REVIEW PAPER
The effects of anthropogenic sources of sound on fishes

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There is increasing concern about the effects of pile driving and other anthropogenic (human-generated) sound on fishes. Although there is a growing body of reports examining this issue, little of the work is found in the peer-reviewed literature. This review critically examines both the peer-reviewed and ‘grey’ literature, with the goal of determining what is known and not known about effects on fish. A companion piece provides an analysis of the available data and applies it to estimate noise exposure criteria for pile driving and other impulsive sounds. The critical literature review concludes that very little is known about effects of pile driving and other anthropogenic sounds on fishes, and that it is not yet possible to extrapolate from one experiment to other signal parameters of the same sound, to other types of sounds, to other effects, or to other species.

Key words: blasts; impulsive noise; noise effects; ocean noise; seismic air guns; sonar.

INTRODUCTION
It has become increasingly apparent that anthropogenic (human-generated) sound has the potential to affect the health and wellbeing of animals as well as humans. There is also an increasing awareness of the presence of anthropogenic sounds in the aquatic environment and concern has arisen that these sounds could affect aquatic mammals, diving birds, fishes, amphibians, reptiles and perhaps even invertebrates (e.g. NRC, 1994, 2000, 2003, 2005; Richardson et al., 1995; Popper, 2003; Popper et al., 2004; Hastings, 2008; Popper & Hastings, 2009).

Despite the concerns raised by the increased presence of anthropogenic sound in the aquatic environment, very little is known about the effects of exposure to such sounds on marine mammals (Southall et al., 2007; Hastings, 2008), and far less is known about the effects on fishes (see reviews in NRC, 1994, 2000, 2003; Popper, 2003, 2006; Popper et al., 2004; Hastings, 2008; Popper & Hastings, 2009). One must always be cautious when extrapolating outside the bounds of empirical data, and because data available for the effects of sound on fishes are so few, extra caution...
is advised when attempting to extrapolate between fish species, even for identical stimuli. Moreover, one must also be cautious with any attempt to extrapolate results between stimuli because the characteristics of the sources (e.g. air guns, sonars, ships, pile driving) differ significantly from one another.

For the purpose of this review, exposure to sound is defined to include a measure of both the received level and the duration of the signal. For example, the received level can be expressed in terms of acoustic pressure, particle velocity, or intensity (i.e. instantaneous or time-averaged energy flow through a unit area; also called energy flux), which all vary with time over the duration of the signal. Sound exposure metrics usually convey characteristics of the received level and, or the time sequence of a single acoustic event. The biological effects of multiple events on an animal or parts of an animal primarily depend on the degree of effect caused by a single event, recovery of the affected region during the time interval between events and the total number of events. If a single sound exposure causes no effect whatsoever, then it is highly likely that no effect will occur for repeated multiple exposures. However, assessment must be made at the cellular or subcellular level by a fish pathologist to determine if a part of the body is affected.

The goal of this review has been to describe what is known about the effects of anthropogenic sound on fishes based on the literature and to identify studies needed to address areas of uncertainty relative to measurement of exposure to sound and the response of fishes. It should be noted that some emphasis is placed on the effects of pile driving on fishes. This was done because (1) pile driving is a major international issue of concern today, especially in shallow waters where the majority of freshwater and marine fish reside; (2) pile driving is the only anthropogenic sound source other than explosives that has caused fish kills in the wild that have been documented in the literature; (3) the original development of this review was supported by concerns specifically about pile driving; and (4) a companion review (Hastings & Popper, 2005) provides a more detailed evaluation of specific damage to fish as it relates to impulsive type sounds, including pile driving.

On the basis of available information in the literature, this review describes the potential of anthropogenic sound to affect fishes. It also sets the stage for future studies by outlining what is known about detection and reception of acoustic signals by fishes and the effects of exposure to sound on various species of fishes. At the same time, it must be noted that too little is known about the effects on fish from exposure to sound to allow for definitive conclusions to be drawn from the literature. A companion review (Hastings & Popper, 2005) analyses the characteristics of impulsive sounds to quantify the interactions between these sounds and fishes in an effort to predict potential effects from pile driving using data in the literature for other impulsive sources.

One issue in trying to understand what is currently known about effects of sound on fish is that much of the material is in technical reports (grey literature) and has not necessarily gone through the scientific peer-review process. While some of this grey literature has significant scientific and methodological problems, it is critical to include it in this review since it makes up so much of what is discussed and actually used in estimating effects of sound on fishes, and in making technical and regulatory decisions. This review is based on evaluation of these grey literature reports as well as on peer-reviewed articles. It is also important to note that the approach and analysis in each study reviewed differs, and so extrapolation between studies, and
especially those done in different locations or by different groups of investigators, is difficult. To help resolve the problems in using the grey literature reports, the authors of this review examined the potentially useful material and have based this review on their opinion of the quality of the science and the validity of the conclusions in these studies.

In addition to primary peer-reviewed literature and grey literature reports, this review also includes citations to a number of reviews and overviews of various aspects of the material presented here. It must be recognized that the earlier reviews, even if they have gone through appropriate peer review, are often the opinions of their authors and may be based on the analysis of material from peer-reviewed articles and, or the grey literature.

PILE-DRIVING SOUND AND ITS EFFECT ON FISHES

Pile driving commonly occurs in shallow water and is related to construction and repair of bridges, docks and other over-water infrastructure, as well as to construction of offshore wind farms, liquid natural gas (LNG) ports and general harbor construction. However, despite pile driving being found throughout the world, very little, if anything, is known about (1) particular characteristics of a pile-driving signal that are responsible for different observed effects and (2) the differential in effects that can be attributed to differences in signals generated by different types of piles (e.g, concrete v. steel). Thus, the emphasis of this part of the review is on what is known about effects related to sound received by fishes, and not on the sounds emitted by any particular type of pile or pile-driving operation (see Illingworth & Rodkin, 2001 for some discussion of the acoustics of piles).

OVERVIEW OF PILE-DRIVING SOUND

Pile-driving sounds are discussed in detail in the companion review (Hastings and Popper, 2005). Briefly, however, the sounds from pile driving result from a rapid release of energy when two objects hit one another. The characteristics of impact sounds depend primarily on the physical properties of the impacting objects. When a pile-driving hammer strikes a pile, sound from the impact radiates into the air and a transient stress wave, or pulse, propagates down the length of the pile. The impact will also create flexural (or transverse) stress waves in the wall of the pile which couple with the surrounding fluids (air and water) to radiate sound into the water and additional sound into the air.

Moreover, the pulse propagating down the length of the pile may couple to the substrate at the water bottom and cause waves to propagate outward through the bottom sediment. These transient waves in the substrate can be transmitted from the bottom into the water at some distance away from the pile to create localized areas of very low and, or very high sound pressure and acoustic particle motion because of interference with the sound pulse directly from the pile that is traveling outward through the water. Thus it is possible that the received levels of sound could be higher at some locations farther from the pile than at locations closer to it. This has been observed in some monitoring data (Caltrans, 2001).
Typically, pile-driving sounds underwater are characterized by multiple rapid increases and decreases in sound pressure over a very short period of time. The peak pressure is the highest absolute value of the measured waveform, and can be a negative or positive pressure peak.

