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EFFECTS OF NOISE ON WILDLIFE AND OTHER ANIMALS

Memphis State University
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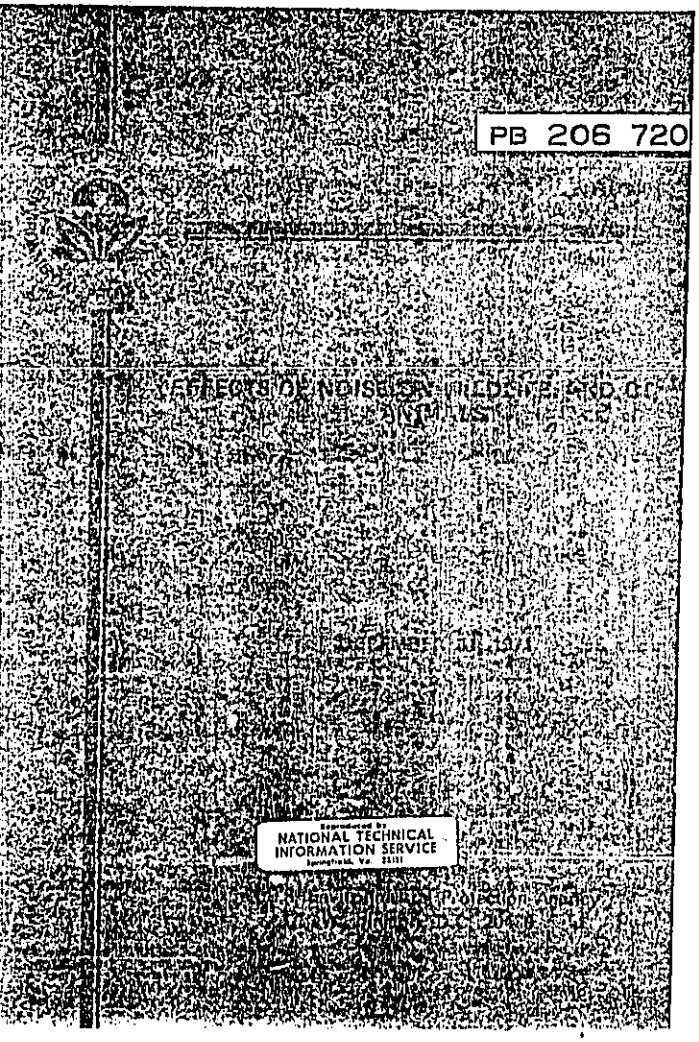

EFFECTS OF NOISE ON WILDLIFE AND OTHER ANIMALS

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INTRODUCTION

For many years before any real scientific information was available, we have known that prolonged exposure to high intensities of noise could cause loss of hearing in humans. "Boilermakers" or "artillerymen's" ears have been known to be defective with the cause of the deficit known for over a hundred years. The effects of sound upon man's hearing are well documented. In the last few years there have been studies suggesting a large and potentially frightening number of non-auditory effects of noise on man; consequently, today there are many investigators considering possible non-auditory effects of sound on man and trying to either demonstrate or disprove them.

In recent years the possible effects of noise on wildlife have become a matter of serious concern, for several excellent reasons. Our rapidly growing population and advancing technology result in ever increasing noise levels. Noise is an unwanted and at times a potentially dangerous by-product of virtually every aspect of modern-day life--construction, transportation, power generation, manufacturing, recreation, etc. Today we find that areas previously considered remote, and therefore relatively non-polluted by noise, are now being exposed or are in danger of exposure to various kinds of noise pollution. The effects that increased noise levels will have on wildlife in these areas are virtually unknown. Obviously animals that

rely on their auditory systems for courtship and mating behavior, prey location, predator detection, homing, etc., will be more threatened by increased noise than will species that utilize other sensory modalities. However, due to the complex interrelationships that exist among all the organisms in an ecosystem, interference with one species might well affect all the other species.

In the past, man's tampering with the balance of nature frequently has proved to have serious consequences for both man and the ecosystem; whatever affects the ecosystem, eventually affects man also. Noise pollution conceivably could disrupt a balanced ecosystem and possibly even contribute to the extinction of a vulnerable species. Many species of wildlife today are endangered. Apart from the threat of the irretrievable loss of a particular species, we have no certain knowledge regarding the possible effects on our ecology from such a loss. To prevent possible irreparable damage to wildlife and to the balance of nature, it is mandatory that we calculate the expected increases in noise levels and try to relate them to their possible impact on our wildlife.

It has become apparent that there is a serious lack of information concerning effects of noise on wildlife. Because of the high likelihood that noise effects on domestic or laboratory animals can provide clues regarding possible effects on wild animals, a summary of the literature concerned with the effects of noise on non-wild animals is

also included, although it is not as exhaustive nor as detailed as it would be if that were the mission of this report.

For the purpose of this report "wildlife" is defined as those animals which were not born or hatched in captivity. The literature search here reported was concentrated on the period from 1950 to the present, but earlier pertinent studies are also reviewed. It was not possible to search the foreign literature thoroughly in the limited time available. Therefore, only clearly relevant and readily obtainable reports from foreign literature are included. A detailed report of libraries, information retrieval services, source materials, and persons and agencies contacted for information is presented in the Appendix.

Effects of Noise on Laboratory Animals

To determine what noise does to an organism, it is important to know:

(1) What sounds an animal is exposed to (e.g., frequency, spectra, intensity, duration, and pattern of exposure);

(2) What factors determine an animal's susceptibility to noise-induced damage (e.g. species, age, audibility range, recovery process, etc.) These factors are best investigated in laboratory experiments using animals, because in laboratory experiments each of these parameters can be controlled and manipulated to determine the relationships between noise

exposure and effects on the animal. Experiments investigating the effects of exposure to noise can be classified in two basic categories: (1) studies of effects on the auditory system, and (2) studies of non-auditory effects.

Effects of Noise on the Auditory System

As with man, the best documented effect of noise on laboratory animals is the production of hearing loss or damage to the auditory system. This can be produced by a brief exposure to very loud sound or by prolonged exposure to moderate levels of sound.

To study hearing loss it is necessary to measure hearing abilities before and after exposure to noise. A major problem in studying auditory effects of noise on animals is the determination of what sounds the animal "hears." Either electrophysiological recordings from the auditory system, or behavioral responses of the animal can be used to assess the sensitivity of the ear. The Preyer reflex, an ear-twitch response to sound, indicates that an animal has heard a sound. This reflex is a reliable, but not a very sensitive test of hearing in animals, because they are capable of hearing sounds that are less intense than the sounds that produce the response. An animal can be trained to respond to a sound stimulus by using the sound as a cue to obtain reward (e.g., food) or to escape from punishment (e.g., electric shock). If the animal is appropriately motivated (i.e., hungry or fearful of shock, depending on the circumstances), his responses can serve as

a very sensitive indicator of what he is hearing. Auditory thresholds in animals are frequently determined by using a "conditioned avoidance response;" the animal is trained to avoid shock by moving from one side of a two-chambered cage to the other. If the animal is well-trained, this procedure can provide a very sensitive measure of his ability to detect tones of known frequency and intensity. An animal's hearing can be tested, the animal then can be exposed to noise, and hearing can be retested to determine the decrease in hearing ability.

Impulse noise is sound which rises very quickly to its maximum intensity; it has a very fast rise time, on the order of a few micro-seconds (i.e., a few millionths of a second). If sufficiently intense, the rapid pressure changes produced by impulse sound can damage the ear by rupturing the ear-drum, by disrupting the chain of tiny bones in the middle ear, or by damaging the sensory cells and other structures in the inner ear. Pecho, Stockwell, and Ades (1969) studied histologic changes in 14 young guinea pigs cochleas following exposure to impulse sound. Five hundred rounds of paper caps, producing an average sound-pressure-level (SPL) of 153 dB, were fired 30 cm from the ear. The noises were 1 to 5-sec apart over a 45-min period. In 11 of the ears, the sensory hair cells were destroyed in a narrow band midway along the organ of Corti. This damage was comparable to histologic changes produced by exposure for 4 hr to a 2,000-Hz tone at a SPL of 125 to 130 dB. Majeau-Chargois, Berlin, and Whitehouse

(1970) studied damage produced by simulated sonic booms in 24 guinea pigs. They determined the animals' hearing abilities by testing for the Preyer reflex over a range of frequencies from 125 to 16,000 Hz. The guinea pigs were individually exposed to simulated sonic booms having durations of either 2.00, 4.76, or 125.00 milliseconds (each animal was exposed to only one of these durations); 1,000 booms were produced, at a rate of one per second. The intensity of each boom was reported as approximately 130 dB, but the reference level was not stated. Tests of the Preyer reflex following exposure to the booms failed to detect any changes in hearing ability in the guinea pigs, although microscopic examinations of their cochleas revealed losses of approximately 10% of the hair cells in the first turn. This was amazingly little damage, considering that each animal was exposed to 1,000 booms at the rate of one per second.

