Acoustic deterrent devices to prevent pinniped depredation: efficiency, conservation concerns and possible solutions

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Supplement 1: Sound exposure sensation levels (SEL$_{sens}$)

As a conservative approach, Southall et al. (2007) suggested an M weighting function to correct for frequency dependency of hearing damage. Their argument is that the weighting function is flat over large parts of the hearing range. However, this is only correct if the subjects used to derive temporary threshold shift (TTS) or permanent threshold shift (PTS) criteria had been tested within their most sensitive hearing range. However, most of the animals used to derive these criteria were tested at mid-frequencies (e.g. Finneran et al. 2005) where their hearing is less sensitive than at higher frequencies. Therefore, the use of the M weighting is not fully adequate as it does not correct for the fact that the test signals used to derive the noise exposure criteria may have been less harmful because they did not fall in the frequency range where odontocete hearing is most sensitive (see also Fig. 1 in main text). In addition, some test subjects had slightly compromised hearing. For example, one animal (Ben) had a hearing threshold of about 90 dB re 1 µPa at 3 kHz under quiet conditions (Finneran et al. 2005) while Johnson (1967) reported a threshold of 76 dB re 1 µPa at the same frequency for a bottlenose dolphin The same animals showed clear signs of permanent hearing damage in the frequency band between 10 and 40 kHz. More importantly, bottlenose dolphin hearing thresholds are much lower at higher frequencies compared to the test frequencies used in many TTS studies i.e. the lowest threshold measured in the ultrasonic range is 43 dB re 1 µPa (Johnson 1967). Thus, it is possible that a bottlenose dolphin that is exposed to signals at higher frequencies develops TTS at lower exposure levels than suggested by Southall et al. (2007). This has also been suggested in a recent study that demonstrated onset of TTS at 20 dB less when exposed to 20 kHz compared to 3 kHz (Finneran & Schlundt 2010). Finally, one needs to consider that other *odontocete* species appear to have somewhat lower hearing thresholds at the test frequencies (e.g. killer whales; see Fig. 1) than bottlenose dolphins and might therefore develop TTS at lower sound exposure levels. In spite of the fact that this data is only based on a few test subjects we believe that in the absence of additional data a more detailed categorization than the 'mid frequency odontocetes' proposed by Southall et al. (2007) should be considered.

   We suggest that exposure criteria should be referenced to the hearing sensitivity of the test subject instead, using sensation levels as a measure. Apart from the previously mentioned arguments this is also supported by the fact that in humans low-frequency noise (300–600 Hz) causes much lower levels of TTS compared to noise that falls in a frequency band between 2400–4800 Hz where hearing is more sensitive (Ward et al. 1959). The importance of sensation levels has been highlighted by studies on fish, birds, terrestrial mammals and humans which found a clear correlation between the amount of TTS and the sound pressure level difference between the baseline hearing threshold and the exposure level (Ward et al. 1959, Smith et al. 2004b). In marine mammals, Mooney et al. (2009a) reported a negative correlation between the sensation level that causes onset of TTS and exposure time indicating that TTS onset depends at least in part on sensation levels.

   We suggest to use SEL$_{sens}$ (sound exposure sensation levels) as a noise exposure criterion with units of dB re (hearing theshold of test subject in µPa)^2 s. Kastak et al. (2005) used a similar unit for comparative purposes and found that sensation levels could explain differences in the onset TTS levels across 3 different species and test subjects with slightly different hearing abilities. Applying SEL$_{sens}$ levels would be beneficial for several reasons: a) to compensate for potential differences in hearing sensitivity between test subjects (in TTS studies) and the average of a population, b) to compensate for differences in the audiograms of different species which are currently classed in the same category (Southall et al. 2007) and c) to allow for the fact that not all test subjects in TTS studies were tested at the frequencies of their most sensitive hearing.