OVERVIEW OF RESULTS FROM RECENT PILE-DRIVING STUDIES

There are seven recent experimental studies that have examined the effects of pile driving on fish (Caltrans, 2001, 2004; Abbott & Bing-Sawyer, 2002; Nedwell et al., 2003, 2006; Abbott et al., 2005; Ruggerone et al., 2008). This section provides a brief critical overview of these studies as background to the sections that follow.

CALTRANS (2001)

Caltrans (2001) involved examining fish that died during exposure to underwater sound from pile driving operations, as well as assessing gross physical damage to shiner surfperch *Cymatogaster aggregata* Gibbons held in cages at different distances from the pile driving source, although these results were listed as being very preliminary.

Results indicate that there was mortality caused by exposure to pile-driving sounds, with dead fish of several different species found within at least 50 m from the pile being driven. There was also an increase in catch by overflying gulls during pile driving, further indicating fish mortality. Dead and dying fish showed a number of forms of injury including bleeding and damage to the swim bladder, although it should be noted that the examination of fish tissue and pathology was not done by an expert fish pathologist. Numbers were relatively low, reflecting difficulty in retrieving dead or dying fish and the possibility that fish did not come to the surface at all, or not until they were away from the collecting operation.

The extent of damage and mortality of caged fish was generally greater for the cages closer to the source than farther away; however, there was significant variability between experiments with the type of hammer used, the duration of exposure and distance of the fish cages from the source. Signal levels at the test cages were not measured, and since there is a potential contribution from energy that has travelled through the substrate interacting with the waterborne signal, it is not clear that sound levels decreased with increasing distance from the pile. That, along with insufficient numbers of exposures or animals, makes it difficult to reach firm conclusions.

ABBOTT & BING-SAWYER (2002)

Abbott & Bing-Sawyer (2002) investigated the effects of pile driving on Sacramento blackfish *Orthodon microlepidotus* (Ayres). The authors reported significant problems with the study in that there were many uncontrolled factors as well as inadequate sample size to allow any definitive conclusions from the work. Sound levels at the test cages were not measured and there was potential acoustic interference in the water that may have affected sound levels so that they could not be predicted based on simple transmission-loss models from a site of known source level.
Orthodon microlepidopterus were placed in cages 45–850 m from a pile that was struck 43 times with the presence of an air bubble curtain around the pile (used to reduce sound transmission) followed by 45 additional strikes without the bubble curtain. Measurements of received sound levels at the location of the fish were not made. At the end of the exposure, fish were removed from the cages, observed for injuries and abnormal behaviour for 5 h, and then placed in plastic bags and frozen on ice. Subsequently, gross pathology was done on each fish to determine any external or internal damage resulting from exposure to the pile driving. There were no controls for tissue artefacts from freezing and thawing, making interpretation of any such material difficult, at best.

Although results of the necropsy suggested that there was more damage to fish that were closer to the source than further away, there was substantial interanimal variation in damage even within the same cage. While the authors concluded that damage was found only in fish subjected to sounds having a peak level of 193 dB re 1 μPa and that there was no damage to fish exposed to sounds with peak levels below 183 dB re 1 μPa, it must be emphasized that sound levels were extrapolated and not actually measured at the cages. Accordingly, these conclusions are not well supported, and the lack of actual measured sound levels and problems with controls and necropsy methods that involved freezing and thawing of fish confounded interpretation of the injury results.

No behavioural effects were seen prior to sacrifice of the fish, although the authors indicate that they did not have suitable facilities in which to observe behaviour. There were no deaths of any animals at the time of removal from the cages or in the subsequent 5 h period prior to sacrifice for necropsy.

NEDWELL ET AL. (2003)

Nedwell et al. (2003) examined the effects on caged brown trout Salmo trutta L. in response to pile driving at the Red Funnell Southampton ferry terminal, U.K. Caged fish were placed at distances of 25–400 m from the piles being driven, with a control cage 10 km away. Animals were observed by closed circuit TV as they were exposed to pile-driving sounds. The sound levels at the various cages were not given, though the authors estimated that the level of the pile driving signal was 134 dB re 1 μPa (peak) at 400 m from the pile.

Behavioural results indicated no reaction to vibropiling (i.e. where a pile is vibrated rather than hit with a hammer) for fish as close as 25 m to the source. The behaviour of animals and fish injuries in response to hammer pile operations were reported only for fish in cages located 400 m from the source. These fish showed no apparent behavioural response to the pile driving and no injuries were observed. Data were not, however, provided for fish closer than 400 m to the pile driving operation.

CALTRANS (2004)

Caged fish monitoring was conducted during the San Francisco–Oakland Bay Bridge East Span seismic safety project using caged C. aggregata and rainbow trout Oncorhynchus mykiss (Walbaum). Fish were exposed to pile-driving sounds at distances from 23 to 314 m from the pile-driving operation, with exposure durations of 1–20 min. Controls included fish that were placed in test cages and in the same
locations as the test animals for 3–10 min but without exposure to pile driving (this was a shorter time period underwater than for fish exposed to pile driving) and fish that were never placed in cages and either transported to the test site (transport controls) or kept in the laboratory.

*Oncorhynchus mykiss* were observed behaviourally following exposure and then held for 48 h to monitor survival. They were then sacrificed by placing them in a plastic bag and freezing, a method that is likely to produce tissue damage due to freezing and thawing. Fish were rapidly thawed for necropsy, which was done without involvement of someone with expertise in fish pathology. A number of fish died before the end of the 48 h holding period, though it is not clear that these mortalities were associated with pile driving. The authors reported that all control animals had the same low level of trauma. They also reported more trauma in animals exposed to pile driving and lesser levels of trauma in animals exposed to pile driving in the presence of an air bubble curtain. However, no statistical analysis could be done between *O. mykiss* exposed to pile driving with and without the presence of the air bubble curtain because of the small sample size of fish exposed with bubble curtains present.

**ABBOTT ET AL. (2005)**

This study investigated the effects of pile driving on caged fish of three species, *C. aggregata*, Chinook salmon *Oncorhynchus tshawytscha* (Walbaum) and northern anchovy *Engraulis mordax* Girard, at the Port of Oakland. The fish were caged and lowered to about 7.62 m at a distance of 9.75 m from the pile being driven. The fish were exposed to 4 min of pile driving (over 200 sound pulses) of 0.61 m diameter jetted concrete piles driven with a diesel-assisted hammer. Following exposure, fish were returned to the surface, mortality determined, behaviour observed for 1 min and then sacrificed using excellent pathology methodology. Controls were treated in the same way as experimental animals and pathology was analysed without the investigator knowing the treatment group of each animal examined.

The results showed no differences in mortality or pathology between sound-exposed and control animals. The investigators suggested that there were also no behavioural differences between sound-exposed and control animals, but these were based on behavioural analysis after the fish had been removed from the test cages and it is impossible to ascertain whether there were behavioural effects during sound exposure. Moreover, no data were provided on how the behavioural analysis was performed.

