Marine Mammal Auditory System Noise Impacts: Evidence and Incidence

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1 Introduction

Sound is an inevitable element of every human activity in the oceans. Some, like exploration and military sonar exercises, produce impulse sounds that are intense but infrequent; others, like shipping, generate non-impulsive, less intense, but continuous noise. A recent NRC review (2003) found that the ocean's acoustic budget has increased by 3 dB, i.e., doubled, per decade in the last half century. In effect, in some ocean areas, and particularly along our fragile coasts, we are creating an environment akin to that of human workplaces.

It is reasonable that we are concerned that any sound added to the marine environment may adversely impact a species within its "acoustic reach". Our concern for marine mammals is particularly acute because hearing is arguably their primary sense. In fact, the important issues are multifactorial: what species will be exposed, for how long, to what frequencies, and at what levels, and then how do these parameters compare with an animal's hearing abilities. Only with all these factors in hand can we reliably determine the probability of adverse impacts affecting fitness or endangering populations. Before we can have a useful perspective, much less responsibly impose regulations and sanctions to prevent impacts from anthropogenic oceanic noise, it is first necessary to understand the susceptibility to noise damage in marine mammal stocks and the current status of their hearing. In the last decade, we have gained substantial ground in testing and documenting both normal and impaired hearing in some marine mammals, but we must still infer hearing characteristics for the majority of species. This paper summarizes our knowledge of sound impact mechanisms in land mammals, the current evidence for

marine mammal hearing loss, and, finally, in the context of these data, the implications for when and how marine mammal ears may suffer noise damage.

2 Noise Induced Hearing Loss (NIHL)

Noise is not synonymous with sound. Sound is a physical phenomenon perceived through hearing, whereas noise is defined essentially as an aperiodic signal that interferes with the perception of sound and has a negative physiological impact. Experimental and human noise effect data reviewed by Davis et al. (2003), Kryter (1996), and Slepecky (1986) are summarized in the following sections.

2.1 Human Incidence

Humans are, in one sense, an on-going, natural experiment for noise impacts. NIHL is second only to aging effects as a cause of loss among humans. We have long been aware that repeated exposure to loud noises may result in hearing loss. Early stage NIHL in humans manifests itself as a "notch," or preferential loss, near 4 kHz, but it may extend as high as 6 kHz for extensive impulse exposures (Humes 2010). As NIHL progresses, distinctive threshold increases occur near the peak frequency and at partial octave intervals of the offending signal as well as frequencies above 4 kHz. Current OSHA regulations permit chronic exposures at an average sound pressure level (SPL) of 85 dB (A-weighted) over an 8-hour period, with a mandatory halving of exposure time for each 3 dB increment in SPL. European standards are comparable. Recent statistics indicate that approximately 15% of people over 20 years of age in the US have high frequency hearing loss attributable solely to noise exposure. By age 45, 20% have substantial NIHL; by 75, approximately 50% of the population has profoundly impaired hearing from presbycusic; i.e., age related, loss that is the result of long-term noise exposure compounded by diminished cellular recovery (www.nidcd.nih.gov).

Although other mammalian species are often used to investigate NIHL mechanisms, natural hearing loss is not commonly studied in any other species. Consequently, we know little about the incidence and nature of long-term noise effects in most mammals, making it difficult to estimate the state of health of "natural" marine ears and the probable risks from anthropogenic sources for wild marine mammal populations.

2.2 Mechanisms of NIHL

There is no simple single factor or formula for estimating loss from noise. Any noise exposure will not necessarily result in a measureable hearing loss, but every ear has tolerance limits. Sounds within an individual's hearing range may be noisome or damaging depending on the synergistic effect of several factors; e.g., intensity, frequency, duration, whether the signal profile is impulsive or continuous, and the subject's sensitivity at that frequency.

The fundamental cause of NIHL is overstimulation of the inner ear sensory cells, which results in metabolic exhaustion of the hair cells, organ of Corti support cell damage, and, in severe cases, retrograde ganglion cell and axonal degeneration. First order damage, the protracted bending and shearing of auditory hair cell stereocilia, prevents production of neurochemical releasers that initiate auditory fiber impulses. In effect, the impacted area of the inner ear becomes chemically "silent" as a result of the loss of the stereociliary triggers. If the stereocilia recover to any extent, the function returns but may require greater energy than previously to respond, and thresholds increase. Longitudinal and radial variations in cell structure along the cochlea also produce micro-regions with different vulnerabilities. Finally, adjunct conditions, such as exposure to ototoxins, heavy metals, hypertension, or stress hormones, may accelerate or exacerbate losses.