SEL$_{sens}$ levels are a combination of 2 different concepts, the sound exposure level and the sensation level. Sound exposure levels (SEL) can be calculated by the following simplified equation (see Madsen 2005)

$$SEL = SPL_{rms} + 10\log_{10}(t)$$

where SPL$_{rms}$ is the root mean square (rms) sound pressure level of the signal the animal is exposed to and t is the exposure time of the signal. The sound pressure level is defined as

$$SPL = 20 \cdot \log(p/p_0)$$

where p is the sound pressure of the signal and p_0 is the reference sound pressure underwater of 1 µPa.
For the SEL$_{sens}$ level we suggest to replace the reference sound pressure level p_0 with the pressure p_h which is the pressure at the hearing threshold for the frequency of the sound and to use the received level p_{exp} at the receiver so that SEL$_{sens}$ is

$$SEL_{sens} = [20 \cdot \log(p_{exp}/p_h)] + 10\log(t)$$

Given the logarithmic nature of the dB scale the values obtained from this calculation are numerically identical to simply subtracting the hearing threshold (in SPL) from the sound exposure level that marks onset of TTS. We did not consider the duration of the test signals used to measure auditory thresholds in these calculations since there is no evidence that hearing thresholds (in contrast to TTS) depend crucially on the duration of test signal as long as the test signal exceeded the integration time of the auditory system (Au 1993). SEL$_{sens}$ can be used to arrive at (a) a noise exposure criterion that marks onset of TTS for untested species by assuming TTS occur at similar sensation levels in different species, (b) a noise exposure criterion for a signal that falls in an untested frequency range where the hearing threshold is known and (c) a noise exposure criterion for animals of the same species with lower hearing thresholds than the test subjects in which TTS was measured. This can be done by inserting the hearing threshold of the respective species into the 'sensation level-sound exposure levels' equation which is numerically identical to simply adding the new hearing threshold to the SEL$_{sens}$ criterion.
Finally, we also included a review of predictions that are based on extrapolation from humans (Taylor et al. 1997). While we are aware that researchers generally tend to call for caution when extrapolating information from human data (e.g. Southall et al. 2007) we believe that they should be mentioned for 3 reasons. First, there is a complete lack of data on how hearing damage develops in marine mammals as a result of exposure to noise for several months or years at moderate received levels. Second, in spite of the fact that some marine mammals have specific adaptations to aquatic hearing there is currently little evidence that the basic functioning of the cochlea is fundamentally different from terrestrial mammals. A closer look at the TTS data does in fact reveal that *odontocetes* do not seem to have a much larger dynamic range than humans. For example, Finneran et al. (2005) proposed that 1 s exposure to 195 dB re 1 µPa would cause onset of TTS. This value is only 110 dB above the hearing threshold of the test subject (approx. 85 dB re 1 µPa) and would therefore fall in the same order of magnitude as in humans. Third, we believe that comparisons to terrestrial mammals are helpful to understand the general problems raised in this review (see 'The problem of loudness perception' section). This can help to sharpen research questions and direct future research more towards answering conservation-related questions based on general biological concepts rather than specific ideas prominent within marine mammal biology.

Supplement 2: Calculation of the zones for hearing damage

General considerations

In the following sections we try to answer the question whether acoustic deterrent devices (ADDs) are likely to cause hearing damage in marine mammals using current physiological data. Such an attempt inevitably requires a variety of different assumptions to be made. In order to predict impact zones, sound propagation needs to be modelled, which can be difficult in shallow water habitats. Shapiro et al. (2009) measured sound propagation in a range of ADDs and showed that that there can be considerable variation in received levels (up to 15 dB) at locations separated by less than a metre. Shapiro et al. (2009) conclude that this challenges the concept of concentric impact zones and animals might not be able to show a directional avoidance response. However, one also needs to consider that there is empirical evidence for both directed avoidance behaviour (Johnson 2002) and directed approach responses (Mate & Harvey 1987) in marine mammals living in shallow water habitats. Also, Shapiro et al. (2009) found that on average received levels dropped with increasing distance and received levels tended to be lower than predicted by spherical spreading. We therefore believe that in the absence of sufficient data using a simple spherical spreading model will provide a reasonably conservative approach.

As previously mentioned the 'equal energy hypothesis' has recently been challenged by a study that showed that disproportionally higher SELs are required to cause TTS when exposure times are short (Mooney et al. 2009a,b). Similarly, Finneran et al. (2010a) showed that while TTS can be predicted by using sound exposure levels for short exposure time a better fit to the empirical data can be achieved by applying a more complicated model that treats exposure time and sound pressure level as independent variables. In spite of these recent advances in our understanding of TTS we feel that data on TTS models is still scarce and we therefore based the following analysis on simple SELs as initially suggested by Southall et al. (2007).

