (the Sound Exposure Level measured in dB at 400' from the AIRCRAFT.DBF files) times NUMFLIGHTS (number of sorties from the MOACRAFT.DBF files) for those routes with MINAGL (minimum ground level altitude in feet from the MOA file) greater than 200 but less than 600. For those routes with MINAGL equal to or less than 200, SEL-AT-200 (from the AIRCRAFT.DBF file) is used instead.
- Low Altitude Night Noise is the same as the previous column, except that MINAGL-PM (minimum ground level altitude in feet during nighttime hours from the MOA.DBF file) is used instead of MINAGL.

Table C-1: Rankings of MTRs by Population.

Route	*Total*	*Week-ends*	*Night-Time*	*Low Level*	*Dupli-cated*	*Low Level Duplicated*
IR0023	45	45	41	40	23	22
IR0042	58	57	52	53	23	22
IR0062	5	5	4	5	13	12
IR0069	18	18	17	16	5	5
IR0074	22	22	21	75	23	22
IR0075	7	7	6	10	2	2
IR0084	39	39	38	98	23	22
IR0089	31	31	30	96	23	22
IR0090	32	32	31	97	23	22
IR0133	2	2	1	2	22	21
IR0715	12	12	11	89	23	22
IR0721	14	14	13	39	23	22
IR0726	23	23	22	20	20	19
SR0035	53	53	49	48	8	7
SR0036	16	16	15	14	10	9
SR0037	24	24	23	21	8	7
SR0040	50	50	46	45	23	22
SR0059	51	51	47	46	23	22
SR0225	28	28	27	25	23	22
SR0701	29	29	28	26	23	22
SR0702	35	35	34	30	17	16
SR0707	49	49	45	44	23	22
SR0732	59	58	53	54	23	22

Table C-1: continued.

Route	*Total*	*Week-ends*	*Night-Time*	*Low Level*	*Dupli-cated*	*Low Level Duplicated*
SR0771	34	34	33	29	23	22
SR0774	57	56	51	52	4	4
SR0801	36	36	35	31	17	16
SR0823	19	19	18	17	23	22
SR0824	41	41	40	35	23	22
SR0825	20	20	19	18	23	22
SR0826	6	6	5	6	6	6
SR0845	37	37	36	32	23	22
SR0847	26	26	25	23	23	22
SR0873	30	30	29	27	9	8
SR0900	1	1	1	1	23	22
SR0901	11	11	10	11	3	3
SR0902	9	9	8	8	23	22
SR0904	4	4	3	4	23	22
VR0058	10	10	9	9	23	22
VR0086	40	40	39	34	23	22
VR0087	8	8	7	7	23	22
VR0088	38	38	37	33	14	13
VR0092	10	10	9	9	23	22
VR0093	3	3	2	3	19	18
VR0095	17	17	16	15	23	22
VR0704	43	43	89	37	23	22
VR0705	54	54	89	49	23	22
VR0707	44	44	89	38	23	22

Table C-1: continued.

Route	*Total*	*Week-ends*	*Night-Time*	*Low Level*	*Dupli-cated*	*Low Level Duplicated*
VR1016	42	42	89	36	23	22
VR1023	55	99	50	50	11	10
VR1050	52	52	48	47	23	22
VR1051	27	27	26	24	23	22
VR1052	21	21	20	19	23	22
VR1055	47	47	43	42	23	22
VR1056	21	21	20	19	23	22
VR1059	33	33	32	28	23	22
VR1060	46	46	42	41	23	22
VR1064	13	13	12	12	23	22
VR1068	58	57	52	53	23	22
VR1145	56	55	89	51	23	22
VR1146	56	55	89	51	23	22
VR1653	13	13	12	12	23	22
VR1721	48	48	44	43	23	22
VR1751	15	15	14	13	23	22
VR1752	25	25	24	22	21	20

Table C-2: Rankings of MTRs by Flight Activity and Product of Activity x Aircraft Noisiness.

Route	*Number of Sorties*	*Low Altitude Noise*	*Low Altitude Night Noise*
IR0023	44	65	57
IR0042	37	45	40
IR0062	21	86	75
IR0069	29	36	34
IR0074	40	55	48
IR0075	7	8	8
IR0084	32	86	75
IR0089	42	61	54
IR0090	41	57	50
IR0133	5	6	6
IR0715	27	86	75
IR0721	18	20	20
IR0726	40	54	47
SR0035	42	63	55
SR0036	49	83	73
SR0037	42	63	55
SR0040	40	62	75
SR0059	48	81	71
SR0225	49	82	72
SR0701	39	58	51
SR0702	38	56	49
SR0707	45	72	63
SR0732	34	49	75

Table C-2: continued.