Whatever the incipient cause of damage, some structural correlates for loss types are now fairly well understood. Damage to inner hair cells results in a total lack of response whereas the loss of outer hair cells produces elevated thresholds. If hair cells recover from noise insults, the attendant loss is a temporary threshold shift (TTS). In experiments, threshold elevations as large as 50 dB returned to baseline sensitivity, although recovery sometimes required as much as 30 days. Shifts over 50 dB are often permanent (PTS). In humans, they most often result from an extreme, acute exposure or from accumulated TTS insults to the ear, some of which may occur without allowing recovery from a prior TTS. It is generally assumed that TTS represents a full recovery with no attendant physical injury, but it has been difficult to explain how even a reversible loss occurs without at least submicroscopic physical damage. Recent data (Kujawa and Liberman 2009) show that in TTS, sensory cells appear intact but there

can be acute afferent nerve terminal damage and cochlear nerve degeneration. Thus TTS to PTS may not

have fundamentally different mechanisms but rather are a graded, although nonlinear, continuum.

There are two intriguing features in threshold shifts. One is that continuous high SPL exposures

result in losses at the center frequency (CF) and at higher loci at half octave intervals that are explained

by cochlear nonlinearities. However, this does not account for anomalous damage near the hook nor for

the notch phenomena that may be related to spiral topography. The second is that individual responses to

identical exposures can be as great as interspecific differences except in genetically identical individuals,

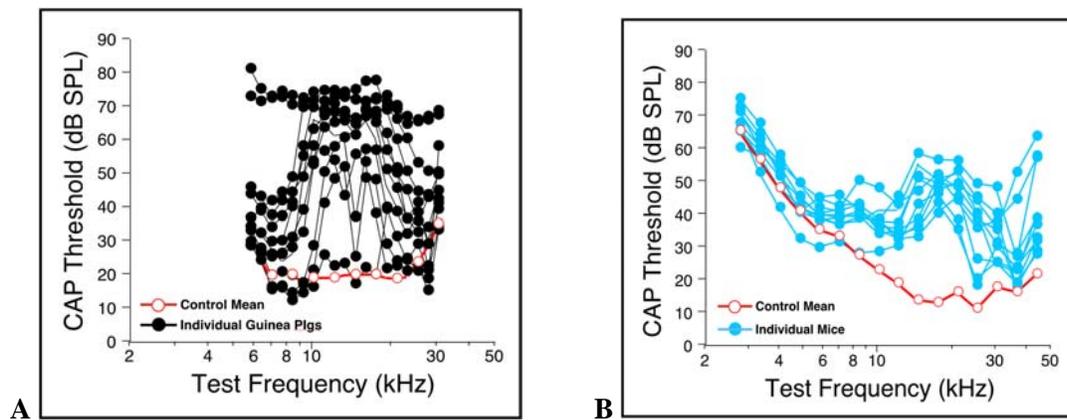as demonstrated in mice (Fig. 1), implying there is a large genetic element in NIHL susceptibility.

Fig. 1. (A) Guinea pigs exposed to12-kHz narrowband noise for 4 hrs, 109 dB SPL. (B) CBA/CaJ mice

exposed to 1 to 16 kHz octave-band noise for 2 hrs, 103 dB SPL. CAP: compound action potential (Figures

updated from Yoshida and Liberman 2000, courtesy of C. Liberman;)

2.3 NIHL Summary

Common findings across species for noise effects are as follows: inner ear damage locations and severity

correlate with the power spectrum of the signal but higher frequencies may also be impacted; intensity

and duration can act synergistically to broaden the loss; there is a critical limit beyond which shifts grow

rapidly; continuous exposures over time are asymptotic; impulse noise produces more profound effects

than continuous noise at equivalent levels; onset limits for TTS are the same for normal and hearing

impaired individuals, thus there is a smaller "shift window" for impaired individuals; effects spread more

to higher frequencies from any stimulus, possibly because the tonotopic structure of the basilar membrane

means all incoming signals first traverse higher frequency encoding regions at the base of the cochlea before reaching lower frequency regions. Temporal integration is reduced, but frequency discrimination may be preserved in both TTS and PTS. Signal rise time and duration of peak pressures are significant factors in PTS but not in TTS.