Because of the very brief durations of impulse sounds, they are described in terms of rise time, maximum intensity (peak pressure level), and duration. Sounds having a longer duration can, in addition, be described by their frequency spectrum. A description of the frequency spectrum provides very useful information because man and other animals are not equally sensitive to all frequencies. Sounds with different frequency spectra have different effects on the auditory system. High frequency pure tones or narrow bands of noise tend to produce changes in localized regions of the inner ear, whereas low frequency tones, and random or

broad-band noise tend to produce changes throughout the length of the cochlea.

In a study of exposure to pure tone, Beagley (1965a, 1965b) exposed 20 guinea pigs to a 500-Hz tone at a SPL of 138 dB. Following exposure for 20 min, there was a decrease in the amplitude of cochlearmicrophonic potentials recorded from the inner ear, indicating that the ear was less sensitive to sound. Also, histological studies revealed extensive damage to sensory cells and supporting structures in the third turn of the cochlea, with little or no damage in the fourth turn. In studies involving 20 guinea pigs, Conti and Borgo (1964) found that exposure for 3 hr at a SPL of 100 dB to frequencies of 250, 2,000, 4,000, or 8,000 Hz produced consistent metabolic changes in the inner ear. Reduction in the activity of the enzyme cytochrome oxidase was detectable in several different structures of the inner ear; this reduction was not related to the frequency of the stimulating noise.

Dogs and guinea pigs were used as experimental animals by Covell (1953) in a study of the histologic changes in the organ of Corti following exposure to intense sound. He exposed 132 guinea pigs and 7 dogs to 50,000 to 100,000-Hz sound. Essentially, Covell found marked histologic changes in the organ of Corti following exposure to intense sound, indicative of a loss of hearing in the animals.

In some preliminary studies of temporary threshold shift (a temporary elevation of the level of lowest intensity

sound that can be heard) in chinchillas, Peters (1965) determined that temporary threshold shift (TTS) increased with increased duration of exposure to an octave band of noise (2,000-4,000 Hz) at 70, 80 or 90 dB (the reference level was not reported). In an experiment to determine the electrophysiological correlates of temporary threshold shifts, Benitez, Eldredge, and Templer (1970) exposed chinchillas for 48-72 hr to an octave band of noise centered at 500 Hz with a SPL of 95 dB. This exposure produced a behavioral TTS of about 48 dB in the animals, with recovery requiring about 5 days. Changes in cochlear microphonics recorded from the second turn corresponded closely to behavioral TTSs; however, losses of sensitivity in activity recorded from the auditory nerve were much greater than losses in behavioral responses. Using an octave band (300-600 Hz) of thermal noise at a SPL of 100 dB, Miller, Rothenberg, and Eldredge (in press) obtained maximum TTSs of 50 dB or more during 7 days of exposure. Recovery from these TTSs required about 5 days, with signs of permanent threshold shifts of less than 10 dB at certain test frequencies. Cochlear potentials were reduced and hair cells were lost in the second and third cochlear turns.

Broad-band noise has also been used to study hearing loss and damage to the auditory system. Lawrence and Yantis (1957) stimulated guinea pigs with white noise; sound pressure levels, measured at the tympanic membrane, were 150 dB for one group of guinea pigs and 130 dB for a second group.

Recordings from the round window indicated that a 20-min exposure produced some permanent loss in sensitivity in both groups. Miller, Watson, and Covell (1963) exposed 37 cats to broad-band noise having nearly equal sound-pressure-levels across octave bands centered at 850, 1,700, and 3,400 Hz. Exposures to a SPL of 115 dB for one-eighth of an hour or 105 dB for one-fourth of an hour produced maximum TTS at 4,000 Hz. Exposure to uninterrupted noise at a SPL of 115 dB for 15 min to 8 hr produced mean permanent threshold shifts ranging from 5.6 dB (for 15 min) to 40.6 dB (for 8 hr). Breaking up the total exposure into small doses resulted in increasingly less permanent loss as the interval between doses increased; a total of 8 hr of exposure having 24-hr intervals between sixteen 7 1/2-min doses produced a permanent threshold shift of only 2 dB. The correlation between amounts of permanent threshold shift and cochlear injury was 0.85. Ward and Nelson (1970) also studied the effects of intermittent noise on hearing. Two groups of four monaural chinchillas (i.e., animals with one ear destroyed) were exposed for 2 hr to a 700 to 3,000-Hz band of noise at a SPL of 117 dB. One group was exposed continuously, the other had eight 15-min exposures separated by intervals of 45 min of quiet. Both exposures produced initial threshold shifts of more than 100 dB, but the animals exposed intermittently had completely recovered within 2 weeks whereas the animals exposed continuously had losses of 40 dB 3 months after exposure.

Twenty guinea pigs were exposed to rocket booster engine noise by Gonzalez, Miller, and Istre (1970). Four groups of five animals each were located at distances from the noise source of 75, 150, 300, and 5,000 ft respectively. For the three closer positions, sound-pressure-levels were above 110 dB from 8 to 8,000 Hz, with peaks near 140 dB between 8 and 31.5 Hz. Peak pressure levels at the fourth position were near 110 dB between 16 and 31.5 Hz and dropped off rapidly in the higher frequencies. Following 5 min, 50.1 sec of exposure to the rocket engine noise, Preyer reflex thresholds indicated almost complete loss of hearing in the two closer groups, up to 57 days post-exposure; there were only slight temporary losses in the third group and no measurable effect in the most distant group.

Ishii, Takahashi, and Balogh (1969) reported that exposure for 30 min to white noise at a SPL of 110 dB produced reductions in the number of glycogen granules in guinea pigs' ears. They suggested that glycogen serves as an energy source in the hair cells.

The extent of noise-induced hearing loss or damage to the auditory system depends upon intensity, spectrum, duration, pattern of exposure and individual susceptibility. Rest intervals interpolated in exposure periods can significantly reduce the amount of damage.

Non-auditory Effects of Noise

Only recently have non-auditory effects of noise

become a matter of concern, due to suggestions that noise may act as a physiological stressor producing changes similar to those brought about by exposure to extreme heat, cold, pain, etc. There is a considerable body of literature concerning physiologic response to stress and now there is also some evidence that exposure to noise may induce similar changes. The general pattern of response to stress includes neural and endocrine activation bringing about a variety of measurable changes, such as increases in blood pressure, available glucose, blood levels of corticosteroids, and changes in the adrenal glands. There is evidence that prolonged exposure to severe stress can exhaust an organism's resources and result in death. On the other hand, an animal raised under conditions that protect it from stress becomes extremely susceptible to disease or even death under even mildly stressful situations. The actual significance for an animal of the physiologic responses to stress is not understood.

In an early study, Yeakel, Shenkin, Rothballer, and McCann (1948) exposed adrenalectomized Norway rats to the sound of a blast of compressed air 5 min a day, 5 days a week, for a year. The average systolic pressure in the noise exposed rats rose from an initial value of 113 mm Hg to 154 mm Hg in the last 2 months, while control values rose from 124 to 127 mm Hg. More recently (Osinstseva, Pushkina, Bonashevskaya, and Kaverina, 1969), rats were exposed to an 80 dB noise for various times from 18 to 126 days. Following

exposure to noise, analyses revealed significant drops in ascorbic acid contents and weights of the adrenals of these rats relative to controls. Adreno-cortical activation has been studied quite extensively in rodents by Anthony and Ackerman (1955, 1957) and by Anthony, Ackerman, and Lloyd (1959). They exposed rats, mice, and guinea pigs to relatively broad bands of intense noise: 150-4800 Hz at 140 dB SPL, 10,000-20,000 Hz at 110 dB SPL, or 2,000-40,000 Hz at 132 dB SPL. Durations of stimulation periods included a single 6-min exposure, 15 min or 45 min per day for up to 12 weeks, and cycles of 100 min on and 100 min off throughout a 4-week exposure period. Although they obtained indications of adrenal activation, as measured by cellular changes in the adrenal glands and a decrease in the number of circulating eosinophils, these changes were generally slight and transient. They did find, however, that intense noise superimposed on another stress, such as restriction of food, could decrease an animal's life span. The authors concluded that rats, mice, and guinea pigs can successfully adapt to noise, but that noise can have damaging effects if it occurs in conjunction with additional stressful situations. They also noted that intense high frequency noise (132 dB SPL, 2,000-40,000 Hz) appears to be more stressful than low frequency noise as evidenced by an increase in noise-induced seizures in mouse strains considered to be seizure-resistant (Anthony and Ackerman, 1957). Jurtshuk, Weltman, and Sackler (1959) subjected two groups of Wistar albino female rats daily to

1 min of noise for 11 days and to 5 min of noise for 15 days, respectively. The noise consisted of 120 Hz at 100 ($\pm$5) dB SPL. Rats that displayed the greatest locomotor response upon cessation of auditory stimulation also had lowest blood glutathione levels. Stimulated rats had higher adrenal weights and ascorbic acid values and lower blood glutathione levels than did their controls. Gober, Anderson, and Van Dyne (1966) investigated the physiologic response of rats to three durations of acoustic stress (15-270 min, 29-96 hr, and 21 days). The stimulus was a 73 to 93-dB SPL 20,000 to 25,000-Hz sound presented 6 min of every hour. They noted lower eosinophil counts, raised serum cholesterol levels and increased ascorbic acid levels in the brain. Although Treptow (1966) stated that dogs had transitory increases in glycemic levels in the blood prior to becoming used to experimenter handling, he did find a predictable increase in glycemic reactions in trials 1 and 8 out of 20 exposures to 80-87 dB noise for 5-10 min. Due to individual reactivities, the measures were highly variable, but by trial 20 the dogs had apparently adapted to the noise stimulus.

