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Sonar-induced temporary hearing loss in dolphins

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There is increasing concern that human-produced ocean noise is adversely affecting marine mammals, as several recent cetacean mass strandings may have been caused by animals’ interactions with naval ‘mid-frequency’ sonar. However, it has yet to be empirically demonstrated how sonar could induce these strandings or cause physiological effects. In controlled experimental studies, we show that mid-frequency sonar can induce temporary hearing loss in a bottlenose dolphin (Tursiops truncatus). Mild-behavioural alterations were also associated with the exposures. The auditory effects were induced only by repeated exposures to intense sonar pings with total sound exposure levels of 214 dB re: 1 µPa·s. Data support an increasing energy model to predict temporary noise-induced hearing loss and indicate that odontocete noise exposure effects bear trends similar to terrestrial mammals. Thus, sonar can induce physiological and behavioural effects in at least one species of odontocete; however, exposures must be of prolonged, high sound exposure levels to generate these effects.

Keywords: auditory; marine mammal; noise; stranding; navy; threshold shift

1. INTRODUCTION

Human use of the Earth’s oceans has steadily increased over the last century leading to an increase in anthropogenically produced noise (National Academy of Sciences 2003). This noise stems from a variety of sources including commercial shipping, oil drilling and exploration, scientific research and naval sonar. In terrestrial habitats, increasing sound levels have been shown to induce various effects across taxa including behavioural changes (Slabbekoorn & Peet 2003), temporary physiological alterations (Ward et al. 1959) and permanent anatomical damage (Kryter 1994). While it is apparent that anthropogenic noise may affect marine animals (Richardson et al. 1995), we know relatively less about the actual causes or mechanisms of these effects.

Marine mammals are of particular concern regarding the effects of noise as they typically have sensitive underwater hearing and they use sound for important activities such as communicating, orienting and finding prey. It has been suggested that overexposure to noise could induce permanent physiological damage and deleterious behavioural alterations (National Academy of Sciences 2003; Tyack et al. 2006). Within the past decade, and perhaps beyond, there have been multiple instances of cetacean mass strandings immediately following naval training activities involving tactical mid-frequency sonar (Balcomb & Claridge 2001; Evans et al. 2001; Fernandez et al. 2005). These temporally and spatially overlapping events seem to indicate that high-intensity sonar may instigate some marine mammal strandings. Recent work has suggested that sonar exposure could induce a variety of effects in marine mammals including changes in dive profile, acoustically induced bubble formation or decompression sickness (Jepson et al. 2003; Tyack et al. 2006). However, these hypotheses typically lack controlled experimental conditions to best evaluate potentially deleterious noise effects. Thus, the actual mechanisms that may be initiated by sonar exposure, which could actually result in multi-species strandings, have yet to be empirically supported.

We directly tested the possibility that sonar may temporarily affect odontocete (toothed whales and dolphins) auditory capabilities, and examined a model to predict the onset of these temporary hearing shifts induced by sonar and longer duration noise exposures.

2. MATERIAL AND METHODS

Experiments were conducted in open-water pens at the Hawaii Institute of Marine Biology, HI, in August–October 2007. The fatiguing noise in the exposure situation was an actual mid-frequency naval sonar signal recorded within the Puget Sound, WA, in the summer of 2005 before a marine mammal stranding event (figure 1; see figure 1 in the electronic supplementary material). Successive three-ping blocks, each block spaced 24 s apart to simulate a ‘typical’ mid-frequency sonar application (Evans et al. 2001), were presented to a captive-born, well-trained Atlantic bottlenose dolphin (Tursiops truncatus) accustomed to noise exposure experiments (Nachtigall et al. 2003, 2004). During exposures, the animal was stationed in a hoop at 1 m depth, 2 m from the transducer and behaviours were monitored by a trainer.

To evaluate temporary threshold shifts (TTS), hearing thresholds for a 5.6 kHz tone were measured before and after noise exposure using the physiological method of auditory evoked potentials (Supan et al. 2001). Pre-exposure thresholds ensured that the subject’s threshold was similar to its ‘baseline’ or average threshold. A threshold shift was considered when post-exposure thresholds exceeded +1 s.d. of the subject’s average threshold. We based all TTS measurements off the subject’s mean threshold because, between measurements, hearing thresholds often vary slightly (±2–3 dB). Data were also compared with hearing variation (Nachtigall et al. 2000) and prior threshold shift work on this and other odontocete species (e.g. Schlundt et al. 2000). Multiple post-exposure hearing thresholds were obtained to determine the magnitude of TTS as well as track the subject’s recovery. Sonar sound pressure levels (SPL; dB re: 1 µPa) were gradually increased up to 203 dB SPL root mean square (measured at the location of the dolphin’s ear) for individual pings. The ping number was then increased over multiple exposure sessions until a threshold shift was induced. These exposure conditions were then repeated. If a second shift was not induced, the number of pings was increased, resulting in higher sound exposure levels (SEL; dB re: 1 µPa·s). SEL equates to sound energy and allows for both sound magnitude and duration to be considered simultaneously. These levels were increased until significant TTSs were reliably induced (deemed three consecutive sessions with threshold shifts).
reliably induced shifts for three consecutive research sessions. Recovery back to the range of normal hearing typically (80%) occurred within 20 min after sonar exposure (n = 10) and always within 40 min. Control sessions, in which no sound exposure occurred, showed no changes in hearing thresholds.