Another aspect which needs to be taken into account is that commercial ADDs differ remarkably in their pulse emission patterns, pulse lengths, duty cycles and source levels. Furthermore, some companies have produced various models which differ in their emission patterns or offer user-selectable duty cycles. To take these differences into account we calculated impact zones for the effects caused by 2 different cumulative sound exposure levels: 203 dB re 1 µPa^2 s and 221.6 dB re 1 µPa^2 s. These 2 SEL reflect continuous exposure to a level of 193 re 1 µPa (SL of the Ace-Aquatec & ~Airmar) for 10 s and 12 min respectively. The actual exposure times needed to reach these cumulative SELs when exposed to ADDs are much larger because of quiet periods between emissions and the short duration of pulses within a pulse train (emission). Therefore, we calculated, for each device the 'effective duty cycle' i.e. the time during which a sound is actually produced taking intervals between pulses and emissions as well as pulse lengths into account. This 'effective duty cycle' is different from the 'emission duty cycle' mentioned in Table 1 which refers to the time during which a pulse train or emission is produced. The effective duty cycle was then used to calculate how long each device will have to operate at its given source level to reach this cumulative SEL. This exposure time was calculated as

$$\text{Time [s]} = \frac{10^{\left[\frac{\text{SEL} - \text{SL}}{10}\right]}}{\text{dc}}$$

where SEL is the sound exposure level, SL the source level of the device and dc the effective duty cycle as a fraction of 1 (=100%).

The following average numbers (derived from the information provided in Table 1) were used in this calculation:

	Source level (dB re 1 µ Pa) rms	Average pulse length (ms)	Average pulse interval within emission	Emission duty cycle (% time emission)	Effective duty cycle: (% time sound)
Ace-Aquatec	193	8.65 ms	40.85 ms	10% 30% (3 transd.)	2.11%, 10.58%
Airmar	192 & 198	1.4 ms	40 ms	50% 200% (4 transd.)	1.75% 7%
Lofitech	182	500 ms	NA (no trains)	12%, 25%	12%, 25%
Terecos	178	4100 ms	NA (continuous)	11%	11%

Marine mammals: temporary threshold shifts (SEL 203 dB re 1 µPa^2 s)

Mooney et al. (2009b) showed that a bottlenose dolphin exposed to series of 500 ms long double pulses (sonar pings) only developed TTS at sound exposure levels (SEL) of 214 dB re 1 µPa^2 s. Finneran et al. (2005) reported levels of around 195 dB re 1 µPa^2 s to be sufficient to cause TTS when the animals were exposed to pulses of 1–8 s duration. Currently available seal scarers produce either pulse trains or continuous emissions of an overall length from 0.5–20 s (see Table 1). Using Finneran et al. (2005) TTS criterion of 195 dB re 1 µPa^2 s and assuming exposure to noise from an ADD reflecting a cumulative SEL of 203 dB re 1 µPa^2 s the difference between the sound exposure level causing onset of TTS and the sound exposure level of the ADD would be 8 dB. Assuming spherical spreading, TTS in bottlenose dolphins would be caused at about 2.5 m distance from a device. However, data on harbour porpoises suggested that SELs as low as 164 dB re 1 µPa^2 s can cause TTS (Lucke et al. 2009). The signals tested by Lucke et al. (2009) were short transients with broad spectra with most energy below the most sensitive hearing range of the harbour porpoise. Assuming spherical spreading, absorption losses of about 0.7 dB per km for a 10 kHz signal (Fisher & Simmons, 1977, 12°C water temperature) and ADD noise reflecting a cumulative SEL of 203 dB re 1 µPa^2 s, TTS would be caused in porpoise closer than around 89 m to the transducer.