Biochemical changes due to noise exposure were studied by Elbowicz-Wariowska (1962). Guinea pigs were exposed for 1 month to daily 45-min periods of noise at 100 ($\pm$5) dB SPL with frequencies from 100 to 50,000 Hz. Increases in lactic acid dehydrogenase activity and pyruvic acid levels in the blood were observed. Hrubes (1964) found that non-esterified

fatty acids, the plasma lipid most implicated in active transport within cells, increased significantly in female white rats when the rats were exposed to a 95 dB transmitter generator noise for 16 hr. Hruben and Bones (1965) demonstrated that white rats subjected repeatedly to 95 dB noise developed increased uremic catecholamines, increased free fatty acids in blood plasma, and increased suprarenal size. Further, exposed animals showed characteristic weight decreases. Friedman, Byers, and Brown (1967) demonstrated that auditory stimulation can interfere with lipid metabolism. White noise at a SPL of 102 dB was presented 24 hr a day and an additional intermittent 200-Hz square wave with a duration of 1 sec and a SPL of 114 dB was programmed to occur at random intervals, with an average interval of 3 min. Thirty rats were exposed to the noise stimuli for 3 weeks and 24 rabbits were exposed for 10 weeks. These animals received standard diets and water, but were administered additional oils to test their abilities to handle excess fat while exposed to noise stress. Plasma triglycerides were higher in sound-exposed rats only during the second week; there were no differences between experimental and control groups of rats at the end of weeks 1 and 3. In the rabbits, however, plasma cholesterol and fasting plasma triglycerides were higher after 4 weeks of auditory stimulation. Additional differences between sound-stressed rabbits and their controls included deposits of fat in the irises of the eyes of the experimental rabbits, plus more aortic atherosclerosis and

higher cholesterol content in their aortas. The authors concluded that auditory stress produces changes in handling exogenously delivered fat, having effects similar to those produced by chronic hypothalamic stimulation.

There is additional evidence that sound stimulation produces its observed effects via cortico-hypothalamic interactions with the hypophyseal adrenal system. Werner (1959) studied the effect of sound on the hypophysis of the rat. He found that long, continuous bell ringing (8 hr per day) for from 1 day to 3 weeks resulted in hypertrophy in the pars intermedia and hyperactivity in the adrenal cortex. Ogle and Lockett (1966) studied the effect in rats of recorded thunderclaps of 3 to 4-sec duration with a frequency range of 50-200 Hz at 98-100 dB SPL, presented at a rate of two claps at 1-min intervals every 5 min for 20 min. They compared this effect with that from a pure tone of 150 Hz at 100 dB presented for 2 min out of every 15 min for 45 min. Urine was collected and analyzed for sodium and potassium. Responses to noise were analyzed through comparisons among animals that were intact, that had denervated kidneys and that had neurohypophyseal lesions. The authors concluded that thunderclaps produced emotional responses which the 150-Hz tone did not produce. Thunderclaps affected the hypothalamus resulting in excretion of oxytocin and vasopressin; these hormones produced increases in sodium and potassium excretion with no increase in urine flow.

In a recent study (Hiroshige, Sato, Ohta, and Itoh,

1969), rats were exposed to bell-ringing for 2 min (spectrum and noise level were not reported). Bell-ringing produced an increase in the activity of corticotropin-releasing factor (CRF) in the hypothalamus. CRF produces the release of adrenocorticotrophic hormone (ACTH) from the pituitary, and ACTH in turn produces the release of corticosteroids from the adrenals. Monastyrskaya, Prakh'e, and Khaunina (1969) reported that sound stimulation produced increases in weights of the pituitary and adrenal glands in healthy rats, but not in a strain of sound-sensitive, audiogenic-seizure susceptible rats. The sound-sensitive rats already had enlarged pituitaries and adrenals. The rats were exposed to a 105 dB sound 10 times, for 1.5 min each time, with one exposure every 3 to 4 days. The frequency characteristics and noise reference levels were not reported. Activity of acetylcholine throughout the rat brain was studied by Mrozinska (1968). Exposure to noise (type and level not reported) for 2 hr a day for 3, 6, 9, 12, or 15 days produced gradual increases in acetylcholine esterase activity, and an initial increase in acetylcholine concentration followed by a decrease with a slow return to normal levels by 15 exposures.

In addition to the pituitary and adrenal glands, the reproductive glands and functions are also affected by exposure to noise. The results are not always consistent, however. Anthony and Harclerode (1959) reported negative results in a study of the effects of noise on sexual scores of sexually mature male guinea pigs. Twelve weeks of daily

exposure, for 20 min out of each 30 min period, to noise at a SPL of 130-144 dB with frequencies of 300 to 4,800 Hz did not affect the sexual scores of the experimental animals relative to their controls. Some evidence of cortico-adrenal activation was found, however, suggesting that tolerance limits were approached. Zoric (1959) exposed 38 male mice for 8 hr per day for 1-21 days to the sound of an electric bell. The level and spectrum of the sound were not reported. Studies of the testes of sound-exposed mice revealed involution of the seminal epithelium, partial blockage of first order spermatocytes, formation of teratocytes, and atrophy of the epithelium. He also observed that the glandular interstitial cells were characterized by hypertrophy and hyperplasia. Zondek and Isachar (1964) examined the effect of acoustic stimulation on genital function in 48 mature rabbits and 3,100 young and mature rats. The animals were housed "near" an electric bell 25 cm in diameter that rang 1 min out of every 10 min, 24 hr per day, for 9 days prior to mating. The peak SPL was 100 dB, with maximum energy at 4,000 Hz, and another peak of 95 dB at 10,000 Hz. Auditory stress resulted in enlargement of the ovaries, persistent estrus, follicle haematomata, and other effects in female rats and rabbits. Effects were more pronounced in female rabbits than in female rats and were hardly visible in males of either type. Auditory stress during the copulatory period induced increased fertility, but during gestation such stress produced a blockage of pregnancy.

However, Zondek (1964) reported that in rats the males' as well as the females' fertilities were decreased. The males' ability to fertilize was reduced to 11% as compared to 70-80% in control males; comparable effects were produced in the female rats. Sexual behavior did not seem to be inhibited (copulation was verified by the presence of a vaginal plug), and there were no changes in the weights of the testes and seminal vesicles, nor any noticeable anatomical changes in the spermatogenic process. In similar fashion, Singh and Rao (1970) studied the effects of auditory stress on rat ovaries. They exposed 74 adult female rats to continuous auditory stimulation by a 2,000-Hz tone at 100 dB C for up to 150 days. They found that 31 animals developed persistent vaginal estrus after 10 consecutive days of stress. As the stress was continued, more and more animals demonstrated the condition.

There is evidence that sound stimulation may induce lasting changes in exposed animals and even in their offspring, at least in strains of mice that have been specially bred to be susceptible to audiogenic seizures. Lindzey (1951) studied emotionality and audiogenic seizure susceptibility in mice exposed to noise. The animals were stimulated by the sound from a bell attached to a metal washtub (spectrum and SPL were not described). He reported increased susceptibility to seizure in certain strains of mice. Thompson and Sontag (1956) described effects of audiogenic seizures in pregnant rats on maze-learning abilities of

their offspring. Each of six male albino rats was bred to one experimental and one control female. Two seizures per day were induced from the fifth through the eighteenth day of pregnancy in each of the six experimental females. Within 24 hr of birth two male and two female pups were selected from each litter and the rest were removed. Three mothers in the experimental group and three in the control group kept their own pups, while the pups of the other three mothers in each group were switched between groups so that pups from experimental (seizure) mothers were cross-fostered on control mothers and vice versa. At 21 days of age, the pups were removed from the mothers and housed in individual cages in the animal room. General activity levels were tested at 30 and at 60 days of age. Training in a water maze began at 80 days of age. Although there were no significant differences in body weights, litter sizes, or activity levels, there were significant differences between experimental and control groups in maze learning. Pups born to mothers that had audiogenic seizures during pregnancy had significantly more errors and required significantly more trials than did pups born to controls even if the control pups were cross-fostered on experimental mothers. Ishii and Yokohori (1960) found that female mice exposed to 90, 100, or 110 phon. white noise for 6 hr per day from the eleventh through the fourteenth day of pregnancy had more malformed young, more young still-born, and smaller embryos than did unexposed mice. Teratogenic effects produced