Route	*Number of Sorties*	*Low Altitude Noise*	*Low Altitude Night Noise*
SR0771	23	30	28
SR0774	45	72	75
SR0801	37	53	75
SR0823	45	73	64
SR0824	45	70	61
SR0825	46	75	66
SR0826	44	69	60
SR0845	40	59	52
SR0847	40	59	52
SR0873	32	43	75
SR0900	6	7	7
SR0901	35	51	45
SR0902	46	76	67
SR0904	42	63	55
VR0058	27	35	33
VR0086	43	64	56
VR0087	9	11	11
VR0088	4	4	4
VR0092	27	35	33
VR0093	47	77	68
VR0095	15	16	16
VR0704	19	21	75
VR0705	12	14	75
VR0707	26	34	75

Table C-2: continued.

Route	*Number of Sorties*	*Low Altitude Noise*	*Low Altitude Night Noise*
VR1016	44	68	75
VR1023	44	22	22
VR1050	32	40	37
VR1051	28	38	35
VR1052	22	32	30
VR1055	16	19	19
VR1056	22	32	30
VR1059	11	13	13
VR1060	24	26	25
VR1064	25	27	26
VR1068	34	46	41
VR1145	14	18	75
VR1146	31	42	75
VR1653	25	27	26
VR1721	48	80	70
VR1751	25	31	29
VR1752	3	3	3

Table C-3: MTR Population Values.

Route	*Total*	*Week-ends*	*Night-Time*	*Low Level*	*Dupli-cated*	*Low Level Duplicated*
IR0023	149203	149203	149203	149203	0	0
IR0042	133240	133240	133240	133240	0	0
IR0062	377004	377004	377004	377004	2533	2533
IR0069	226278	226278	226278	226278	14596	14596
IR0074	204916	204916	204916	112405	0	0
IR0075	367993	367993	367993	280339	21856	21856
IR0084	165966	165966	165966	54421	0	0
IR0089	174624	174624	174624	96883	0	0
IR0090	173950	173950	173950	96384	0	0
IR0133	446392	446392	446392	446392	1	1
IR0715	261446	261446	261446	102223	0	0
IR0721	258654	258654	258654	149583	0	0
IR0726	204131	204131	204131	204131	27	27
SR0035	140374	140374	140374	140374	7968	7968
SR0036	247847	247847	247847	247847	4446	4446
SR0037	198981	198981	198981	198981	7968	7968
SR0040	146204	146204	146204	146204	0	0
SR0059	144655	144655	144655	144655	0	0
SR0225	180452	180452	180452	180452	0	0
SR0701	177963	177963	177963	177963	0	0
SR0702	169723	169723	169723	169723	56	56
SR0707	147692	147692	147692	147692	0	0
SR0732	131109	131109	131109	131109	0	0
SR0771	170804	170804	170804	170804	0	0

Table C-3: continued.

Route	*Total*	*Week-ends*	*Night-Time*	*Low Level*	*Dupli-cated*	*Low Level Duplicated*
SR0774	133590	133590	133590	133590	20287	20287
SR0801	167478	167478	167478	167478	56	56
SR0823	223264	223264	223264	223264	0	0
SR0824	163643	163643	163643	163643	0	0
SR0825	214196	214196	214196	214196	0	0
SR0826	368517	368517	368517	368517	10789	10789
SR0845	166582	166582	166582	166582	0	0
SR0847	184652	184652	184652	184652	0	0
SR0873	175931	175931	175931	175931	7644	7644
SR0900	623442	623442	623442	623442	0	0
SR0901	277721	277721	277721	277721	21359	21359
SR0902	303758	303758	303758	303758	0	0
SR0904	405301	405301	405301	405301	0	0
VR0092	284023	284023	284023	284023	0	0
VR0086	165878	165878	165878	165878	0	0
VR0087	307170	307170	307170	307170	0	0
VR0088	166380	166380	166380	166380	262	262
VR0058	284023	284023	284023	284023	0	0
VR0093	411609	411609	411609	411609	34	34
VR0095	229306	229306	229306	229306	0	0
VR0704	159402	159402	0	159402	0	0
VR0705	137723	137723	0	137723	0	0
VR0707	150746	150746	0	150746	0	0
VR1016	161014	161014	0	161014	0	0

Table C-3: continued.