**NEDWELL ET AL. (2006)**

Nedwell and colleagues examined the effects of impact and vibropiling on *S. trutta* in Southampton Water, an estuary in the south coast of England. Fish were caged at different distances from the pile driving operations. Piles were either 50.8 or 91.4 cm in diameter. The investigators found no mortality or indications of any external tissue damage on any fish. There were no investigations of internal anatomy other than of the inner ear, and the investigators reported no damage to sensory hair cells. Source levels for impact piling were 193 dB *re* 1 μPa peak at 1 m for the smaller diameter piles and 201 dB *re* 1 Pa peak at 1 m for the larger piles. Source levels were not given for the vibropiling. Moreover, the authors did not measure...
sound levels at the cages, but reported that they determined a transmission loss of 0·13 dB m⁻¹ for both sources. Since the closest cage was 30 m from the source, the sound level at this cage therefore was about 189 dB re 1 μPa for the smaller piles and 198 dB re 1 μPa for the larger piles. Fish were exposed to about 200 min of pile driving total, with no information given on interstrike intervals or the duration of quiet as the drivers were moved between piles.

**RUGGERONE ET AL. (2008)**

Ruggerone and colleagues investigated the effects of pile-driving exposure on caged yearling coho salmon *Oncorhynchus kisutch* (Walbaum). Fish were placed in cages near (1·8–6·7 m) and distant (15 m) to 14 hollow steel piles (0·51 m diameter), and exposed to sound from 1627 strikes over a 4·3 h period. Sound levels were measured in both the near and far cages. In the near cage, peak sound pressure levels (SPL) reached 208 dB re 1 μPa and sound exposure levels (SEL) reached 179 dB re 1 μPa²-s, leading to a cumulative SEL of approximately 207 dB re 1 μPa²-s during the 4·3 h period. (SEL is simply the integration over time of the square of the acoustic pressure. It is an indication of the total acoustic energy received by an organism.) Sounds did not exceed ambient in control cages that were kept far away from the region of pile driving.

Caged fish were sampled at 10 and 19 days post exposure. The investigators found no mortality in any animals, and examination of the external and internal anatomy (gross observations and not histopathology) showed no differences between exposed and control animals.

While the authors saw no significant changes in behaviour during pile driving, as observed with closed circuit TV, it is important to note that these observations have little bearing on how animals would respond in their natural habitats. Thus, while this is, overall, an excellent study, it highlights the continuing need to develop methods to investigate behaviour in free-swimming animals during pile-driving operations.

**THE ROLE OF SOUND IN FISH BIOLOGY**

Since the purpose of this review is to summarize effects of pile driving and other anthropogenic sounds on fish, only a brief introduction to fish bioacoustics will be provided to set the scene for material presented later in this review and the companion article (Hastings & Popper, 2005). There are several reviews that can provide further background on fish bioacoustics including papers in Webb *et al.* (2008a) and Fay & Popper (1999). In particular, sound production and communication is discussed by Tavolga (1971); Myrberg (1980); Zelick *et al.* (1999) and Bass & Ladich (2008). Bass & Clarke (2003) discuss the underwater acoustic environment, and Rogers & Cox (1988) provide an excellent discussion of underwater acoustics. Reviews of fish hearing and related material are provided by Popper *et al.* (2003); Ladich & Popper (2004) and Popper & Schilt (2008). Fay (2003) provides details about sound source localization, one of the fundamental roles of hearing.
FISH BIOACoustics

Fishes, as all animals, glean a good deal of information about the world around them from listening to the sounds from abiotic and biotic sources. Abiotic sources include the sounds of waves on the shore, geologic events, rain on the water surface and numerous other non-biological sounds. Biotic, or biological, sources include not only sounds produced by conspecifics, but also sounds produced by other fish species, marine mammals and invertebrates. All of these sounds make up what Bregman (1990) called the ‘auditory scene’. The auditory scene can be thought of very much like a visual scene – everything that an animal or human can see with its light receptors – except that the auditory scene involves sound and sound receptors. The value of the auditory scene is that it provides information that is at far greater distance from a fish than vision does, especially in dark water, at night and in rocky inshore areas. It follows that interference with the detection of the auditory scene could have significant effects on fishes that use sounds to gather information about predators and that prey, conspecifics, the location of a reef and many other things about the fish world.

FISH HEARING

Fishes are able to detect and respond to a wide range of sounds. Experimental studies determine the range of frequencies (or bandwidth) that a fish can detect, and then determine the lowest levels of the sound detected at each frequency (the ‘threshold’, or lowest signal that an animal will detect in some statistically determined percentage of signal presentations – most often 50%). The graphic representation of the threshold as a function of frequency is called an ‘audiogram’ (Fig. 1). Figure 1 demonstrates that species differ in the range of frequencies (bandwidth) and in the lowest SPL (threshold), that they are able to detect.

The majority of fish species detect sounds from below 50 Hz up to 500–1500 Hz. A smaller number of species can detect sounds to over 3 kHz, while a very few species can detect sounds to well over 100 kHz. Fishes with the narrower bandwidth of hearing are often referred to as ‘hearing generalists’ or hearing ‘non specialists’ whilst fishes with the broader hearing range are often called ‘hearing specialists’. The difference between hearing generalists and specialists is that the latter usually have specialized anatomical structures that enhance hearing sensitivity and bandwidth (see Popper et al., 2003; Ladich & Popper, 2004). Generalists include salmonids, cichlids, tunas and numerous other species (see Ladich & Popper, 2004). Specialists include all the Otophysi and Clupeiformes, and some representatives in a wide range of other fish groups such as a few holocentrids, sciaenids, etc. The fish known to have the widest hearing frequency bandwidth are limited to the members of the clupeiform genus Alosa (Mann et al., 2001).

As noted later, the critical issue for understanding whether an anthropogenic sound affects hearing is whether it is within the hearing frequency range of a fish and loud enough to be detectable above threshold. Moreover, the hearing data in the literature are only for about 100 of the 29 000 or more extant species of fish (see Popper et al., 2003; Ladich & Popper, 2004) and so any extrapolation of hearing capabilities, and effects of anthropogenic sound, between different species, and especially those that are taxonomically distant, must be done with the greatest caution.
FIG. 1. Hearing thresholds for a select group of fish species to illustrate fish hearing capabilities. *Carassius auratus*; *Salmo salar*; *Euthynnus* sp.; *Gadus morhua*; *Melanogrammus aeglefinus*. *Carassius auratus* is considered a hearing specialist, whereas the other species would be considered hearing generalists. To do more accurate measures, one would need to determine not only sound pressure, as done in the studies reported here, but also particle motion because that is what hearing generalists are probably detecting. There are also recent data suggesting that salmonids (*S. salar* and related species) are able to detect sounds below about 35 Hz (e.g., Knudsen *et al.*, 1992, 1994) and that some clupeids can detect sounds well above 3 KHz (Mann *et al.*, 2001). Data are from behavioural studies compiled in Fay (1988).