by audiogenic stress were also reported by Ward, Barletta, and Kaye (1970). A motorcycle horn producing 82-85 dB SPL at 320-580 Hz was timed to deliver noise intermittently for 60-75% of each hour. Female albino mice (Swiss-Webster strain) were placed in the chamber and exposed to the noise for at least 5-hr periods at different stages of pregnancy (vaginal plug was used as indicant of pregnancy). The most severe effects were obtained with stress 8 hr per day on days 8 to 17 of pregnancy. In these cases, 40% of the litters were resorbed and mean fetal weight was 0.44 g while mean fetal weight in control litters was 1.45 g. Although only moderate noise levels were used, there were severe results if stimulation occurred during critical periods. Stress during days 7-8 resulted in 100% resorbtion by day 18. Observed teratogenic effects (cranial hematoma, dwarfed hind limbs, and tail defects) were attributed to endocrinologic effects of stress on the mother and/or the fetus. These stress effects resulted in discharge of catecholamines and steroids from the adrenals. Decreased uterine and placental blood flow were considered to be responsible for fetal hypoxia, and perhaps delayed implantation. At least one experiment has shown there is a relation between noise exposure and susceptibility to viral infection in audiogenic seizure susceptible strains of mice. Jonson and Rasmussen (1970) used an 800-Hz tone with an intensity of 120-123 dB for 3 hr each day on 6-8 week old Swiss Webster HRVS mice. Mice innoculated intranasally with vesicular stomatitis

virus just before exposure to sound were more susceptible to the infection, while mice innoculated after the exposure were more resistent. The sound stressed mice were also more susceptible to polyoma virus and developed more tumors than controls that were not sound-stressed. The sound suppressed the progression of Rauscher virus lukemia. The inflammatory and interferon responses were also impaired by sound. They also found that the sound stressed mice had periods within each day when they might be more, less or just as susceptible to viral challenge as non-stressed control subjects. This transitory change in susceptibility was found to be independent of adrenal function. In addition to undesirable effects of noise that have been demonstrated in audiogenic-seizure susceptible mice, a recent study reports noise-induced hemorrhages in dogs. Ponomar'kov, Tysik, Kidryavtseva, Barer, Kostin, Leshchenko, Morozova, Nosokin, and Frolov (1969) exposed dogs to 0.6- to 3.5 sec bursts of white noise at 105 to 155 dB. Two hours after exposure, 3 mm diameter hemorrhages were found in the lungs, if noise levels exceeded 125 dB. Increased noise levels resulted in increased numbers of hemorrhages, but not in increases in the size of each spot. Emphysematous changes induced by noise exposure were still detectable at 60 days postexposure, even though hemorrhaged blood had been resorbed.

Noise has also been demonstrated to disrupt behavior in laboratory animals. Monakhov (1958) reported that rats exposed for 7 days to sounds produced by electric bells (for

45 min to 2 hr per day) became untidy and less active, refused to eat, and became aggressive. Borisova (1960) stated that white rats exposed to 85-dB noise displayed weakened conditioned reflexes. Five days of rest were necessary for the reflexes to return to normal.

Permanent effects produced by raising 80 albino rats in two different litter sizes and under two different sound levels were reported by Groh (1965). The rat pups were divided into litters of either 3 or 13 animals then randomly assigned to lactating females other than their own mothers. Half the rat pups in each litter size were raised in sound-proof boxes; the other half were raised in regular wire cages in a noisy animal room. There were 10 male and 10 female pups in each of the four groups. After 21 days under these conditions, the rats were weaned and placed, four animals to a cage, in the common animal room for an additional 21 days. At the end of this period (42 days) measures were made of body weights, spontaneous activity in an open-field test, heart rate increases following electric shock, and response latency in a straight runway at the end of 20 trials. Open field measures were repeated at 56 days and body-weights at 57 days. After these tests, relative weights of the adrenal gland were measured. Rats in large litters weighed less and had larger adrenal glands. Rats raised in soundproof boxes learned faster (had lower latencies) in the straight runway than did rats raised in the animal room. Decreased activity in the open field test and increased heart

rate responses were more pronounced in rats raised in large litters in soundproof boxes and in those raised in small litters in the animal room than were those in the other two groups. With the possible exception of the cardiac response, all these morphological and behavioral changes appeared to be stable.

There are several factors which most of the studies cited above have in common and which merit general comment. The SPLs used were mostly those which would be described as high or intense, and the duration of exposure in most cases was sufficiently short that it would be typified as acute rather than chronic. A danger in generalizing from "acute" high or relatively high intensity level studies to "chronic" low levels of stimulation is that there may be no relationship at all. The longest exposure duration cited in non-auditory effects was 150 days. That should probably be considered a chronic exposure; however, the next longest exposure was 42 days, which would hardly qualify as a chronic exposure except perhaps for relatively short-lived organisms. The levels of stimulation cited were as high as 160 dB with most in excess of 100 dB and with few below 90 dB. These are levels much beyond what we would normally find animals exposed to around airfields, industries, highways, or other intrusions by man into their habitat. It would seem logical to expect little or no auditory damage to animals from the usual invasions by man into the animals' world. Other physiological or endocrinological damage may result, however, the evidence

for such damage is at best conflicting and in need of elaboration. It would appear that experiments to determine the effects of long term exposure to lower sound levels have not been performed. With respect to non-auditory effects, it is unlikely that lower levels of stimulation for moderate durations would produce observable changes in laboratory animals in sexual function, cholesterol or ascorbic acid levels, etc. Another important fact which should be made explicit here is that the audible range of hearing varies widely from organism to organism. This might be expected to be a significant factor in studies to determine the effects of sound on the organism. Little or no mention of this is found in most of the studies cited, nor is there any evidence of concern about this factor.

In summary, in laboratory animals high levels of stimulation for fairly short durations have produced results suggestive of significant effects of noise on sexual function, blood chemistry, auditory function, seizure susceptibility, etc. Extreme caution should be used, however, in generalizing from results obtained on these animals stimulated at the levels and durations used, to other animals stimulated at lower levels for different durations.

Effects of Noise on Farm Animals

Although some studies have been conducted on domestic animals of economic importance, experimental controls and

adequate response measurement techniques have been lacking. Since no criteria have been established as far as measurement and recording of sound stimuli and animal responses to these stimuli, it is difficult to compare the effects of noise on one type of domestic animal with effects found in other domestic animals.

Effects of Noise on Mammals

Swine exposed to five trials of aircraft sound of 120-135 dB showed no injury to gross anatomy or the organ of Corti when compared to a control group exposed to ambient noise levels of 70 dB from an airfield (Bond, Winchester, Campbell and Webb, 1963).

Bond (1963) made extensive tests on the effects of noise on swine. During acoustic stress consisting of 15 sec of 130 dB noise repeated four, eight, or more times, heart rate monitored by telemetric equipment attached to naive swine increased significantly from normal heart rate. Heart rate decreased 30 sec after cessation of the sound stress but had still not returned to pre-exposure level. Frequencies employed were between 300 and 600 Hz. Bond (1970) also found that although no differences in reactions of nursing sows to frequencies ranging from 200 to 5,000 Hz were noted at 100 to 120 dB, a recording of a squeal of a baby pig at 100 dB elicited the same response. The reaction consisted of the nursing sow rising to her feet and searching for the sound

source followed by indifference. Baby pigs in the absence of the dam, exposed to the same sounds as cited above typically reacted by huddling together. The same investigator (Bond, 1970) found that exposure to loud sounds (frequency and intensity not specified) caused negligible reactions in mating swine. Sows and boars appeared indifferent to the sounds. Effects on partuition included heavier piglets at birth and a weaning from sows exposed to sound of 120 dB from 6 AM to 6 PM for three days before partuition until weaning. Bond (1963) found that pigs exposed to jet and propeller aircraft sounds reproduced at 120 to 135 dB daily from 6 AM to 6 PM from weaning time or before, until slaughter at 200 pounds body weight, showed no differences from pigs unexposed to the sounds with regard to feed intake, feed utilization and rate of gain. (In his 1970 review of the literature on the physiology and behavior of farm-raised animals, Bond cites Bugard, et al. (1960) in reference to effects of noise on young, castrated, male pigs) Bugard (1960) found that 93 dB noise for several days (frequency not specified) resulted in aldosteronism and severe retention of water and sodium in young, castrated, male pigs. He further stated that "alarm signals" recorded from pigs in the slaughter house disturbed the pigs more than mechanically produced sounds.

Parker and Bayley (1960) reported that milk cow herds within 3 miles of eight air force bases using jet aircraft, with 13% of the herds within 1 mile of the end of an active

runway, showed no differences in milk production when compared to herds which were not exposed to the aircraft noise. No differences were found between herds close to the end of the runway and those farther removed.

Casady and Lehmann (1966) reported that studies conducted on herds of milk cows at Edwards Air Force Base may have been biased in that the animals used had been exposed to 4-8 sonic booms a day for several years. Therefore, even though the intensity of the booms used during testing was higher than those the cows heard daily, the cattle may have adapted before the actual testing began. The investigators found, over all, few abnormal behavioral reactions in large animals due to sonic booms.

Bond (1956) in his review of the literature on sound stimuli effects on man and lower animals, stated that cows exposed to exploding paper bags every few seconds for 2 min during milking did not give milk while the sound stimuli were present. Thirty min following the sound stimulation, 70% of normal milk production occurred. Bond also cites Oda (1960) who stated that motorboat noise also produced a decrease in milk production. However, calf and heifer growth was unaffected by motorboat noise. Bond (1956) also reported that observers found a mild reaction in dairy and beef cattle to only 19 out of 104 sonic booms of 2.6-0.75 lb per sq ft. Milk production was unaffected during the test period. In fact, Bond noted that reactions to low subsonic

aircraft noise were more pronounced than were reactions to sonic booms. Further, the same reactions were observed in response to flying paper, strange persons, or other moving objects. This observation may indicate that "fright" reactions occur more strongly when the animal sees rather than hears the object.