Route	*Total*	*Week-ends*	*Night-Time*	*Low Level*	*Dupli-cated*	*Low Level Duplicated*
VR1023	136534	0	136534	136534	2562	2562
VR1050	144220	144220	144220	144220	0	0
VR1051	181641	181641	181641	181641	0	0
VR1052	212721	212721	212721	212721	0	0
VR1055	148788	148788	148788	148788	0	0
VR1052	212721	212721	212721	212721	0	0
VR1059	171045	171045	171045	171045	0	0
VR1060	149026	149026	149026	149026	0	0
VR1064	260830	260830	260830	260830	0	0
VR1068	133240	133240	133240	133240	0	0
VR1146	134133	134133	0	134133	0	0
VR1145	134133	134133	0	134133	0	0
VR1653	260830	260830	260830	260830	0	0
VR1721	148363	148363	148363	148363	0	0
VR1751	256445	256445	256445	256445	0	0
VR1752	194122	194122	194122	194122	11	11

Table C-4: MTR Flight Activity and Product of Activity x Aircraft Noisiness.

Route	*Number of Sorties*	*Low Altitude Noise*	*Low Altitude Night Noise*
IR0023	6	712	712
IR0042	13	1578	1578
IR0062	37	0	0
IR0069	21	2577	2577
IR0074	10	1187	1187
IR0075	120	13936	13936
IR0084	18	0	0
IR0089	8	957	957
IR0090	9	1088	1088
IR0133	146	15225	15225
IR0715	23	0	0
IR0721	48	5580	5580
IR0726	10	1211	1211
SR0035	8	762	762
SR0036	1	95	95
SR0037	8	762	762
SR0040	10	953	0
SR0059	2	207	207
SR0225	1	100	100
SR0701	11	1069	1069
SR0702	12	1164	1164
SR0707	5	477	477
SR0732	16	1525	0
SR0771	34	3240	3240

Table C-4: continued.

Route	*Number of Sorties*	*Low Altitude Noise*	*Low Altitude Night Noise*
SR0774	5	477	0
SR0801	13	1311	0
SR0823	5	465	465
SR0824	5	503	503
SR0825	4	408	408
SR0826	6	599	599
SR0845	5	1037	1037
SR0847	10	1037	1037
SR0873	18	1715	0
SR0900	131	14213	14213
SR0901	15	1430	1430
SR0902	4	381	381
SR0904	8	762	762
VR0058	23	2790	2790
VR0086	7	725	725
VR0087	94	10343	10343
VR0088	200	21734	21734
VR0092	23	2790	2790
VR0093	3	351	351
VR0095	53	6352	6352
VR0704	46	5481	0
VR0705	67	7966	0
VR0707	24	2927	0
VR1016	6	664	0

Table C-4: continued.

Route	*Number of Sorties*	*Low Altitude Noise*	*Low Altitude Night Noise*
VR1023	6	5112	5112
VR1050	17	2024	2024
VR1051	22	2171	2171
VR1052	36	3182	3182
VR1055	50	5809	5809
VR1056	36	3182	3182
VR1059	78	8477	8477
VR1060	33	3610	3610
VR1064	28	3436	3436
VR1068	16	1574	1574
VR1145	61	5813	0
VR1146	19	1811	0
VR1653	28	3436	3436
VR1721	2	272	272
VR1751	3	3235	3235
VR1752	208	24329	24329

Table C-5: Triple Product of Population x Flight Activity x Aircraft Noisiness for MTRs in 10 Units of $\log_{10}$.