As we argue in Supplement 1 there is a possibility that TTS maybe caused at lower SELs at frequencies where hearing is more sensitive (see Supplement 1 for justification of the calculations). We therefore provide alternative impact zones based on an approach that takes the hearing sensitivity of the test subject from which TTS data was derived and the hearing threshold of the target species within the respective frequency band into account ('sound exposure sensation levels (SEL$_{sens}$), see Supplement 1 for justification of the calculations). This approach seems justified given that TTS is caused by lower sound pressure levels in a frequency range where dolphin hearing is more sensitive (Finneran & Schlundt 2010). Studies on odontocetes generally found SELs between 193 and 214 dB re 1 µPa^2 s to cause mild to moderate but fully recoverable TTS (Finneran et al. 2000, Schlundt et al. 2000, Finneran et al. 2002, Nachtigall et al. 2004, Finneran et al. 2005, Mooney et al. 2009 a,b, Finneran et al. 2010a,b). Finneran et al. (2005) suggested an onset-TTS criterion of 195 re 1 µPa^2 s while the test subjects hearing threshold was between 80 dB re 1 µPa (rms) and 90 dB re 1 µPa (rms) at the exposure frequency under quiet conditions (see Fig. 3 in Finneran et al. 2005). Mooney et al. (2009a) found an onset of TTS at an SEL of 198 dB re 1 µPa^2 s for exposures in the range of 2min. Their subjects' hearing threshold was about 95 dB re 1 µPa (rms) at the test frequencies. Summarising these data we find that SEL$_{sens}$ levels between 100–130 dB re re (hearing threshold in µPa)^2 s are likely to result in onset of TTS. We therefore used an average value of 115 dB re (hearing threshold in µPa)^2 s for the following calculations. The sound exposure levels re 1 µPa^2 s that cause onset of TTS can then be calculated by adding the most sensitive hearing threshold in dB re 1 µPa within the relevant frequency range (10–35 kHz). These hearing thresholds were 37 dB re 1 µPa (rms) for the harbour porpoise (Kastelein et al. 2002), 43 dB re 1 µPa (rms) for the bottlenose dolphins (Johnson 1967), and 30 dB re 1 µPa (rms) for killer whales (Hall & Johnson 1971). The respective onset-TTS sound exposure levels would then be 152 dB re 1 µPa^2 s for the harbour porpoise, 158 dB re 1 µPa^2 s for the bottlenose dolphin, and 145 dB re 1 µPa^2 s for the killer whale. Assuming spherical spreading and absorption loss of 0.7 dB per km (Fisher & Simmons, 1977, for a 10 kHz signal), exposure to a 10 s continuous ADD signal of 10 kHz at 193 dB re 1 µPa (rms) (SEL =

203 dB re 1 μPa^2 s) would therefore result in TTS zones of 345 m for the harbour porpoises, 175 m for the bottlenose dolphins and over 748 m for killer whales.

The lowest SEL causing TTS in a pinniped was found to be 183 dB re 1 μPa^2 s for the harbour seal (Kastak et al. 2005). Assuming spherical spreading in the vicinity of the device temporary threshold shifts in a harbour seal would occur up to a distance of 10 m from the seal scarer. In the harbour seal, SEL has been measured directly at 1–4 kHz (Kastak et al. 2005). Since this was in the most sensitive hearing range of the species (see Kastelein et al. 2009) and since the harbour seal audiogram is flat over a large frequency range (Kastelein et al. 2009) referencing the directly measured sound exposure level to the auditory threshold in the frequency range where ADDs operate would not change the results. Therefore, no SEL_{sens} calculations were carried for the pinnipeds.

Marine mammals: permanent threshold shift (PTS)

The noise exposure criteria published by Southall et al. (2007) would suggest permanent injury in most *odontocetes* at SELs of 198 dB re 1 μPa^2 s for multiple pulses and 215 dB re 1 μPa^2 s for continuous noise. As most seal scarers emit pulse trains (e.g. Airmar; see Table 1) we used the criterion for multiple pulses for our calculations. When calculating impact zones based on the same assumptions mentioned in the previous section this would mean that a cumulative sound exposure levels of 203 dB re 1 μPa^2 s would only cause hearing damage in *odontocetes* if an animal is closer than 2m to the sound source. Exposure to a cumulative sound exposure level of 221.6 dB re 1 μPa^2 s would result in an impact zone for *odontocetes* of up to 15 m.

Lucke et al. (2009) found that onset of TTS in harbour porpoises may occur at sound exposure levels of 164 dB re 1 μPa^2 s. Data on humans showed that threshold shifts of about 40 dB are correlated with an increase of the exposure level by approximately 20 dB (Ward et al. 1958). Therefore, one might attempt to derive at a noise exposure criterion for PTS by adding 20 dB to the onset TTS criterion (see Southall et al. 2007). Based on the assumption that permanent hearing damage may occur at levels 20 dB higher than those that cause onset of TTS permanent hearing would occur at SELs of 184 dB re 1 μPa^2 s. Assuming spherical spreading and previously mentioned absorption losses (0.7 dB/km), exposure to a cumulative SEL of 203 re 1 μPa^2 s would be only sufficient to damage hearing at distances of up to 9 m. However, exposure to ADD noise reflecting a cumulative SEL of 221.6 dB re 1 μPa^2 s would result in a damage zone of 76 m.