Fishes, like other vertebrates, have two inner ears that lie within the cranial cavity just lateral to the brain. Unlike terrestrial vertebrates, however, fishes have no middle or external ear. The structure of the fish inner ear is similar to that found in all other vertebrates (Ladich & Popper, 2004). The basic mechanism for transduction of sound into electrical signals compatible with the nervous system in the ear is the sensory hair cell (Coffin *et al.*, 2004). Significantly, high intensity sounds are able to fatigue, damage, or kill these cells, resulting in temporary or permanent hearing loss. At the same time, fishes, unlike most tetrapods other than amphibians, continue to add sensory hair cells throughout much of their lives (e.g., Popper & Hoxter, 1984; Lombarte & Popper, 1994, 2004; Higgs *et al.*, 2003). In addition, there is evidence that fishes, unlike mammals, can replace or repair sensory cells that have been damaged as a result of exposure to certain drugs (Lombarte *et al.*, 1993) or fatigued due to sound exposures (Smith *et al.*, 2006) that cause a shift in auditory thresholds. These studies need replication with additional species and the time course of recovery of hair cells needs to be examined in detail to understand how hearing recovery takes place in fishes.
EFFECTS ON FISH FROM EXPOSURE TO SOUND

Interest in the effects of anthropogenic sound on aquatic organisms has grown considerably in the past two decades (e.g. NRC, 1994, 2000, 2003, 2005; Richardson et al., 1995; NRDC, 1999; Wartzog et al., 2004; Southall et al., 2007; Hastings, 2008). While these reports, and a handful of research studies, have focused primarily on marine mammals, several have raised the issue that sounds that potentially affect marine mammals may also affect other aquatic organisms, including fishes and invertebrates (e.g. NRC, 1994, 2000, 2003; Popper, 2003, 2006; Popper et al., 2004; Hastings, 2008; Popper & Hastings, 2009). The basis for concern about the potential effects of sound on fishes is the well-documented effects of exposures to sound on behaviour, hearing, and overall physiology in terrestrial animals and humans (Lehnhardt, 1986; NIH, 1990; Henderson et al., 2008). Although this extension from terrestrial animals to fish is certainly valid, there is also a more direct, albeit limited, body of literature that demonstrates the effects of exposure to sound on fishes, including the aforementioned observations that sound from pile driving can potentially injure and even kill fish close to the source. These more specific studies are the basis of the following discussion.

Results of the few peer-reviewed studies and additional studies reported in the grey literature on the effects of sound on fishes are discussed in this section. The specific studies are outlined by type in Table I to give an overview of the investigations and to show gaps in the literature that must be filled to enable understanding of the overall effects of sound on fishes, and the specific effects of exposure to sounds produced by pile driving.

BEHAVIOURAL RESPONSES OF FISH TO SOUND

There have been very few studies of the effects of anthropogenic sounds on the behaviour of fishes. Data are lacking not only on the immediate behavioural effects on fishes close to a source, but also effects on fishes further from the source. Moreover, nothing at all is known about the long-term effects of exposure to sound on fish behaviour or about the effects of cumulative exposure to loud sounds. It is also critical to differentiate between ‘behaviour’ of fish in an experimental cage or tank and behaviour in the natural environment. Most importantly, while behaviour in a test cage or tank during sound exposure provides some small insight into how fish may respond to a sound at its onset (e.g. McCauley et al., 2000; Boeger et al., 2006; Popper et al., 2007; Sarà et al., 2007), these observations in no way indicate how an unrestrained animal would behave when exposed to the same sound. And, the concern for effects on behaviour is how a fish will respond when it is in the wild and perhaps on its feeding site or at a breeding location.

Several studies have demonstrated that anthropogenic sounds may affect the behaviour of at least a few species of fish. For example, field studies by Engås et al. (1996) and Engås & Løkkeborg (2002), while not actually observing the behaviour of fish per se, showed that there was a significant decline in catch rate of haddock Melanogrammus aeglefinus (L.) and Atlantic cod Gadus morhua L. that lasted for up to 5 days after termination of use of seismic air guns for geologic exploration; after that time the catch rate returned to normal. The authors concluded that the catch decline resulted from the sound of the air guns, and that the sound probably caused the fish to leave the area of ensonification, although there were no direct...
Table I. Citations of selected studies examining the effects of exposure to sound on fishes that have most relevance for helping understand effects of sound on fishes. [Note that there are no data for non teleosts (e.g. sturgeon, elasmobranchs)]

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<th>Issue</th>
<th>Hearing generalists</th>
<th>Hearing specialists</th>
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<tr>
<td>Mortality</td>
<td>Yelverton et al., 1975 (Poecilia reticulata, Lepomis macrochirus, Oncorhynchus mykiss and Micropterus salmoides: explosive blasts)</td>
<td>Yelverton et al., 1975 (Carassius auratus, I. punctatus, Gambusia affinis and Cyprinus carpio: explosive blasts)</td>
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<td></td>
<td>Ruggerone et al., 2008 (Oncorhynchus kisutch: pile driving)</td>
<td>Hastings, 1995 (Carassius auratus and Trichogaster trichopterus: pure tones)</td>
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<td>Physical injury</td>
<td>Yelverton et al., 1975 (Poecilia reticulata, Lepomis macrochirus, Oncorhynchus mykiss and Micropterus salmoides: explosive blasts)</td>
<td>Yelverton et al., 1975 (Carassius auratus, Ictalurus punctatus, Gambusia affinis and Cyprinus carpio: explosive blasts)</td>
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<td>Hastings, 1995 (Carassius auratus and Trichogadster trichopterus: pure tones)</td>
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<td>Auditory tissue damage</td>
<td>Enger, 1981 (Gadus morhua: pure tones)</td>
<td>Hastings, 1995 (Carassius auratus: pure tones)</td>
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<td>Hastings et al., 1996 (Astronotus ocellatus: pure tones)</td>
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<td>McCauley et al., 2003 (Pagrus auratus: air gun)</td>
<td>No data available</td>
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<td>Temporary threshold shift (TTS)</td>
<td>Popper et al., 2005 (Coregonus nasus and Esox lucius: seismic air guns)</td>
<td>Smith et al., 2004a, b, 2006* (Carassius auratus: band-limited noise)</td>
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<td></td>
<td>Popper et al., 2007* (Oncorhynchus mykiss: SURTASS LFA sonar)</td>
<td>Popper et al., 2005 (Couesius plumbeus: seismic air guns)</td>
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<td>Scholik &amp; Yan, 2001* (Pimephales promelas: band-limited white noise)</td>
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<td>Popper &amp; Clarke, 1976 (Carassius auratus: pure tones)</td>
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<td>*Hearing did not recover by conclusion of study</td>
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### Table I. continued

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<th>Issue</th>
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<th>Hearing specialists</th>
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<tr>
<td>Behavioural changes</td>
<td>Wardle <em>et al.</em>, 2001 (Reef fish and invertebrates: minor response during air gun-shots, but no permanent change)</td>
<td>No data available</td>
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<td>Eggs and larvae</td>
<td>Banner &amp; Hyatt, 1973 <em>(Cyprinodon variegatus</em> and <em>Fundulus similis</em>: decreased egg viability and larval growth in tanks with increased noise)</td>
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<td>Kostyuchenko, 1973 (Marine fishes: increased egg mortality up to, 20 m from seismic source)</td>
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<td>Booman <em>et al.</em>, 1996 (<em>Gadus. morhua</em>, <em>Pollachius virens</em> and <em>Clupea harengus</em>: variable results with some stages showing decreased growth in a few species when exposed to air guns)</td>
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<td>Govoni <em>et al.</em>, 2003, 2008 (<em>Lagadon rhomboides</em> and <em>Leiostamus xanthurus</em>: physical injury and mortality; explosive blasts)</td>
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<td>Jørgensen <em>et al.</em>, 2005 (<em>Clupea harengus</em>, <em>Gadus morhua</em>, <em>Pollachius virens</em> and <em>Anarhichas minor</em>: physical injury and mortality; mid-frequency sonar)</td>
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<td>Miscellaneous</td>
<td>Skalski <em>et al.</em>, 1992 (<em>Sebastes</em> sp.: catch decreased after one air-gun shot)</td>
<td>Smith <em>et al.</em>, 2004a <em>(Carassius auratus</em>: no change in corticosteroid levels after continuous exposure to band limited noise)</td>
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<td>Engås <em>et al.</em>, 1996 <em>(Melanogrammus aeglefinus</em> and <em>Gadus morhua</em>: catch reduction after seismic survey with air-guns)</td>
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<td>Slotte <em>et al.</em>, 2004 (<em>Clupea harengus &amp; Micromesistius poutassou</em> potential avoidance of region during use of air guns)</td>
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data to support this conclusion. Subsequently, Slotte et al. (2004) showed parallel results for several additional pelagic species including blue whiting *Micromesistius poutassou* (Risso) and Norwegian spring spawning herring *Clupea harengus* L. These investigators found that fishes in the area of the air-guns appeared to go to greater depths after sound exposure compared to their vertical position before the air-gun usage. Moreover, the abundance of animals 30–50 km away from the ensonification increased, suggesting that migrating fish would not enter the zone of seismic activity. A non-peer-reviewed report by Gausland (2003) suggests, however, that the declines in catch rate observed in these studies may have resulted from other factors and are not statistically different than the normal variation in catch rates over several seasons.