Route	*Total*	*Week-ends*	*Night-time*	*Low Alti-tude Night*	*Low Altitude*	*Dupli-cated*	*Low Alti-tude Dup.*
IR0023	59.52	59.52	59.52	80.26	80.26	0	0
IR0042	62.39	62.39	62.39	83.23	83.23	0	0
IR0062	71.45	71.45	71.45	0	0	52.73	0
IR0069	66.77	66.77	66.77	87.66	87.66	57.87	78.76
IR0074	63.12	63.12	63.12	81.25	81.25	0	0
IR0075	76.49	76.49	76.49	95.92	95.92	67.23	87.85
IR0084	64.75	64.75	64.75	0	0	0	0
IR0089	61.45	61.45	61.45	79.67	79.67	0	0
IR0090	61.95	61.95	61.95	80.21	80.21	0	0
IR0133	78.14	78.14	78.14	98.32	98.32	24.65	44.84
IR0715	67.79	67.79	67.79	0	0	0	0
IR0721	70.94	70.94	70.94	89	89.22	0	0
IR0726	63.10	63.10	63.10	93.94	83.93	27.32	48.16
SR0035	60.50	60.50	60.50	80.29	80.29	51.05	70.85
SR0036	53.94	53.94	53.94	73.73	73.73	39.49	49.28
SR0037	62.02	62.02	62.02	81.81	81.81	51.05	70.85
SR0040	61.65	61.65	61.65	0	81.44	0	0
SR0059	54.61	54.61	54.61	74.77	74.77	0	0
SR0225	52.56	52.56	52.56	72.56	72.56	0	0
SR0701	62.92	62.92	62.92	82.79	82.79	0	0
SR0702	63.09	63.09	63.09	82.96	82.96	31.28	51.15
SR0707	58.68	58.68	58.68	78.47	78.47	0	0
SR0732	63.22	63.22	63.22	0	83.01	0	0
SR0771	67.64	67.64	67.64	87.43	87.43	0	0
IR0023	59.52	59.52	59.52	0	78.04	53.07	72.86

Table C-5: continued.

Route	*Total*	*Week-ends*	*Night-time*	*Low Alti-tude Night*	*Low Altitude*	*Dupli-cated*	*Low Alti-tude Dup.*
SR0774	58.25	58.25	58.25	0	78.04	53.07	72.86
SR0801	63.38	63.38	63.38	0	83.41	31.63	51.67
SR0823	60.48	60.48	60.48	80.16	80.16	0	0
SR0824	59.13	59.13	59.13	79.16	79.16	0	0
SR0825	59.33	59.33	59.33	79.41	79.41	0	0
SR0826	63.45	63.45	63.45	83.44	83.44	51.12	71.11
SR0845	62.22	62.22	62.22	82.37	82.37	0	0
SR0847	62.66	62.66	62.66	82.82	82.82	0	0
SR0873	65.01	65.01	65.01	0	84.80	54.4	74.19
SR0900	79.12	79.12	79.12	99.47	99.47	0	0
SR0901	66.20	66.20	66.20	85.99	85.99	58.07	77.86
SR0902	60.85	60.85	60.85	80.64	80.64	0	0
SR0904	65.11	65.11	65.11	84.90	84.90	0	0
VR0058	68.15	68.15	68.15	88.99	88.99	0	0
VR0086	60.65	60.65	60.65	80.80	80.80	0	0
VR0087	74.61	74.61	74.61	95.02	95.02	0	0
VR0088	75.22	75.22	75.22	95.58	95.58	50.2	70.56
VR0092	68.15	68.15	68.15	88.99	88.99	0	0
VR0093	60.92	60.92	60.92	81.60	81.60	23.1	43.78
VR0095	70.85	70.85	70.85	91.63	91.63	0	0
VR0704	68.65	68.65	0	89.41	89.41	0	0
VR0705	69.65	69.65	0	90.40	90.40	0	0
VR0707	65.76	65.76	0	86.45	86.45	0	0
VR1016	59.85	59.85	0	80.29	80.29	0	0

Table C-5: continued.

Route	*Total*	*Week-ends*	*Night-time*	*Low Alti-tude Night*	*Low Altitude*	*Dupli-cated*	*Low Alti-tude Dup.*
VR1023	59.13	0	59.13	88.44	88.44	44.88	74.18
VR1050	64.14	64.14	64.14	84.65	84.65	0	0
VR1051	66.02	66.02	66.02	85.96	85.96	0	0
VR1052	68.84	68.84	68.84	88.30	88.30	0	0
VR1055	68.72	68.72	68.72	89.37	89.37	0	0
VR1056	68.84	68.84	68.84	88.30	88.30	0	0
VR1059	71.25	71.25	71.25	91.61	91.61	0	0
VR1060	66.92	66.92	66.92	87.31	87.31	0	0
VR1064	68.64	68.64	68.64	89.52	89.52	0	0
VR1068	63.29	63.29	63.29	83.22	83.22	0	0
VR1145	69.13	69.13	0	88.92	88.92	0	0
VR1146	64.06	64.06	0	83.85	83.85	0	0
VR1653	68.64	68.64	68.64	89.52	89.52	0	0
VR1721	54.72	54.72	54.72	76.07	76.07	0	0
VR1751	68.56	68.56	68.56	89.19	89.19	0	0
VR1752	76.06	76.06	76.06	96.74	96.74	36.60	57.29

Table C-6: Rankings of MOAs by Population.