Effects of Noise on Poultry

Stadelman (1958a) found that when fertilized eggs from white hens were held 1-7 days after laying and then subjected to incubation under conditions of sound (over 120 dB) or no sound (under 70 dB), no adverse effects occurred. The sound produced inside the incubation boxes consisted of playbacks of recorded background airfield noises, and noise from propeller and jet aircraft. Sound was present eight out of every 20 min from 8 AM to 8 PM each day and from 8 PM to 8 AM every third night. There were no effects on hatchability of eggs or on the quality of chicks hatched.

Eighteen New Hampshire and Plymouth Rock hens were observed for broodiness for 3 days and then divided into two groups. Broodiness is defined as the cessation of egg laying and the onset of egg incubation. One group was exposed to the sound levels mentioned above while incubating 12 hatching eggs each. Hens in the other group were given 12 hatching eggs each but were not exposed to sound. In the group not

exposed to sound, all eggs were hatched. In the group exposed to sound, all except one hen stopped brooding within 2 hr. The exceptional hen, although she remained broody, hatched only one chick from 12 fertilized eggs (Stadelman, 1958a).

Stadelman (1958a) also reported that recorded aircraft flyover noise at 80 to 115 dB at 300 to 600 Hz played daily from 8 AM to 8 PM and from 8 PM to 8 AM every third night for 5 out of 20 min from onset of brooding until chicks were 9 weeks old resulted in no difference in weight gain, feeding efficiency, meat tenderness or yield, or mortality between sound exposed and non exposed chicks. It was, however, noted that the chicks subjected to the noise were observed and that the presence of the observers could have rendered the chicks more adaptable to changing situations than chicks raised under natural conditions.

In another experiment by the same investigator (Stadelman, 1958b) 2,400 crossbred meat chicks were exposed to the same noise levels as described above. However, the chicks were on a different schedule. The chicks were not exposed to sound until they were 31 days old, at which time they were exposed for 5 out of every 20 min for 4 hr. Chicks were not exposed to the noise again until they reached 45 days old. The sound schedule above was then reinitiated, with a 3 day break due to equipment failure, until they reached 10 weeks old. There was no difference in weight gain or feeding efficiency in chicks which were or were not

exposed. One chick was trampled to death when noise was initiated at 31 days and chicks ran to the end of the cage away from the speaker where the sound level was 20 dB less. The investigators hypothesized that during an actual flyover, the sound would not be louder at one end of the pens than the other; therefore, there would be no running away from the sound source.

Seventy-eight broody broad breasted bronze turkeys were exposed to recordings of low flying jet planes at 110 to 135 dB for 4 min in the third day of broodiness. This sound treatment typically resulted in a cessation of broodiness and a resumption of egg laying in a period of time shorter than the time period prior to resumption of egg laying in hens whose broodiness was interrupted by injections of hormones such as progesterone. In addition, hens injected with progesterone showed a reduction in egg production during resumption of egg laying whereas the sound treatment of broody hens produced no decrease in egg laying when egg laying was resumed following sound stimulation (Jeannoutot and Adams, 1961).

Embryonic chicks exposed to artificial "peeps" which mimicked the "peeps" actually emitted by bobwhite quail chicks were speeded up or slowed down as a function of the rate of speed at which the peeps were emitted. Three or more peeps per sec were instrumental in causing eggs to hatch whereas less than 3 peeps per sec did not increase hatchability in eggs (Vince, 1956).

Daily sonic booms with SPLs of 0.75 to 1.25 lb per sq ft had no adverse effects on the hatchability of chicken eggs exposed for 21 days during incubation (Bell, 1970).

One hundred twenty mink were exposed to simulated sonic booms with peak overpressure in the housing shed decreasing from 2.0 lb per sq ft in the front of the shed to 0.5 lb per sq ft in the back of the shed in a smooth gradient. A mean boom frequency of 485 Hz was used. Litter sizes of boomed mink were larger than those born to non-boomed mink. Although the first boom resulted in some apparently curious emergence from nests, no racing, squealing, or other evidence of panic was observed. Autopsies of kits which died of natural causes disclosed no disorders which could be traced to booming (Travis, Richardson, Minear, and Bond, 1968).

Tests in 1967 in Minnesota showed little or no response to 8 sonic booms in 10 days with reference to mink bitch behavior during breeding, birth of kits, or whelping. No cannibalistic behavior toward kits or any other evidence of panic was observed (Bell, 1970).

Demonstrated Effects of Noise on Wildlife

Few data are available regarding demonstrated effects of noise on wildlife and much of what is available lacks specific information concerning noise intensity, spectrum, and duration of exposure.

Effects of Noise on Mammals

Sprock, Howard, and Jacob (1967) subjected caged wild rats and mice to sounds of varying frequencies (100-25,000 Hz) and SPL (60-140 dB). The only effects of noise were decreased nesting near the sound source and death at very high intensities. Recorded rat distress calls were observed to reduce time spent by rats in the area of the sound source.

Confined colonies of wild Norway rats and house mice were exposed to pulsed ultrasound provided by an ultrasonic generator for 76 and 81 days respectively (Greaves and Rowe, 1969). The frequency, intensity, pulse duration, and length of time between pulses were not reported. After exposure, the rodents displayed aversion to the sonic field and did not re-enter the testing ground.

Cummings (1971) reported that underwater projections of recorded killer-whale sounds caused migrating gray whales to reverse their direction of movement. Similar recordings were used by Fish and Vania (1971) to prevent movement of white whales into an Alaskan river during the time that red salmon fingerlings were migrating to the ocean. Pure tone stimuli at 500 and 2,000 Hz and random noise in the band from 500 to 2,000 Hz were projected with the same intensity and the same on-off times as the killer whale sounds. These sounds also kept the white whales from moving up the river, but since the whales had previously been exposed to the

killer whale sounds no conclusions could be drawn about the effectiveness of the tones and the random noise in themselves.

It has been shown that bats are resistant to jamming (Griffin, McCue, and Grinnell, 1963). Apparently they orient themselves so that noise and signal are received from different angles. Signal masking is greatest when noise and signal are received from the same direction. A 60-dB electric bell rung twice a day from 6 to 7 AM and from 8 to 9 PM for 7 days resulted in histophysiological changes in the pineal gland and in the supraoptic nucleus in hibernating bats (Milino, Devorcorski, and Kratic, 1969). Hill (1970) reported the use of high frequency sound produced by 12 adjustable (4,000-18,000 Hz) dog whistles to drive 500-1000 bats from a nuclear power station. According to Crummett (1970), rabbits, deer, and some species of birds were repelled by an acoustic jamming signal (no details regarding the levels of the acoustic signal were given) produced by Av-Alarm, a commercially available noise unit. This unit produces 2 signals having frequencies of 2,000 and 4,000 Hz, which are amplitude and frequency modulated to maximize jamming efficiency relative to the particular species under observation.

Effects of Noise on Birds

Birds were most effectively repelled by high-intensity (not defined) recordings of the species' own distress calls (Langowski, Wight, and Jacobson, 1969; Mossersmith, 1970;

Wight, 1971). However, the same investigators reported rapid adaptation even to species specific distress calls when presented continuously. For maximum effectiveness, intermittent presentation was suggested.

Pearson, Skon, and Corner (1967) reported that residents of Denver, Colorado, were successful in dispersing flocks of starlings by playing recordings of starling distress calls for four evenings as the birds arrived at roosts. The recordings consisted of repeated cycles of 30 sec of starling distress calls played for 12 min. Participation in the dispersal effort of about one half of the human population in urban roost areas appears to be sufficient to disperse the birds to outlying areas where they are no longer a nuisance. Habituation to the recordings was not evident, although some residents played the recordings continuously.

Thompson, Grant, Pearson, and Corner (1968a) subjected groups of starlings to one of five different sounds and found evidence that the birds perceived specific information through differential auditory stimulation. The response measure was heart rate, telemetrically recorded. Distress calls produced by physically restrained starlings were fright producing as evidenced by a high heart rate acceleration and slow habituation to the sound. Escape calls emitted by other starlings subjected to avian predators caused slight heart rate acceleration and required two or three applications before

habituation occurred. A human voice produced elevated acceleration of heart rate and required two to three applications before habituation occurred. Feeding calls appeared to be "neutral" in that a negligible heart rate acceleration occurred and habituation took place after an average of 1.2 applications. The starlings, it appeared, were able to discriminate among sound stimuli and react to them in discrete adaptive ways.

Thompson, Grant, Pearson, and Corner (1968b) found that the normal heart rates of wild starlings were elevated during the day relative to night heart rate values. The birds studied were housed individually in acoustical chambers wherein normal day and night lighting regimes were simulated. Starling distress calls were used as an acoustical stimulus. Starlings are normally active during the day, and initial heart rate responses to 10 sec of the auditory stimulus during the day were significantly different from baseline heart rate. Although the same stimulus produced an initial, slow increase of heart rate at night, the decrease to baseline was slower than during the day. When starlings were tested individually, the initial response was lower and the decrease in heart rate faster than when the birds were tested in groups of five. Therefore, a "flock effect" seemed to be operating.