Similar to the aforementioned studies, Skalski et al. (1992) showed a 52% decrease in rockfish (*Sebastes* spp.) catch when the area of catch was exposed to a single air-gun emission at 186–191 dB re 1 μPa (mean peak level) (see also Pearson et al., 1987, 1992). The investigators also demonstrated that fishes would show a startle response to sounds as low as 160 dB (re 1 μPa), but this sound level did not appear to elicit decline in catch.

Hassel et al. (2004) examined effects of seismic air-gun exposure on caged lesser sandeel *Ammodytes marinus* Raitt. Received sound levels were not measured in the cages. Mortality for *A. marinus* was the same in experimental and control cages, and was attributed to deployment of the cages and handling and confinement of the animals. The authors reported a small decline in *A. marinus* abundance in the study region shortly after the seismic activity, but this quickly returned to preseismic levels.

While not directly relevant to fish, internationally there is growing interest regarding the effect exposure to seismic air guns can also have on invertebrates. In one of the few published evaluations of air guns on invertebrates, Andriguetto-Filhoa et al. (2005) examined catch rate of two shrimp species and the Atlantic seabob before and after seismic shooting in about 2–15 m of water, using transects that would mimic an actual seismic survey. The measured source level of the air guns was 196 dB re 1 μPa at 1 m (peak). The investigators reported that there was no apparent change in catch rate for all three species before and after the seismic shooting. However, they point out that post shooting fishing was about 12–36 h after air-gun exposure and that there was the possibility of shrimp mortality and then recruitment before fishing was resumed, thereby ‘masking’ any immediate effects of the air guns. The authors also cite a technical report by Östrensky et al. (2002) that evaluated effects of air guns on caged shrimp of several species and reportedly found no mortality or tissue damage using what appear (from images in the report) to be excellent histopathological techniques.

Wardle et al. (2001) used a video system to examine the behaviour of fish and invertebrates on a rocky reef off Scotland in response to emissions from seismic air guns that were carefully measured to have a peak level of 210 dB re 1 μPa at 16 m from the source and 195 dB re 1 μPa at 109 m from the source. The investigators found only minor behavioural responses to the air-gun emissions. They also found no permanent changes in the behaviour of the fish or invertebrates on the reef throughout the course of the study, and no animals appeared to leave the reef. There was no indication of any observed damage to the animals.
Boeger et al. (2006) examined the effects of exposure to seismic air guns on a few specimens of six reef species in Brazil held in a cage near air guns that emitted signals at 196 dB re 1 μPa (peak) at 1 m from the source. Sound levels in the cages were not provided, nor it is clear from the paper exactly how fish were exposed to the sound, though distances from the source were indicated as from 0 to 7 m. The investigators reported no immediate mortality or apparent damage to the test animals, though there were no reports how the animals were maintained, and observed at any time after exposure. The authors did observe the behavioural responses of the fish in the cage, and while they recognized that responses of caged fish are not indicative of how animals would behave in the wild, they did note, as did Wardle et al. (2001) on uncaged animals, that the caged fish showed a small level of response and startle at the start of the air-gun exposure, but this appeared to decline at subsequent air-gun emissions.

Finally, Turnpenny et al. (1994) examined the behaviour of three species of fish in a pool in response to different sounds, but results are not useable due to lack of calibration of the sound field at different frequencies and depths and many other problems with experimental design. In enclosed chambers that have an interface with air, such as tanks and pools used by Turnpenny et al., the sound field is known to be very complex and will change significantly with frequency and depth (Parvulescu, 1967; Blackstock, 2000; Akamatsu et al., 2002). As a consequence, responses of the animals in the Turnpenny et al. (1994) study cannot be correlated with any aspect of the acoustic signal, and the findings are highly questionable.

STRESS AND OTHER PHYSIOLOGICAL RESPONSES

The effect of stress is much more difficult to define because it is hard to quantify this measure in fish since it has not been extensively studied; however, increased background noise is known to increase stress in humans (e.g. NIH, 1990; von Gierke & Eldred, 1993; Pearsons et al., 1995). There is evidence that the effects on non-auditory aspects of an animal’s physiology, such as an increase in stress levels, can come from increased background noise or exposure to a sudden increase in sound pressure (e.g. Hattingh & Petty, 1992). In turn, increased stress affects overall human health and wellbeing and it is reasonable to suggest that sound is also likely to cause stress in fishes. Thus, a considerable concern with regard to aquatic organisms is not only related to the effect of exposure to sound on the function of the auditory receptor, but also to the effect of any sounds that are above ambient levels on overall health and wellbeing.

Gilham & Baker (1985) used vibration of the aquarium walls to produce stress responses in O. mykiss. Although the stressors were not quantifiable, this study demonstrated that a general stress response occurred in fish between 1 and 5 days after signal onset, as demonstrated by significant increases in serum cortisol levels. Other studies have demonstrated that exposure to non-traumatic stressors (e.g. crowding, spawning, rapid environmental changes, suboptimal water quality or physical environment, altered conductivity and pollution) can predispose fish to opportunistic infections (Walters & Plumb, 1980; Pickering, 1981; Noga et al., 1998; Wedemeyer, 1999). Mesa (1994) found that salmonid smolts going over a hydroelectric dam showed significant stress effects and this made them more susceptible to predation than fish that had not gone over the dam. While the stimulus
in this study was vastly different from high-level sound, the results do suggest that if exposure to sound results in highly stressed fish, even if fish do not die as a direct result of stimulation, they may be more susceptible to predation or other environmental effects than non-stressed fish.

Smith et al. (2004a) demonstrated that corticosteroid levels in goldfish *Carassius auratus* (L.) do not change at a statistically significant level in response to continuous exposure to band-limited noise in the 0.1–10 kHz frequency band with an overall root-mean-square (rms) pressure level of 170 dB re 1 μPa (rms pressure level is used primarily to characterize continuous sounds; it is calculated by taking the square-root of the mean value over time of the square of the sound pressure). Corticosteroid level is a measure of stress, and the results of the Smith et al. (2004a) study led to the suggestion that stress levels in these animals were not influenced by noise exposure for up to a month. Similarly, Wysocki et al. (2007) exposed *O. mykiss* to continuous band-limited noise at 150 dB re 1 μPa for the first 9 months of their lives and found no stress effects, even when assessed with a variety of measures. While these results are of considerable interest, they provide only the first data in what must be a larger-scale examination of effects of sounds on stress levels in fishes. One must be cautious, however, in extrapolating between fish species and between different experimental paradigms in trying to understand the effects of potential stressors on physiology.