Alternatively, one might try to calculate damage zones using the SEL_{sens} approach (Supplement 1). If we add the 20 dB increase in SPL needed to cause 40 dB TTS to the SEL_{sens} that causes onset TTS in bottlenose dolphins (115 dB re (hearing threshold in $\mu Pa)^2$ s), then permanent threshold shifts would occur at 135 dB re (hearing threshold in $\mu Pa)^2$ s. The auditory thresholds of the respective species are 37 dB re 1 μPa for the harbour porpoise (Kastelein et al. 2002), 43 dB re 1 μPa for the bottlenose dolphins (Johnson 1967), and 30 dB re 1 μPa for killer whales (Hall & Johnson 1971). Inserting these thresholds we arrive at SEL damage criteria of 165 dB re 1 μPa^2 s for killer whales, 172 dB re 1 μPa^2 s for harbour porpoises, and 178 dB re 1 μPa^2 s for bottlenose dolphins. Assuming spherical spreading and absorption of 0.7 dB per km (10 kHz signal) a cumulative sound exposure level of 203 dB re 1 μPa^2 s would cause PTS at distances up to 18 m, 35 m and 79 m for the bottlenose dolphin, harbour porpoise and killer whale respectively. The scenario based on the higher SEL of 221.6 dB re 1 μPa^2 s would result in larger impact zones of 150 m (bottlenose dolphin), 295 m (harbour porpoise) and 642 m (killer whale)

Using the Southall et al. (2007) criterion for pinnipeds (multiple pulses: 186 re 1 μPa^2 s), the cumulative SEL of 203 dB re 1 μPa^2 s would cause a damage zone of 7 m (assuming spherical spreading) while the higher SEL (221.6 dB re 1 μPa^2 s) would result in a larger impact zone of 60 m.

Marine mammals: extrapolations from human data and long-term exposure scenarios

There is no data available on hearing damage in marine mammals as a result of exposure for several months or years at moderate to low source levels. Therefore, predictions have been based on human damage criteria. Gordon & Northridge (2002) used Kryter's (1994) damage threshold of 115 dB above the hearing threshold. They yielded PTS ranges between 79 m and 562 m for a high power device (200 dB re 1 μPa @ 1 m) and values between 40 and 281 m for a 194 dB re 1 μPa ADD depending on the species' hearing threshold. In humans, noise levels at industrial workplaces should not exceed 85 dB above the hearing threshold (NIOSH 1998). Taylor et al. (1997) applied an even more conservative threshold of 80 dB above the hearing to marine mammals in the context of ADDs. The zone where this value is exceeded would be over a kilometre for most ADDs. In areas with dense fish farming, animals could potentially be exposed to such levels for an extensive amount of time. However, it should be noted that there is no empirical basis for such extrapolation to marine mammals and hence the results should be interpreted with caution.

ADD effects on other marine life

The literature provides less data on the effects of underwater noise on hearing in fish than it does in mammals (Popper & Hasting 2009). Assessing hearing damage in fish is complicated by the fact that auditory sensitivity and hearing ranges vary dramatically across species. While some species evolved specific adaptations which result in more sensitive hearing over a broader frequency range other species have much more restricted hearing abilities (Popper & Hasting 2009). Temporary threshold shift and hair cell damage have only been demonstrated in a limited number of species (Popper & Hasting 2009). Hair cell damage has been found in cod exposed to a 400 Hz signal sound pressure levels of 180 dB re 1 μPa for several hours (Enger 1981). Similarly, hair cell damage was found in oscars *Astronotus ocellatus* that were exposed to 300 Hz sine wave sounds of the same source levels (Hastings et al. 1996) and snappers that were repeatedly exposed to airgun emissions (received levels up to 180 dB re 1 μPa, peak frequencies between 20–100 Hz) used for seismic surveys (McCauley et al. 2003). The signals used in these studies had most energy concentrated in a frequency range where the tested fish species were most sensitive. In contrast, very few ADDs emit any significant amount of energy at frequencies below 2–3 kHz making it unlikely that ADDs could cause similar effects. For instance, the signal component with the lowest frequency emitted by an Ace-Aquatec ADD has a frequency of 6 kHz and a source level of only 178 dB re 1 μPa (Lepper et al. 2004). This is well below the received levels found to cause hair cell damage in all previously mentioned studies.