Block (1966) cited the use of tape-recorded distress calls to disperse roosting starlings during three series of treatments in 1962. The number of starlings was reduced from

10,000 to a few hundred during the experiment. It was also reported, however, that the roosts were subsequently reinfested by a majority of the former resident population.

In the final report of a Committee on the Problem of Noise (1963) it was reported that to scare birds a noise level of approximately 85 dB SPL at the bird's ear was required. Noise used consisted of loud bangs and birds' distress calls. Birds adapted quickly to the noise and it was recommended that in the case of distress calls they be used no more than 2 min out of each 20-30 min and only during the day.

A U. S. Department of the Interior report on Environmental Impact of the Big Cypress Swamp Jetport (1969) discussed B-720 jet flyovers at altitudes of 500 to 5,000 ft over two sites in the park. Observers reported that no birds were flushed and no disturbances observed. Noise levels ranged from SPLs of 75 dB (with plane at 3,000 ft) to 96.5 dB (with plane at 500 ft). However, it was also reported that few birds were in the area at the time and wind effects interfered with proper sound level readings.

Effects of Noise on Fish

The effects of sound on fish have also been studied (F A O Fisheries Rep. No. 70, 1968). It was noted in this report that fishing vessel noise, especially sudden changes in noise levels, can scare schooling fish. Both diving and

changes in direction by fish were observed. Low frequency noise appears to be the most frightening type of noise to fish.

Malar and Kleerekoper (1968) analyzed locomotor patterns of single naive goldfish before and after exposure to a 2,000-Hz sound at varying intensities, 30 cm from the source. Locomotor patterns of the fish were affected significantly above an intensity of 2.0 dynes/cm^2 (≈ 80 dB SPL).

Aplin (1947) reported that underwater explosions for seismic exploration kill some fish that have air bladders, especially if they are hit broadside by the pressure wave. These explosions clearly do not drive fish out of the area and most species of fish are resistant to these explosions.

Fitch and Young (1948) also reported fish kills while using explosives for seismic exploration. Deaths were caused primarily by rupture of the air bladders of the fish. They also mentioned that on at least three occasions explosions killed California sea lions, and that occasionally cormorants were killed while diving and California brown pelicans were killed if their heads were below the surface during an explosion.

Effects of Noise on Insects

The desirability of protecting stored grain from destruction by insects has led to several studies directed at the effects of noise on insects. Kirkpatrick and Harein (1965) reported a 75% reduction in emerging Indian-meal moth

adults following exposure during 4 days of the larval stage to a 120 to 2,000-Hz sound (SPL unreported). Lindgren (1969) used a variety of frequencies and intensities to study effects of sound on Indian-meal moths and flour beetles. He used pure tones of 70 Hz at 110 dB, 200 Hz at 113 dB, 1,700 Hz at 134 dB, 2,000 Hz at 120 dB, 10,000 Hz at 90 dB, 20,000 Hz at 71 dB, and 40,000 Hz with SPL not reported. He also used variable frequencies of 180-2,000 Hz at 90-105 dB and 180-2,000 Hz at 90-102 dB. He exposed the insects during the latter part of the pupal stage and for 2 to 4 weeks as unmated and/or mated adults. Very little, if any, effect was noted, with the possible exception of mated flour beetles exposed continuously to 40,000 Hz. Even though large numbers of insects were used in many replications, effects of sound exposure were difficult to demonstrate, because of variability in egg production. The conflict between the data of Kirkpatrick and Harein (1965) and of Lindgren (1969) possibly can be explained by stimulation at different stages of the insects' life cycles (larval vs. pupal and adult respectively) as well as by differences in the sound itself.

Tsao (1969) reported that Indian-meal moths ceased moving when stimulated by loudspeakers, bells, and whistles. He noted some evidence of sex-related differences in the range of 2,000-40,000 Hz. Cutkomp (1969) reported that a 72-hr exposure to a pulsed sound, having a frequency of 50,000 Hz, with 25 pulses per sec at 65 dB SPL, reduced longevity from 20 to 10 days in corn earworm moths and

Mediterranean flour moths. The sound was an aversive stimulus in that the insects were observed to move away from the sound source. In addition to longevity effects, the mean number of eggs per female was reduced 59% in the treated relative to the untreated group. Arkhepov (1969) reported that lethal effects of ultrasonic waves occurred with extensive exposure to high intensities (undefined) which resulted in thermal and physiochemical changes in organs and tissues of various animals.

In a progress report, Shulov (1969) described effects of pure tones on locusts. Although tones of 4,000 Hz at 80 dB SPL had little effect on feeding behavior, tones of 1,000, 4,000, and 10,000 Hz elicited a flying response on more than two out of three trials.

Honeybees cease moving in response to certain sounds. Frings and Little (1957) reported that frequencies between 300 and 1,000 Hz with intensities ranging from 107 to 119 dB SPL produced cessation of movement for up to 20 min. No habituation was observed although the study was continued for 2 months. Experiments by Little (1959) demonstrated that stimulation with sounds having frequencies from 200 to 2,000 Hz produced cessation of movement in honeybees. Vibration of antennae did not produce the effect, but vibration of any of the three pairs of legs produced the "freezing response."

Frings and Frings (1959) found that certain sounds attracted swarms of male midges. Frequencies of 125 Hz at 13-18 dB above the ambient noise level produced agitated

circling of the insects with aggregation around the sound source.

The above studies of wildlife show that intense sound is an aversive stimulus for most organisms studied. Sound, under somewhat longer exposure conditions, appears capable of inducing measurable physiological and behavioral changes in some organisms. Commercial use is now being made of acoustic devices to repel certain undesirable animals; it is logical to assume sound may also repel desirable animals as well. Insects also seem to be significantly influenced by sound, something to consider because insects are important items in many animals' diets and significant links in the food chain. Apparently an insect's life span and reproductive capacity may be affected by exposure to certain sounds. These findings certainly suggest caution should be exercised in allowing sound intrusion into animal habitats, not only because of possible direct effects on the animals themselves but also on items in the food chain of the animal.

Suspected Effects of Noise on Wildlife

Although there is a limited body of literature dealing directly with the effects of noise on wildlife, possible effects can be inferred from information dealing with: (1) signal production and communication; (2) auditory ranges for different species; (3) direct effects of noise that have

been demonstrated in laboratory or domestic animals, and (4) incidental observations of responses to noise in wild animals. The suspected effects can be categorized as either interference with signals or direct effects on the animal.

Interference with Signals

Thorpe (1969) discusses the significance of bird vocalizations and reports that the various calls convey many types of information such as distress, danger or alarm, warnings about territorial boundaries, recognition of a mate or of young, and presence of food. Increases in background noise can mask these signals and thus potentially influence such processes as spacing to obtain optimum population densities in an area, nesting and care of young, and detection of prey or escape from a predator.

Dooling, Mulligan, and Miller (in press) reported that the common canary has its greatest auditory sensitivity to the range of frequencies from 2,000 to 4,000 Hz, which is also the range of frequencies maximally represented in its songs. If this finding is representative, it would permit prediction of which species would be most likely to be affected by a noise having defined frequency characteristics. They discussed the relative importance of range of sensitivity, thresholds, frequency discrimination, and sound localization and concluded that the auditory capacity that is most essential to the organism will have the greatest representation in the

auditory system, at the expense of the others. That is, a capacity such as hearing sensitivity would be greatest in animals that rely heavily on auditory signals to survive (e.g., nocturnal predators and nocturnal prey) whereas frequency resolution would be more important to an animal that utilizes intra-specific signals to recognize and call a mate or to stake out a territory.

Potash (in press) reported that male Japanese quail, isolated from their mates, increased the frequency of their "separation calls" when ambient noise levels were increased from 36 dB A to 63 dB A. The increase in the frequency of the calls improved the signal to noise ratio. Such an increase should make detection and recognition of the signal and localization of the caller more likely. The ultimate significance to the quail is determined by whether the mate responds to the "separation call" before a predator does.

In attempting to analyze possible signal-masking effects of noise on animals, it is important to remember that different species are able to detect "sounds" that man cannot hear (e.g., the dog's response to the "silent" dog whistle). Sewell (1970) reported that rodents both emit and respond to ultrasonic frequencies ranging up to 40,000 Hz or even to 70,000 or 80,000 Hz in special cases. Pye (1970) reported the production of ultrasonic (i.e., above 20,000 Hz) signals by certain grasshoppers and moths, as well as from many kinds of rodents and bats. However, the audible range of most birds and reptiles lies well within man's audible range (Konishi, 1970; Manley, 1970).

Interference with signals has sometimes been used by man in attempts to control unwanted species. A commercially available device that broadcasts an acoustic jamming signal was described by Crummett (1970). The signal consisted of two different frequencies, at about 2,000 Hz and 4,000 Hz, which were frequency and amplitude modulated to provide a signal said to be compatible with species' specific neural time constants, thus maximizing jamming efficiency and minimizing adaptation. In a progress report, Messersmith (1970) described results of tests using acoustic signals to control crop depredations by birds. A commercially available device was used on blackbird flocks feeding on grain and recordings of starling distress calls were used on starlings feeding on grapes. Both "...were temporarily effective when used at high volumes and aimed directly at the birds." Diehl (1969) reported that a 22,000 Hz sound prevented new populations of rodents from entering the area protected by the sound, although it was necessary to remove resident populations of rodents by trapping or poisoning. It is possible that similar signal interference effects were produced by the "hum" of power lines which were reported to disturb reindeer and to contribute to difficulties in herding (Klein, 1971). The use of recorded distress calls also represents attempts to interfere with signals, and thus control certain unwanted species (e.g., Block, 1966; Fitzwater, 1970; Frings and Frings, 1957; Frings and Jumber, 1954; Pearson, Skon, and Corner, 1967).