It is also important to note that all of these studies were done in the laboratory. How these results relate to fishes in their natural habitats is unknown as free-living animals may not have physiological stress markers (in response to sound) similar to those found in a laboratory environment.

While not totally germane to fishes, there is some evidence that an increased background noise (for up to three months) may affect the invertebrate brown shrimp *Crangon crangon* (L.). Legardère (1982) demonstrated that shrimp exposed in a soundproof room to noise that was about 30 dB above ambient for 3 months demonstrated decreases in both growth and reproductive rates. In addition, Legardère & Régnauld (1980) showed changes in the physiology of the same species with increased noise and that these changes continued for up to a month following termination of the exposure.

**HEARING LOSS AND DAMAGE TO AUDITORY TISSUES**

There are two classes of effects of exposure to sound on hearing. Exposure to low levels of sound for a relatively long period of time or exposure to higher levels of sound for shorter periods of time may result in temporary hearing loss, referred to as temporary threshold shift or TTS (e.g. Lonsbury-Martin et al., 1987). The level and duration of exposure that causes TTS varies widely and can be affected by factors such as repetition rate of the sound, SPL, frequency, duration, health of the organisms and many other factors. By definition, hearing recovers after TTS. The extent of the hearing loss, including how many dB threshold shift occurs and the duration of the hearing loss may vary considerably and depends upon many variables associated with exposure. Depending on these variables, TTS may continue from minutes to days after the end of exposure.
The second possible effect is referred to as permanent threshold shift or PTS. PTS is a permanent loss of hearing and is generally accompanied by death of the sensory hair cells of the ear in mammals (e.g., Saunders et al., 1991; Henderson et al., 2008).

Laboratory studies have been used to determine whether there may be temporary or permanent changes in hearing ability in animals exposed for short or long periods of time to different types of sound (e.g., pure tones or white noise), and a few field studies have investigated the effects of sound from anthropogenic sources on hearing in fish exposed to actual military sonar or seismic air-gun sources. Hearing loss has been measured using behavioural or electrophysiological tests for several fish species, including *C. auratus*, fathead minnows *Pimephales promelas* Rafinesque, northern pike *Esox lucius* L., lake chub *Couesius plumbeus* (Agassiz) and *O. mykiss* (e.g., Popper & Clarke, 1976; Scholik & Yan, 2001, 2002; Smith et al., 2004a, b, 2006; Popper et al., 2005, 2007).

Smith et al. (2004a, b) tested hearing in *C. auratus* and Nile tilapia *Oreochromis niloticus* (L.) to determine more detailed parameters of hearing loss, including the effects of different exposure durations and recovery times. They demonstrated that *C. auratus* had a 5 dB TTS after only 10 min of exposure to band limited noise (0–10 kHz, approximately 170 dB re 1 µPa overall SPL), and with a three-week exposure to the same stimulus fish experienced a 28 dB threshold shift and took over two weeks to return to normal hearing. Similarly, Scholik & Yan (2001) demonstrated that *P. promelas* did not recover to control thresholds even as long as 14 days after termination of a 24 h exposure to white noise from 0.3 to 2.0 kHz with an overall SPL of 142 dB re 1 µPa.

Both Smith et al. (2004a) and Scholik & Yan (2002) reported no hearing loss in two species of hearing generalists (*O. niloticus* and bluegill sunfish *Lepomis macrochirus* Rafinesque), and Wysocki et al. (2007) did not find TTS in *O. mykiss* after 9 months of exposure to 2 Hz to 2 kHz broadband noise with an overall rms SPL of 150 dB re 1 µPa in an aquaculture facility. In all three studies, however, the sound stimulus was primarily pressure, whereas the major stimulus for hearing generalists is believed to be acoustic particle motion. Moreover, all of these researchers controlled only the SPL during electrophysiological measurements to determine the audiograms before and after sound exposure. Thus, these results are not surprising because hearing sensitivity and TTS in hearing generalists are likely to be most meaningful in terms of acoustic particle motion or, possibly, acoustic intensity.

In contrast, two investigations using high-level anthropogenic sources to test hearing loss and effects on other aspects of fish biology reported hearing loss in two different species of hearing generalists in addition to one hearing specialist (Popper et al., 2005, 2007). Moreover, a recent field study with a large air-gun array found no hearing loss in one hearing specialist and three hearing generalists (Hastings et al., 2008).

In one study, Popper et al. (2007) used a U. S. Navy Surveillance Towed Array Sensor System (SURTASS) Low-Frequency Active (LFA) sonar transducer and tested hearing in *O. mykiss*. Popper et al. (2007) found that exposure to three emissions of the sonar, each for 108 s (with 9 min between signals) with a received rms SPL estimated to be 193 dB re 1 µPa (frequency bandwidth about 160–325 Hz), produced up to 25 dB of threshold shift. However, hearing tests could not be carried out past 2 days to determine if the fish recovered from hearing loss.
In the second study, Popper et al. (2005) tested three species, including another salmonid [broad whitefish Coregonus nasus (Pallas)]. After stimulation with five shots of a small seismic air-gun array, each shot having a received mean peak sound level of about 205–210 dB re 1 μPa (a received mean SEL of about 176–180 dB re 1 μPa²-s per shot), C. nasus showed no TTS, whereas E. lucius (a hearing generalist) and C. plumbeus (a hearing specialist) showed 10–25 dB of hearing loss that recovered within 24 h after exposure.

In the first study in the field to examine hearing loss in reef fishes during a seismic survey in the Indian Ocean with a full air-gun array, Hastings et al. (2008) found no hearing loss following sound exposures up to 190 dB re 1 μPa²-s cumulative SEL in a hearing specialist, the pinecone soldierfish Myripristis murdjan (Forsskål), and in three species of hearing generalists: the blue green damselfish Chromis viridis (Cuvier), sabre squirrelfish Sargocentron spiniferum (Forsskål) and bluestripe seaperch Lutjanus kasmira (Forsskål). Fishes were caught on-site by professional aquarium collectors, placed in cages at 5 m depths and at three different distances perpendicular to a survey line, and exposed to multiple shots from a 2055 cubic-inch (33·68 l) air-gun array as it traversed the length of the survey line. Received SEL was determined from recordings of SPL with data loggers attached to the cages.

In humans and other terrestrial vertebrates, exposure to very high SPLs, for even a short period of time, may result in permanent hearing loss. This occurs because the sound mechanically destroys the sensory hair cells of the inner ear and, or fractures or dislocates the ossicular chain of the middle ear (Roberto et al., 1989; Patterson & Hamernik, 1997; Henderson et al., 2008). It is significant that exposure to sound at lower levels but for longer periods, as in a noisy work environment, can also lead to permanent hearing loss through death of sensory cells (Kryter, 1985; Hamernik et al., 1994). At the same time, the data on the effects of exposure to these types of sounds on fishes are very limited as compared with data for terrestrial vertebrates; but there is a small body of peer-reviewed literature showing that such exposures to sound can damage sensory cells in ears of some fish species (Enger, 1981; Hastings et al., 1996; McCauley et al., 2003), while two other studies, with different species and sound sources, did not find such damage (Popper et al., 2007; Song et al., 2008). No study, however, has yet examined the relationship between damage of hair cells and permanent hearing loss in fishes.