MOA Name	*Non-Rural*	*Weekend*	*Nighttime*
BIRMINGHAM 2	17	4	8
BISON	16	14	14
BRADY LOW	8	33	19
BRUSH CREEK	23	2	6
BULLDOG A	15	33	7
COMPLEX 1	11	6	10
EGLIN B	6	33	15
EGLIN E	1	33	1
EUREKA LOW	19	16	35
FARMVILLE	7	33	4
FREMONT	5	33	18
GAMECOCK C	14	5	9
GAMECOCK I	9	33	11
HOTROCK 2	12	3	35
JENA 1	13	7	35
MORENCI	10	33	17
QUICK THRUST E	21	33	35
QUICK THRUST F	24	33	35
QUICK THRUST I	3	33	35
SNOWBIRD 2	4	33	3
SYRACUSE 2	22	17	35
SYRACUSE 3	20	21	35
TYNDALL F	18	33	24
WILLIAMS 1	2	33	2

Table C-7: Rankings of MOAs by Flight Activity and Product of Activity and Aircraft Noisiness.

MOA Name	*Number of Sorties*	*Low Altitude Noise*	*Low Altitude Night Noise*
BIRMINGHAM 2	19	15	15
BISON	17	36	36
BRADY LOW	--	--	--
BRUSH CREEK	12	11	11
BULLDOG A	7	6	6
COMPLEX 1	1	1	1
EGLIN B	35	36	36
EGLIN E	41	34	34
EUREKA LOW	--	36	36
FARMVILLE	26	22	22
FREMONT	--	36	36
GAMECOCK C	11	10	10
GAMECOCK I	37	30	30
HOTROCK 2	10	8	8
JENA 1	18	17	17
MORENCI	14	36	36
QUICK THRUST E	15	12	12
QUICK THRUST F	15	12	12
QUICK THRUST I	15	12	12
SNOWBIRD 2	--	36	36
SYRACUSE 2	5	4	4
SYRACUSE 3	8	9	9
TYNDALL F	13	36	36
WILLIAMS 1	--	36	36

Tables C-8, C-9, and C-10 show the same columns, with computed values entered rather than rankings. Columns in Table C-10 follow those of Table 6-2 in Section 6.1.2.

C.2 Accuracy and Completeness of ORNL Database Information

The ORNL database contains numerous minor discrepancies which may be attributable to changes subsequent to its publication. Some examples of these include:

- The Air Force magazine referred to as the source for scheduling commands lists Elmendorf AFB as a TAC and MAC base, while the database lists that facility as an AAC base. Several other such discrepant listings were found.
- Incomplete data in the aircraft file for MTRs (ROUTECRA.DBF) includes missing noise characteristics from three of the aircraft encountered in candidate MTRs: F4, F14, and B52. One route, VR1726, show no aircraft at all.
- The ROUTE.DBF file shows a total population under MTR IR0133 of about 450,000; however, that route crosses the desert, with no towns nearby. For a few MOAs (Marian, Moody 2A, Salem) the MOA.DBF shows the entire population to be rural (defined as less than 500 people per square mile), while charts show sizable towns in these areas.
- On the basis of the ranking process, SR0900 was selected selected as the highest ranked route on the basis of 131 reported overflights per month in a highly populated locale. Verification with the current airspace manager revealed an average of only 13 flights per month. (It is not clear whether this is an error or a change in level of operations.)

Several inconsistencies in the Restricted Area (RA) files led to abandonment of the attempt to rank the areas:

- For a number of RAs the RACRAFT.DBF file does not show the total number of flights and for some (e.g., R-5314A and R-5314B) the files list more than one total.
- There are blank entries in the ENDTIME field (indicting whether nighttime flights are permitted) for some of the areas (e.g., R-32002E, R-3202A-D, R-5314B-J).
- For many RAs, type of aircraft is listed as OTHER, for which no sound exposures can be estimated. Several types of aircraft are listed in the RACRAFT.DBF file for which there is no sound exposure data, such as helicopters and aircraft types H53 and O2.

Spot checks of the ROUTE (MTR), MOA, and RESTAIR (RA) major files revealed the following:

- Approximately 10% (75) of the ROUTE.DBF entries were checked against AP/1B

Table C-8: MOA Population Values.