Direct Effects of Noise

It is very possible that many of the noise-induced physiological and behavioral changes that have been demonstrated in laboratory animals could also occur in wild animals. Of course, it is very unlikely that wild animals will be subjected to noises intense enough or of sufficient duration to produce permanent hearing losses. However, chronic exposure to moderate noise levels could produce some hearing loss or influence processes that are hormonally regulated due to noise-induced stress responses. Until studies are performed in which effects due to exposure to noise are separated from effects due to capture, handling, or other kinds of interference, these answers will not be known.

Sonic booms, and especially the threat of the SST's "super-boom," generated extensive speculation about their effects on animals. Davis (1967) described his observations of some ravens in Wales. When the boom occurred, three or four ravens that had been cruising in the area were rapidly joined by others. Within 5 min approximately 70 ravens were agitatedly circling; 30 min later about 30 ravens were still flying in the area. Shaw (1970) reported that adult condors were very sensitive to noise and abandoned their nests when disturbed by blasting, sonic booms or even traffic noise. The most deleterious effects attributed to sonic booms were recent mass hatching failures of sooty terns

in Dry Tortugas, Florida, discussed by Bell (1970) and Henkin (1969). Following 50 years of breeding success, 99% of the terns' eggs failed to hatch in 1969. Extremely low-altitude supersonic flights over the area may have driven birds off their nests and damaged the uncovered eggs. Graham (1969) reported observations of destruction of pelican eggs by gulls when white pelicans were driven off their nests by sonic booms. Graham also said that a fisherman described the reaction of fish to sonic boom as "similar to those dynamited in a fishpond." (Author's Note: With the impedance mismatch between air and water this would seem an obvious impossibility and appear to lend credence to allegations made regarding the veracity of fishermen). Bell (1970), in a recent review of animals' responses to sonic booms, described only minimal reactions to sonic booms among domestic animals, ranch mink, and wild animals. The only clearly detrimental effect that he discussed was the Dry Tortugas sooty terns' hatching failure. A startle response to a sonic boom was the typical reaction that he reported.

Clearly, the animals that will be directly affected by noise are those that are capable of responding to sound energy, and especially the animals that rely on auditory signals to find mates, stake out territories, recognize young, detect and locate prey, and evade predators. These functions could be critically affected even if the animals appear to be completely adapted to the noise (i.e., they

show no behavioral response such as startle or avoidance). Ultimately it does not matter to the animal whether these vital processes are affected through signal-masking, hearing loss, or effects on the neuro-endocrine system. Even though only those animals capable of responding to sound could be directly affected by noise, competition for food and space in an ecological niche appropriate to an animal's needs, results in complex interrelationships among all the animals in an ecosystem. Consequently, even animals that are not responsive to or do not rely on sound signals for important functions could be indirectly affected when noise affects animals at some other point in the ecosystem. The "balance of nature" can be disrupted by disturbing this balance at even one point. We would do well to have some knowledge of what to expect from noise pollution in wildlife habitats before it produces its effects.

Discussion

It is now time for an overview of the literature found and a discussion of what it might mean. The best documented, most clearly proven effect of high intensity noise exposure on hearing organisms is that of damage to the auditory structure with a resulting loss of hearing. Now, assuming that the levels of noise produced are sufficient in an area to produce a loss of hearing in a given animal, what are the likely or possible consequences of

such a decrease in auditory sensitivity? First, it should be noted that animals differ in their audible range and the audible range for all animals is not known. Anticipated consequences of a loss of hearing ability are many. The prey-predator situation could be drastically changed. The animal that depends on its ears to locate prey could starve if auditory acuity decreased, the animal that depends on hearing to detect and avoid its predators could be killed. Reception of auditory mating signals could be diminished and affect reproduction. (Masking of these signals by noise in an area could also produce the same effect). Detection of cries of the young by the mother could be hindered, leading to increased rates of infant mortality or decreased survival rates. Distress or warning calls may not be received, again significantly affecting survival.

Considerably less assurance is possible in discussing the likely consequences of non-auditory effects. For one thing, at best some of the effects are small, many are not clear cut and reproducible under precisely controlled conditions, and some are only suggested. But assuming that there are non-auditory effects, as reported, an attempt will be made to anticipate some of their consequences.

The reports of significant changes in reproductive organs (testes and ovaries) and sexual function (estrus) should be viewed as possible serious threats to the animal's

reproductive capacity. If chronic exposure to sound pressure levels expected to result from known or projected sound sources could be shown to produce such effects, there can be little doubt about the danger to the species. Studies to verify and elaborate such effects should be made as soon as possible.

The literature describing audiogenic seizures following noise exposure, and possibly demonstrating increased susceptibility to audiogenic seizures in fetuses exposed to sound during critical stages of pregnancy can almost be dismissed summarily. First, audiogenic seizures can be induced in only certain strains of animals of a particular species. It is exceedingly difficult to induce seizures to acoustic stimuli in animals other than genetic strains known to be susceptible. There are references to such seizures in isolated individuals of various species including man but they are apparently rare. Thus we dismiss this effect as one meriting little or no further concern.

A number of physiological measures have revealed noise-induced changes in a variety of animal species. Apparently noise can affect the hypothalamic-hypophyseal system, producing alterations in electrolyte excretion, circulating blood levels of eosinophils, and release of catecholamines and steroids from the adrenals. Such changes can affect animals' abilities to withstand additional stress, and influence such hormonally-regulated functions as

mating and reproduction. Obviously these effects could have serious consequences for the species as well as for the individual organism. Sophisticated equipment and techniques will be necessary to obtain measures of such changes in wild animals in their natural habitat, if at all possible, so that noise-induced changes will not be confounded or masked by changes due to captivity and restraint.

The possible consequences of some of the behavioral effects noted are difficult to evaluate. Decreased exploratory behavior, immobility, and things of like nature could have significant consequences if they occur under conditions of chronic stimulation and do not adapt out over time. Any panic type behavior such as piling up or huddling, could well lead to problems for survival of an animal. Also, avoidance behavior could restrict access to food or shelter and therefore adversely affect an animal's or even a species' chances for survival.

In general then, few if any of the reported or suggested effects of noise on animals would benefit the animal or increase his chances for survival. On the other hand, some of them might possibly lead to his death or decrease his chances of survival.

Suggestions for Research

In examining the literature on the effects of noise on animals in general and on wildlife in particular, it is

extremely difficult to find where to begin in detailing needs for research. There are at least two reasons for this. With the exception of the large, well done body of literature exploring the effects of noise upon auditory structures and hearing, well controlled, well designed experiments substantiating non-auditory effects of noise are rare. In the case of wildlife, such studies are virtually nonexistent.

It is apparent then, that at least two different concomitant programs of research are indicated in order to fill the large gaps in our scientific understanding of the nature and extent of the effects of noise upon wildlife. A thorough, meticulous, and precise program systematically studying the effects of long term low level "chronic" noise exposure should be initiated to eliminate the uncertainties, ambiguities, and even conflicts in reports of non-auditory physiological, metabolic, sexual, and other physical effects of noise. It could well be that effects noted with "acute" exposure might not be observed under conditions of "chronic" exposure. It should not be necessary to add that the intensity, spectrum, and duration of exposure should be precisely set and controlled. Such a program should consider the auditory sensitivity of the specific animal studied and tailor acoustic stimulation to maximize the likelihood of results.

Concurrent with careful examination of physiological and other physical and chemical effects of noise on animals

should be a program of research devoted to the study of effects of noise on true wildlife, existing in their native habitat under normal conditions. Such a program would have many aspects and would of necessity require a multi-disciplinary approach. An adequate approach to the problem would entail study of many factors. Census counts of animals in their natural habitat would be necessary as well as detailed studies of their normal blood chemistry, reproductive functions, and any other aspects that there is reason to believe may be affected by changes in ambient noise exposure levels. A survey of the habitat should be made in depth, i.e., over long enough periods of time so that sufficient knowledge is amassed regarding infrequently occurring but relevant events. Once sufficient knowledge is available about the environment and its inhabitants, the sound levels in the environment should be systematically varied and the effects of such changes on the population compared with pre-change data for all of the levels considered. The changes in level, for the sake of validity, could well be due to sound one might expect from technological advances, i.e., aircraft noise, other transportation noise, or industrial noise. Such a course would at least provide face validity for the results. Such changes in level should be maintained for a considerable length of time to provide "chronic" rather than "acute" data. A minimum time course for a study of this nature, in the field, and under the conditions outlined above, would be 3 to 4 years. For some types of animals in some

places more time would be needed. At the same time in field studies, efforts should be made to determine whether the animals leave the area upon stimulation by higher levels of sound, and if so, do they later return, or is their place taken by other animals of the same, or other species. Other relevant questions to be answered would include, does the animal density level in the area increase, decrease, or remain the same? Does the general health, weight, etc. of the animal change? A study of predator-prey relations might also be valuable, to determine possible noise related but unobvious causes for changes in the population. Certainly the food supply of animals is important and if the data suggesting noise effects on insects were correct, the food source of some of the other animals in an area could change and thus be responsible for subsequent changes in the animal population. An essential part of a research program such as that suggested above would be to provide a control study area contiguous to the experimental areas and as similar as possible in every way. This kind of design is mandatory because of the wide normal variations in the population density of a great many animals. If unaccounted for, these cyclic normal fluctuations in animal populations might completely mask any real effects, if any, from the noise.