While looking for evidence of frequency discrimination in the ears of G. morhua, Enger (1981) found that some sensory cells lost their ciliary bundles (and were possibly killed) after 1–5 h exposure to pure tones at frequencies from 50 to 400 Hz with an SPL of 180 dB re 1 μPa. This is 100–110 dB above threshold in the most sensitive hearing frequency range for G. morhua. The sensory epithelia were examined using scanning electron microscopy (SEM). Enger used a waveguide instrumented with an actively controlled sound projector at each end to produce an exposure that had negligible particle motion.

In a similar study, Hastings (1995) reported damage to auditory hair cells in C. auratus exposed to continuous tones having peak levels of 189, 192 and, 204 dB re 1 μPa at 250 Hz and 197 dB re 1 μPa at 500 Hz for approximately 2 h. These SPLs are at least 120–140 dB above auditory thresholds for C. auratus at these frequencies. This study also used a waveguide that allowed exposure to sound with negligible particle velocity. Four fish were exposed at each set of conditions, and damage was found to correlate with SPL at a 95% confidence level. This study also
included controls (fish placed in the waveguide and held for 2 h, but not exposed to sound). In addition, *C. auratus* exposed to tones with a peak level of 182 dB re 1 μPa at 500 Hz had no hair-cell damage. This is approximately 120 dB above auditory threshold at a frequency in their most sensitive bandwidth.

Hastings *et al.* (1996) demonstrated similar effects on the ears of the oscar *Astronotus ocellatus* (Agassiz). Some sensory cells in the ears of four out of five animals were damaged after 1 h of continuous exposure to a 300 Hz pure tone with a peak level of 180 dB re 1 μPa, while no damage occurred after 1 h exposure to the same tone applied for 20% of each minute. It is important to note that damage was not evident in animals after one day but was evident in the animals that were kept alive for four days following exposure. These results suggest that damage from exposure to sound takes some time to become visually apparent.

McCauley *et al.* (2003) investigated the effects of exposure to the sounds of a seismic air gun on the Australian fish, the pink snapper *Pagrus auratus* (Forster) (see also McCauley *et al.*, 2000 for detailed experimental methods). Fish were caged and exposed to hundreds of emissions from an air gun as it approached and moved over and beyond the cage for approximately 1.5 h. Received SEL exceeded 180 dB re 1 μPa^2^-s for several of the shots. The animals were sacrificed after different post-exposure time intervals and their ears examined for signs of damage using electron microscopic techniques very similar to those used by Hastings *et al.* (1996). The results clearly showed about 15% of the sensory hair cells severely damaged in the caudal end of the saccular epithelium in several of the animals after a post exposure period of 58 days (the maximum survival interval described); however, this was only about 2.7% of the total number of sensory hair cells overall.

While the McCauley *et al.* (2003) study further substantiated the potential for destruction of hair cells in fish when exposed to high-level sounds, both the McCauley *et al.* (2003) and Hastings *et al.* (1996) studies were careful to provide a number of important caveats to their work. These included (1) use of only a few species, which may not be representative of other species; (2) the inability of the caged fish to escape from the sound field; and (3) the relatively long duration of exposure as compared to exposures to what would be expected from other types of anthropogenic sounds of that SPL.

One difference between these studies that needs to be controlled in future investigations is the relationship between acoustic pressure and particle velocity in the sound stimulus. Whilst it was possible for Hastings *et al.* (1996) to measure pressure and maintain a plane wave in their waveguide test chamber, this could not be done by McCauley *et al.* (2003) in the field. The importance of having full characterization of the stimulus in these and future studies is to enable correlation of results with specific metrics of the sound stimulus and thus comparison of results between studies.

It again needs to be pointed out that hair-cell damage observed in these four species (*G. morhua, C. auratus, A. ocellatus* and *P. auratus*) was only a visual manifestation of what may have been a much greater effect. Furthermore, in two of the studies observable physical evidence took days to show up. It may be more important to evaluate the more immediate effects of the sounds on hearing capabilities of the fish, as was done by Popper *et al.* (2005, 2007). Even temporary deafness could result in a fish being unable to respond to other environmental sounds that indicate the
presence of predators and facilitate the location of prey and mates. Effects, however, depend on the use of sound by that species in those situations.

While it is clear from the data discussed above that some types of sounds can affect the ear and hearing over time, it is important to note that at this stage of our knowledge, and the very limited data, that one must be extremely cautious in extrapolating results between fish species or received signals. Thus, results for one species may not be indicative of the results one would obtain for another species using the same type of signal. Moreover, the results from one type of signal (e.g. air gun) may not be germane to another signal (e.g. pile driving).

The reasons for not being able to extrapolate results are many and include (1) differences in the hearing systems of different fish species and too little knowledge about the effects of exposure to sound on such different auditory systems; (2) limited data on the precise nature of a stimulus (e.g. pressure and, or particle velocity) which might affect the hearing apparatus; and (3) the time course (e.g. continuous v. transient) and frequency components of the different signals. To be able to reasonably extrapolate between fish species and types of signals, much more will need to be known about the effects of sounds on different auditory systems.

Furthermore, it will be imperative to have common ways of expressing exposure to sound so that it is possible to compare stimulus parameters between signals of different types, especially for impact and impulsive sounds. Indeed, several studies on guinea pigs and chinchillas have shown that the $L_{eq}$ and SEL metrics do not correlate with TTS and PTS when the stimulus includes impulsive or transient sounds. ($L_{eq}$ is a theoretical equivalent continuous SPL for a given period of time that is calculated to have the same acoustic energy as an interrupted sound.) Lataye & Campo (1996) found that $L_{eq}$ was not an accurate predictor of noise-induced hearing loss (NIHL) in guinea pigs when the temporal characteristics of a sound exposure were varied. Their results indicate the existence of a ‘critical intensity’ as a demarcation between metabolic and mechanical damage mechanisms, which is not accounted for in the time-averaged $L_{eq}$.

Hamernik et al. (1974) showed that when continuous and impulsive noises were combined, chinchillas experienced higher levels of TTS and hair cell damage than would be expected by simply adding the effects of individual exposures to each type of noise. In more recent studies, Hamernik & Qiu (2001) also found that NIHL did not correlate with $L_{eq}$ in chinchillas when the sound exposure included impact signals mixed with band-limited noise even though all exposures had the same total energy.

Both Hamernik & Qui (2001) and Hamernik et al. (2003) reported that the kurtosis metric (a statistical measure used to estimate the deviation of the waveform amplitude distribution from a normal distribution over the time duration of the signal) correlated with TTS, PTS and outer hair-cell loss for exposures that contained high-level transients (impacts or noise bursts). Given the results of these studies, it is premature to provide any recommendations on exposure levels that could cause TTS or PTS in any fish species.