MOA Name	*Non-Rural*	*Weekend*	*Nighttime*
BIRMINGHAM 2	1815	33884	33884
BISON	1893	17884	17884
BRADY LOW	4657	0	12423
BRUSH CREEK	1193	43090	43090
BULLDOG A	1931	0	40470
COMPLEX 1	4098	29613	29613
EGLIN B	7578	0	17331
EGLIN E	72753	0	98414
EUREKA LOW	1595	12863	0
FARMVILLE	5656	0	63069
FREMONT	7813	0	12943
GAMECOCK C	2157	32245	32245
GAMECOCK I	4251	0	25729
HOTROCK 2	3688	42488	0
JENA 1	3416	27648	0
MORENCI	4176	0	13601
QUICK THRUST E	1320	0	0
QUICK THRUST I	11778	0	0
SNOWBIRD 2	11491	0	69952
SYRACUSE 2	1278	11813	0
SYRACUSE 3	1348	8136	0
TYNDALL F	1647	0	7584
WILLIAMS 1	39142	0	78622

Table C-9: MOA Flight Activity and Product of Activity x Aircraft Noisiness.

MOA Name	*Number of Sorties*	*Low Altitude Noise*	*Low Altitude Night Noise*
BIRMINGHAM 2	176	20257	20257
BISON	200	0	0
BRADY LOW	--	--	--
BRUSH CREEK	256	31436	31436
BULLDOG A	388	41587	41587
COMPLEX 1	3982	463554	463554
EGLIN B	52	0	0
EGLIN E	3	369	369
EUREKA LOW	--	0	0
FARMVILLE	96	10426	10426
FREMONT	--	0	0
GAMECOCK C	294	31700	31700
GAMECOCK I	46	4977	4977
HOTROCK 2	303	32807	0
JENA 1	177	19173	0
MORENCI	218	0	0
QUICK THRUST E	211	23956	0
QUICK THRUST I	211	23956	0
SNOWBIRD 2	--	0	0
SYRACUSE 2	600	64470	0
SYRACUSE 3	350	32385	0
TYNDALL F	223	0	0
WILLIAMS 1	--	0	0

Table C-10: Triple Product of Population x Flight Activity x Aircraft Noisiness for MOAs in Units of 10 $\log_{10}$.

MOA Name	*Non-Rural*	*Week-end*	*Night-time*	*Low Altitude*	*Low Altitude Night*
Birmingham 2	55.04	67.76	67.76	75.65	75.65
Bison	55.78	65.53	65.53	0	0
Brady Low	--	0	--	--	--
Brush Creek	54.85	70.43	70.43	75.74	75.74
Bulldog A	58.75	0	71.96	79.05	79.05
Complex 1	72.13	80.41	80.72	92.79	92.55
Eglin B	55.96	0	59.55	0	0
Eglin E	53.39	0	54.7	74.29	74.29
Eureka Low	--	0	--	--	--
Farmville	57.35	0	77.82	74.29	77.71
Fremont	--	0	--	0	0
Gamecock C	58.02	69.77	69.77	78.35	78.35
Gamecock I	52.91	0	60.73	73.25	73.25
Hotrock 2	60.48	71.1	0	80.83	0
Jena 1	57.81	66.90	0	78.16	0
Morenci	59.59	0	64.72	0	0
Quick Thrust E	54.45	0	0	75.00	0
Quick Thrust I	63.95	0	0	84.50	0
Snowbird 2	--	0	--	0	0
Syracuse 2	58.85	68.51	0	79.16	0
Syracuse 3	56.74	64.54	0	76.4	0
Tyndall F	55.65	0	62.28	0	0
Williams 1	--	0	--	0	0

(12/18/86), of which 5 contained errors. These errors include: failure to list minimum altitude (3 routes); and use of the city as the scheduling agency, rather than the actual agency (2 routes).

- Approximately 35% (40) of the MOA.DBF entries were checked against the "Chapter 10" document (DMA, 1987), revealing 6 errors. For 3 areas, the scheduling unit is different. The memo field listing altitude exceptions is inaccurate in at least one area. In a final route, minimum altitude is different.
- Approximately 25% (40) of the RESTAIR.DBF listings were checked against AP/1A (10/23/86), of which six show errors. In one case, there is no listing of maximum altitude or use times. Two entries show difficulties with memo fields; one does not match AP/1A and the other is missing a memo field.