Both the animal’s respirations per min (mean ± 1 s.d.; sonar: 6.95 ± 2.54; control: 5.38 ± 2.13) and the latency of time from surface station to noise exposure station (sonar: 8.60 ± 3.20; control: 5.42 ± 1.08) were significantly elevated during the sonar exposures (two-sample t-tests; p = 0.018; p < 0.001). While this animal previously demonstrated an uneasiness during noise exposure (Nachtigall et al. 2003), no dramatic differences in the animal’s behaviour were determined here, probably as a result of previous exposure, habituation and good training.

4. DISCUSSION
We compared the onset of threshold shift found in these data with that of other studies to devise a model to predict when sonar signals, as well as other types of noise, would affect odontocete hearing (Mooney et al. 2009). Previous studies suggest that odontocete TTS could be predicted by an equal-energy model of noise exposure (Finneran et al. 2005), which theorizes that, if noise SELs are constant, similar threshold shifts will be induced regardless of the exposure’s temporal pattern (Ward et al. 1959). However, this equal-energy hypothesis was not supported here because, as exposure duration decreased, increasing SELs were required to induce threshold shifts (figure 2b). This was true for various types of noise (broadband noise and frequency-modulated sonar pings), where exposure levels required to induce shifts increased linearly with log time.

Figure 1. (a) Spectrogram and (b) waveform of the mid-frequency sonar ping recorded off of San Juan Island, WA, and used in the experiment. The two downsweeps spaced by 0.5 s constitute one ping. Note the harmonics of the fundamental for each signal.

Figure 2. (a) Mean amount of TTS, for sonar (black bars; n = 7) and control (grey bars; n = 13) exposure sessions and s.d. Significant shifts, relative to baseline hearing thresholds, were found at 5 and 10 min after sonar exposure (one-way ANOVA, F5,60 = 8.29, p < 0.001; Tukey’s pairwise comparison). The dotted line represents +1 s.d. greater than the mean threshold. (b) Onset of TTS found after sonar (circled) and noise (Mooney et al. 2009) exposure. Hearing shifts may be predicted by: exposure duration (log-min) versus SEL (dB re: 1 µPa² s) and regression (F1,28 = 256.6, p < 0.001, y = −10.5×log(x) + 204.7, r² = 0.902). (c) Surface plot of measured and predicted dolphin TTS for sonar and noise exposures of varying exposure SPLs and durations using the equation of the line plotted in (b).

To establish a new model to predict noise exposure hearing shifts, we evaluated threshold shift onset and growth using data compiled from both sonar-ping and broadband-noise exposure experiments, where the fatiguing stimuli varied across a broad range of sound pressures (157–203 dB SPL) and exposure durations (30–0.25 min) (Mooney et al. 2009). Both the linear model and surface plot demonstrate that when using acoustic signals of approximately 0.5 s, which are typical for sonar sounds, SEL must be very high, at least 210–214 dB re: 1 µPa² s, to induce threshold shifts. These levels were higher than previous continuous-exposure experiments (Schlundt et al. 2000;
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Finneran et al. 2005), perhaps indicating individual differences or that intermittent exposures might require higher SELs than if continuous. The results do not preclude other noise or sonar-induced effects on marine mammals, which may occur at lower sound levels. However, a usual sound attenuation rate of 6 dB per doubling of distance (Urick 1983), the 203 dB level used in this experiment would be the received level approximately 40 m from the sonar source, a distance that can be considered ‘close’ with respect to naval ships. Second, the animal would then have to maintain at most that distance for the approximate 2–2.5 min of operating the sonar to receive SELs of near 214 dB. Alternatively, the animal could be located closer to the sonar source and receive a more intense signal. However, the animal would still need to remain within a close range long enough to receive the SELs that would induce auditory threshold shifts, a potentially unlikely situation.

Both scenarios entail the subject being relatively close to the sonar source for a ‘prolonged’ duration. Exceptions may be if the sonar signals are rapidly repeated (which is unlikely due to overlap of returning echoes) or if oceanographic conditions are such that sound levels do not attenuate regularly over short distances (i.e. less than several 100 m) and thus remain intense (Urick 1983; Evans et al. 2001). Perhaps such a situation could occur with multiple sonar sources over steep bathymetric conditions.

Our results demonstrate that mid-frequency sonar can induce at least temporary physiological hearing loss in odontocete cetaceans, although repeated exposures are necessary to generate effects. Furthermore, subtle behavioral changes are also associated with sonar exposure. In contrast to previous studies, we show that SEL must increase as noise duration decreases for continued shift onset. This agrees with terrestrial mammal and pinniped noise exposure results (Ward 1991; Kastak et al. 2005), suggesting similarities in auditory characteristics and substantiates potential extrapolations. While these data demonstrate that marine animals are susceptible to ‘adverse’ effects related to intense sonar exposure, it remains uncertain how frequently they are exposed to conditions in which TTS can occur.

All research was in accordance with the U.S. National Marine Fisheries Service permits and University of Hawaii’s IACUC guidelines and ethics policies.

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