3 Marine Mammal Hearing Loss: Evidence

Potential impacts from noise in marine mammals, just as in land mammals, may be physiological, pathological, acute or chronic, and even subclinical or largely behavioral. Many papers in this volume provide details on all these aspects; therefore, this review is confined to the key points of physiological and anatomical elements of hearing loss.

It is not news that some marine mammals may be hearing impaired. This is evident in hearing curves published over the last 50 years (Fig. 2). In the last 10 years, awareness of preexisting loss and testing to determine the onset of TTS via behavioral and noninvasive auditory evoked potential (AEP) techniques have both increased.
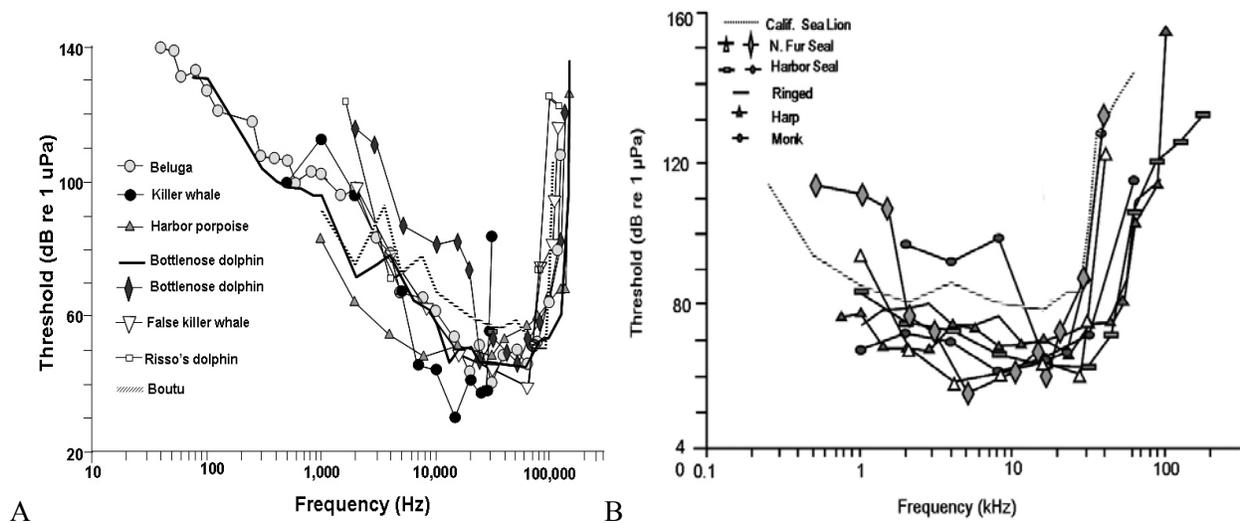

Fig. 2. Odontocete (A) and pinniped (B) audiograms. Elevated thresholds for one of the bottlenose dolphins, fur seals, and harbor seals indicate hearing deficits. (Modified from Wartzok and Ketten 1999)

Published results are now available for 12 species of odontocetes and pinnipeds for tonal, impulse, and octave-band noise stimuli. With the exception of tests on a few stranded juvenile whales and

dolphins, all data were obtained from captive animals with prior test experience, some of which are older subjects with high frequency losses. Southall et al. (2007) provides a detailed discussion and original citations for the studies summarized below. All SPLs are dB re 1 μPa and SELs are dB re 1 μPa^2-s unless otherwise noted.