An important consideration in planning research should be the frequencies to be investigated, as well as the sound levels. Frequencies that are inaudible to humans (ultrasound) are well within the audible range of many animal species.

Aquatic mammals, bats, and rodents, emit cries having very high frequency components, which are considered to play an important role in communication. Potential noise sources must be analyzed to determine what ultrasonic (to humans) as well as what audible frequencies will be produced, and the impact of the entire range of expected frequencies on wildlife must be investigated.

Another area where research effort would seem to be justified and indicated would be that of effects of noise on various domestic animals. There are clear suggestions of possible influences of noise on sexual function, on the fetus and mother during pregnancy, on weight gain and utilization of food. In view of the economic importance of cattle, chickens, turkeys, sheep, and the many other domestic animals it is clear that research in this area might prove of value. Research of this general type is currently underway at the Institute National Recherche Agronomique, Jouy En Josas, France (personal communication, Dr. R. G. Busnel, INRA). For example, a problem they are currently considering is how to deafen young chickens cheaply and safely. They have evidence which leads them to suspect deaf chickens might gain more weight from the same amount of food, presumably because they were less distracted by the noises of the other chickens around them, were less nervous, or perhaps had lower activity levels.

It is exceedingly difficult to assign priorities to the research suggested above. When all of it is necessary

and should be done in order to provide the complete information essential for decisions, all that can be done by way of assigning priorities is to point out that possibly more information of immediate and practical use could be gleaned from field studies than from laboratory studies. If conducted on a sufficiently large scale and encompassing a large enough scope, vital information regarding the effects of noise on wildlife could be secured in 3 or 4 years. It would still be required that concurrent laboratory studies be conducted, however, in order to obtain information that could only be secured through laboratory research.

Appendix*

The literature search can generally be divided into a search by manual, computer, personal interview, and written communication means. To assure depth of coverage, the literature of medicine, agriculture, conservation, and science was searched.

Manual searches were conducted in the public catalogs of Memphis State University Library (John Brister Library), University of Tennessee Medical Library (Mooney Memorial Library), and other libraries listed in the source bibliography. A relatively small number of books and monographs was found in the catalogs. A comprehensive manual search on the abstracts, indexes, and bibliographies listed in the source bibliography was carried out.

It is the desire of everyone who has worked on this search to thank the many people who helped in any way, especially those who gave time for personal interviews and correspondence.

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*The literature search was conducted under Contract No. 68-04-0024 from the Environmental Protection Agency under the direction of Dr. John L. Fletcher, Professor of Psychology, and Dr. Michael J. Harvey, Associate Professor of Biology. Wilma P. Hendrix compiled the source bibliography and served as library consultant. The information was obtained and analyzed by June W. Blackwell, Virginia M. Norton, Clara B. Davis, and Richard L. Taylor.

Library Catalogs Searched

Department of the Interior Library

H. W. Calhoun Medical Library (Administrative Headquarters for the Southeastern Regional Medical Program)

John Bristor Library, Memphis State University

Library of Congress

Library of the National Academy of Science

Mooney Memorial Library, University of Tennessee Medical Units

National Library of Medicine

Smithsonian Institution, Library of Natural History

Robert W. Woodruff, Library for Advanced Studies, Emory University

Computer Searches

Alabama MEDLARS Center
The University of Alabama
Medical Center Library
Birmingham, Alabama 35233

Effects of Noise Pollution on Wildlife, January, 1964 - December, 1968.

Key Words: Animal kingdom - invertebrates
Animal kingdom - vertebrates
Acoustic trauma
Acoustics
Audiometry
Auditory perception
Auditory threshold
Hearing
Hearing tests
Noise
Pitch discrimination
Sound
Ultrasonics

Effects of Sound on Wildlife, January, 1969 - July, 1971.

Key Words: Animal kingdom - invertebrates

Computer Searches (continued).

Animal kingdom - vertebrates
Acoustic trauma
Acoustics
Auditory perception
Hearing
Hearing tests
Noise
Sound

Library Reference Service, Current and on going research
Conservation Library Center
Federal Aid in Fish and Wildlife
Denver Public Library
1357 Broadway
Denver, Colorado 80203

Noise Pollution and its Effects on Wildlife

North Carolina Science and Technology Research Center (STRC)
Research Triangle Park, North Carolina 27709

Biological Abstracts, 1959 - June 1971

Key words taken from B.A.S.I.C. Keyword and Subject Index

Preliminary searches were conducted on each of the following:

The NASA Information File
Department of Defense File
Engineering Index
Chemical Abstracts

The results of the preliminary searches were such that the STRC engineers advised that no further attempts be made to search these files for materials on noise and its effects on wildlife

Science Information Exchange
Smithsonian Institution
A National Registry of Research in Progress
Madison National Bank Building
1730 M. Street, N. W.
Washington, D. C. 20036

Effects of Noise, Ultrasonics, and Other Sound Frequencies on Wildlife and Insects

Computer Searches (continued).

Aircraft Noise and Sonic Boom Studies: Effects on People, Animals and Buildings
Generation and Propagation of Noise
Development and Design of Low Noise Aircraft Engines
Noise in the Vicinity of Airports
Noise Abatement Studies

Abstracts, Indexes, and Catalogs Searched

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Index Catalogue of the Library of the Surgeon General's Office, United States Army. Washington, Superintendent of Documents. Series, 1880-1961.

Index Medicus

Quarterly Cumulative Index to Current Medical Literature. AMA, Chicago, 1916-26.

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Current List of Medical Literature. Army Medical Library, Washington, (Vols. 19-36), 1950-1959.

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International Abstracts of Biological Sciences. London, Pergamon Press, 1956 - May 1970.

Monthly Catalog. Washington, Superintendent of Documents, 1950 - May 1970.

National Library of Medicine Catalog. Washington, D. C., National Institute of Health, Public Health Service, Health, Education and Welfare Department, 1955 - 1965.

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Carlisle, John G., Jr. Associate Marine Biologist, Department of Fish and Game, Marine Resources Region, 350 Golden Shore, Long Beach, California 90802.

Chatham, George N. Analyst in Environmental Policy, Environmental Policy Division, Legislative Reference Service, Library of Congress.

Cope, Oliver. Fisheries Research, Bureau of Sport Fisheries and Wildlife, Fish and Wildlife Service, Department of the Interior, Washington, D. C.

Crummett, James G. Av-Alarm Corporation, 960 N. San Antonio Rd., Suite 170, Los Altos, California 94022

Curtis, William H. The Wilderness Society, Washington, D. C.

Fish, James F. Naval Undersea Research and Development Center, Department of the Navy, San Diego, California 92132.

Foster, Charles R. Department of Transportation, 400 7th Street, S. W., Washington, D. C.

Gales, Robert S. Naval Undersea Research and Development Center (The Listening Group), San Diego, California 92132.

Gates, Doyle. Manager, Marine Resources Region, California Department of Fish and Game, 350 Golden Shore, Long Beach, California 90802.

Konishi, Masakazu. Associate Professor, Department of Biology, Princeton University, Princeton, New Jersey 08540.

Lemke, Darrell H. Coordinator of Library Programs, Consortium of Universities, Washington, D. C.

Lipscomb, David M. Associate Professor of Audiology and Speech Pathology, Director, University of Tennessee Noise Study Laboratory, Knoxville, Tennessee 37916.

Marler, Peter. Professor of Animal Behavior, Department of Animal Behavior, Rockefeller University, 66th Street and York Avenue, New York, New York 10021.

Miller, James D. Head, Psychology Laboratories, Research Department, Central Institute for the Deaf, 818 South Euclid, St. Louis, Missouri 63110.

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Segal, Migdon. Analyst in Environmental Policy, Environmental Policy Division, Legislative Reference Service, Library of Congress.

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Thompson, R. D. U. S. Bureau of Sport Fisheries and Wildlife, Denver, Colorado.

Tombaugh, Larry. National Science Foundation, Washington, D. C.

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Agricultural Research Center. U. S. Department of Agriculture, Beltsville, Maryland.

Bell Aerospace Company. Buffalo, New York.

Bell Laboratories. 600 Mountain Ave., Murray Hill, New Jersey 07974.

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Bureau of Sport Fisheries and Wildlife. U. S. Department of the Interior, Washington, D. C.

Citizens League Against the Sonic Boom, 19 Appleton Street, Cambridge, Massachusetts 02138.

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Defenders of Wildlife. 2000 N Street, N. W., Washington, D. C. 20036.

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National Wildlife Federation. 1412 Sixteenth Street, N. W., Washington, D. C. 20036.

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