Temporary threshold shift has been most thoroughly documented in freshwater *cyprinids* which possess excellent hearing abilities. Goldfish developed weak temporary threshold shift after just 10 min of sound exposure to white noise at received levels of 170 dB re 1 μPa (Smith et al. 2004a). Fathead minnows (*Pimephales promelas*) exposed to band-limited noise (0.3 and 2 kHz) for 1 hour exhibited TTS at even lower received levels of only 142 dB re 1 μPa (Scholik & Yan 2001). While these levels were rather low, both studies used broadband noise with significant energy within the main hearing range of the species which is below the typical frequency range of ADDs. Smith et al. (2004b) showed that the sound pressure level difference (sensation levels) between the baseline hearing threshold and levels that cause onset of TTS are a good predictor for the amount of TTS caused. Their data can be used for a rough estimate of the risk that ADDs may pose: sensation level of at about 68 dB would be required to cause significant TTS in the order of 15 dB. The auditory sensitivity of herring at a frequency of about 4 kHz is 136 dB re 1 μPa (Enger 1967) while a 4 kHz segment of a Terecos ADD has a source level of 166 dB re 1 μPa (Lepper et al. 2004). The maximum sensation level caused by the Terecos device would therefore only be 30 dB and hence no such TTS is likely to be caused. In addition the experiment by Smith et al. (2004b) involved noise exposure for 24 h while ADDs usually only produce short bursts of pulses at low frequencies which are then followed by a pause or emissions at even higher frequencies. Similarly, sensation levels would be low in fish with ultrasonic hearing (e.g. shad *Alosa sappidissima,* Mann et al. 1997): the absolute sensitivity of shad at a frequency of 40 kHz is 141 dB re 1 μPa (Mann et al. 1997) while an Ace-Aquatec ADD produces a source level of about 165 dB at 30 kHz (Lepper et al. 2004). Hence, even in the most conservative scenario, assuming maximum overlap between the most sensitive hearing range of shad and the spectrum produced by an Ace-Aquatec ADD, the sensation level would only be 24 dB. The only scenario in which a sensation level caused by ADDs would be somewhat higher is if a marine fish species existed with an absolute hearing sensitivity similar to that of a freshwater cyprinid (i.e. goldfish, minnow). The auditory threshold of goldfish is about 116 dB at 2.5 kHz (Fay 1969) and hence sensation levels caused by a 4 kHz segment of a Terecos ADD would be in the order of 50 dB. Farmed fish species like salmon are unlikely to be affected by ADDs since they are insensitive to frequencies above 1 kHz (see Fig. 1). This has also been confirmed by empirical studies which found no evidence for behavioural responses or any influence on egg fertility at frequencies above 800 Hz (Mate et al. 1987)

Thus, while marine fish species with high-frequency hearing abilities (e.g. *clupeids*) are generally more likely to be affected by ADDs, sensation levels and absolute sound pressure levels inflicted by ADDs are probably insufficient to cause hair cell damage. Similarly, sensation levels in species with ultrasonic hearing would probably be insufficient to cause damage but no empirical TTS data is available for these species. Finally, it is also important to note that in contrast to mammals hair cells in fish can re-grow after acoustic trauma and depending on the exposure scenario damage caused might in some cases only be temporary (Corwin 1981, Popper & Hoxter 1984, Lombarte et al. 1993). However, this does not mean that hearing damage is never of any concern in fish since temporary threshold shifts may have a fitness consequence.

The effect of noise on hearing in reptiles and invertebrates is poorly understood but will depend on the ability of these animals to detect sound. Sea turtles seem to be primarily sensitive to low-frequency sound below 1 kHz (Bartol et al. 1999). Invertebrate detection of vibration stimuli is primarily low-frequency and mostly limited to the particle motion component of the signals. The lateral line of some cephalopods is

sensitive to water movement stimuli up to 100 Hz (Budelmann & Bleckmann 1988). Early studies showed that the statocysts of cephalopods are sensitive to about 100Hz (Packard et al. 1990) while more recent studies revealed sensitivity to the particle motion component of sound up to 500 Hz (Mooney et al. 2010). The fact that auditory sensitivity at high frequencies is low or non-existent is also supported by a study that showed cephalopods exhibiting no avoidance responses to simulated high intensity ultrasonic odontocete clicks (Wilson et al. 2007). This suggests that the effects of noise on some of these species may be similar to that on fish with no specialised hearing and hence ADDs are unlikely to affect these animals.

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