For two odontocete species, *Tursiops truncatus* (bottlenose dolphin) and *Delphinapterus leucus* (beluga whale), tested in a natural harbor, a 6 dB or greater shift for single, short duration pulses required exposures of 160 kPa peak (SPL 226 dB peak-peak; SEL 186 dB) whereas for pure-tone stimuli (3-80 kHz) with short exposures (up to 8 s), the mean shift onset was 195 dB SPL (SEL 192-201 dB). The lowest onset was at 182 dB SPL for 1 subject at 75 kHz. With longer exposures (up to 130 s), greater shifts occurred (23 dB) at equal or lower exposures. As seen in land mammals, shifts occurred also at octave intervals and at frequencies above the probe stimulus. Equivalent shifts were obtained at similar received SELs under more controlled pool conditions, implying that masking from harbor noise was not a factor. Studies with longer stimuli (30 min) using octave band noise (OBN) reported 11 dB shifts with stimuli of 179 dB SPL and 212-214 dB SEL, but exact exposures required monitoring the subject for surface breaths. Parallel AEP studies for *Tursiops* using longer exposures at surface (50 min OBN) found 4 to 8 dB shift onsets at 160 dB and 193-195 dB SEL. AEP and auditory steady state response (ASSR) studies show the same trends as behavioral studies but often report shifts 10-20 dB greater. SEL growth is the most consistent finding among these studies, with a typical rate of 1 dB TTS/dB SEL. Recovery rates vary somewhat from 1-2 dB per doubling of time for short exposures to low or mid-frequency signals vs. 5-6 dB per doubling of exposure time for frequencies closer to peak sensitivities.

TTS studies in pinnipeds have been conducted in air and in water for 6 species with parameters similar to those for cetaceans. TTS, like basic hearing, among pinnipeds covaries with aquatic vs. aerial adaptation and body mass; i.e., smaller phocids are impacted at lower exposures underwater than larger species or more air-adapted otariids. At 152 dB SPL (183 dB SEL), *Phoca vitulina* (harbor seals) sustain a TTS of 6-8 dB after a 30-min exposure to 2.5 kHz OBN compared with TTS onsets near 170 dB (SEL 205 dB) for *Zalophus californianus* (California sea lion) and an adult *Mirounga angustirostris* (northern

elephant seal). In air measures for the same subjects and regimens required 99 dB re 20 μPa (SEL 131 dB) for 6 dB of TTS in the harbor seal vs. 121-122 dB re 20 μPa (SEL 154-163 dB) in the sea lion and elephant seal. As in cetaceans, recovery times were relatively short but longer exposures of 50 min required 3 days recovery, in common with land mammal data. SEL values were lower than in cetaceans, with a growth rate of ~2.5 dB TTS/dB noise and ~2.5 dB/doubling.

4 Conclusions

Undeniably, there have been serious consequences from noise exposures for marine mammals, including mass strandings. However, despite the importance of such events in bringing underwater noise to our attention, ironically, to date, there has been no demonstrable evidence of acute, traumatic, disruptive, or profound auditory damage in any marine mammal as the result anthropogenic noise exposures, including sonar (d'Amico et al. 2009; Ketten et al. 2003). This does not negate our concern but rather underscores our need for a better understanding of the many facets and consequences of sound use.

Although we are still uncertain about how robust or fragile these ears are, we have clear evidence that despite adaptations for diving and high-pressure environments, they are not impervious to permanent noise damage. Longitudinal studies report broad hearing losses and steep notches in both odontocetes and cetaceans (Ridgway and Carder 1997; Schusterman et al. 2002. Recent postmortem studies of ears from some of these subjects (Ketten et al. 2008) found evidence of sensorineural hearing loss in the form of extensive ganglion cell and auditory nerve fiber degeneration consistent with profound hearing deficits. Ears from strandings also show NIHL and age related changes as well as other ear pathologies, including labyrinthitis ossificans, parasitic infestations, trauma, and chronic otitis media.

Despite these indicators that marine mammal hearing losses likely occur by the same mechanisms and etiologies as in land mammals, current data are insufficient to formulate population level hazard criteria. Southall et al. (2007) attempted a risk matrix approach for noise exposures using available audiometric and anatomical data and weighted SEL and peak SPL criteria but concluded that data gaps prevent assigning definitive exposure criteria. Furthermore, the fact that marine mammals sustain natural hearing loss from aging, trauma, and disease complicates definitive attribution of hearing loss from anthropogenic

sources based on sampling a few individuals in coastal populations. Multiple possible loss causes should be considered and eliminated in any animal for which there is little or no history; therefore, the simple finding of a single animal with a hearing decrement cannot be taken as a clear indicator of a population-level hazard from any source without corroborative evidence. Protracted exposures and TTS to PTS conversions rather than acute individual impacts may be our major concern for populations. A high incidence of NIHL is unlikely to occur naturally across genders and ages in any wild population. Such a finding coincident with long-term or frequent intense exposures would be an appropriate cause for significant concern and action.

Acknowledgments

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FIGURE LEGENDS

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