Finally, it should be noted that the same concerns regarding stimulus variables and extrapolation between species apply to all other aspects of the effects of exposure to sound on fishes. Some of these other effects are discussed below.
STRUCTURAL AND CELLULAR DAMAGE OF NON-AUDITORY TISSUES AND MORTALITY

Compared to data for the effects of exposure to sound on fish hearing capabilities and the ear, there are even fewer peer-reviewed data regarding non-auditory effects on fish biology. In contrast, it is widely known that exposure to sounds at high levels can alter the physiology and structure of terrestrial vertebrates (e.g. Fletcher & Busnel, 1978; Saunders et al., 1991). Indeed, there are strong standards set by the U. S. Occupational Safety and Health Administration (OSHA) recognizing that high levels of background sound have an effect on human wellbeing (e.g. NIH, 1990; von Gierke & Eldred, 1993; Pearsons et al., 1995). These changes may include cellular changes, organ system changes, or stress-level effects caused by exposure to sound. Exposure to sound at ultrasonic frequencies (c. 750 kHz and higher) has even induced cardiac arrhythmias in humans and premature ventricular contractions in frogs (Dalecki et al., 1991); however, these effects have not been observed at lower frequencies that characterize the sound produced by pile driving or other low frequency underwater sources.

While there are far fewer data on the effect of exposure to sound on wild animals (including aquatic animals), it is reasonable to suggest that long-term exposure to relatively high levels of sound could affect all vertebrates (e.g. Richardson et al., 1995) just as does shorter-term exposure to higher-level signals. For example, one of the organ systems of most concern with marine mammals is the lungs and the resultant damage that may occur in this organ due to the presence of air. Many fishes have at least one air chamber, the swim bladder, which provides a similar discontinuity between water and air as does the lung in marine mammals.

Studies on terrestrial mammals suggest that gas-filled structures (i.e. lung) or gas pockets (such as could occur in the gastrointestinal tract) within a body make it susceptible to damage by sound (Richmond et al., 1973; Fletcher et al., 1976; Yang et al., 1996; Bauman et al., 1997; Dodd et al., 1997; Elsayed, 1997). Tissue damage is possible when sound passes through the interface from a tissue structure (e.g. adipose tissue and muscle) to a gas void because the gas is more compressible than tissue, resulting in a relatively large increase in the motion between the two. In addition, sound will cause gas organs such as the swim bladder and lung to oscillate and push on the surrounding tissues. The amplitude of these oscillations can be relatively large at high SPLs or even at lower SPLs if the gas organ is excited at its fundamental resonance frequency.

In fishes, gas oscillations induced by high SPLs can potentially cause the swim bladder to tear or rupture, as has been shown in several studies using explosives (e.g. Aplin, 1947; Coker and Hollis, 1950; Gaspin, 1975; Yelverton et al., 1975; Wiley et al., 1981; Govoni et al., 2003). Similar results have been observed in some (but far from all) fish exposed to pile driving sound (Caltrans, 2001, 2004; Abbott & Bing-Sawyer, 2002). Popper et al. (2007), however, found no swim bladder damage in _O. mykiss_ following exposure to SURTASS LFA sonar for three 108 s intervals separated by 9 min of quiet environment and Ruggerone et al. (2008) found no effect of exposure to over 1600 pile driving strikes on _O. kisutch_.

Additional studies, primarily in the grey literature, using explosives suggest that there is more damage to fishes with swim bladders than to species, such as flatfish, that do not have such air chambers (e.g. Coker & Hollis, 1950; Gaspin, 1975; Baxter et al., 1982; Goertner et al., 1994), and at least one study showed no difference in
damage between fish with ducted (physostome) and non ducted (physoclist) swim bladders (Yelverton et al., 1975). It has also been shown that the effects on fish decline rapidly with distance from the explosion (e.g. Houghton & Mundy, 1987; Goertner, 1994; Govoni et al., 2008) as the peak overpressure and impulse (i.e. time integral of the pressure waveform) decreases. Similarly, a study by Kearns & Boyd (1965) suggested that the extent of fish kill decreases with increasing distance of the fish from an explosive source. Based on these and other studies (e.g. Yelverton et al., 1975), it is clear that there is considerable variability in the effects of explosive blasts on fishes, and that the variables include received sound energy, presence or absence of gas bubbles (e.g. swim bladder), mass of fish and perhaps body shape (e.g. Teleki & Chamberlain, 1978), and biomechanical properties of the swim bladder wall (Wiley et al., 1981).

It has been suggested that the large negative overpressure characteristic of pile driving sounds may be more damaging to the swim bladder than the initial positive overpressure (Traskey, 1976; Caltrans, 2004) because of expansion of the swim bladder during the negative phase. Bailey et al. (1996), working with mice submerged in water, found that a sound pulse having a large positive peak overpressure was at least as damaging to the lungs as one having a large negative peak overpressure of approximately the same level and time duration. Damage increased with magnitude of pressure incident at the lung, but histology showed no qualitative or quantitative differences between the effects of positive and negative pressures. Mouse lungs had increasing haemorrhage with increasing exposure levels regardless of the polarity of the peak overpressure. These findings indicate that injury would correlate with the work done on the lung tissue, which would be equivalent to the total received energy from a passing sound wave.

Govoni et al. (2003, 2008) also concluded that an energy metric, the acoustic ‘impulse’ (the integral of the pressure waveform over time with units in psi-ms or Pa-s), correlated with observed effects of submarine detonations on juvenile and larval pinfish *Lagodon rhomboides* (L.) and spot *Leiostomus xanthurus* Lacépède. Moreover, Stuhmiller et al. (1996) concluded that incidence of blast injury to the lung and lethality correlates with total energy in the wave normalized by lung volume in terrestrial animals.

Other structures within the body can be affected by exposure to sound because of their small size or dynamic characteristics. There is some evidence to suggest that sound at sufficiently high pressure levels can generate bubbles from micronuclei in the blood and other tissues such as fat (ter Haar et al., 1982). In fish, blood vessels are particularly small in diameter so bubble growth by rectified diffusion (Crum and Mao, 1996) at low frequencies could create an embolism and burst small capillaries to cause superficial or internal bleeding. This type of bubble growth may also occur in the eyes of fish where the tissue might have high levels of gas saturation (see non-peer-reviewed reports by Turnpenny et al., 1994; Gisiner, 1998). Internal bleeding was found in small juvenile and larval fish exposed to explosions (Govoni et al., 2003, 2008) and observed through the skin in *C. auratus* exposed to pure tones at high sound levels for 1–2 h (Hastings, 1995), but no such effects were found in studies on exposure of larger fish to pile driving (Abbott et al., 2005; Ruggerone et al., 2008) and SURTASS LFA sonar (Popper et al., 2007).

Another type of tissue damage caused by exposure to high-level, transient sound is traumatic brain injury (TBI) or neurotrauma. In humans, TBI can occur with no
marks of external injury, but manifests itself with instantaneous loss of consciousness or sustained feelings of anxiety and confusion, or amnesia, and may result in death (Elsayed, 1997; Knudsen & Oen, 2003). The underlying physical mechanisms for these manifestations are cerebral oedema, contusions and lacerations, as well as hemorrhages in the meninges (protective tissues around the brain), brain substance, nerve roots and ventricles (fluid-filled spaces within the brain and spinal cord) that may result from extreme relative motion between the skull and brain during exposure to high overpressures. Hastings (1990, 1995) reported ‘acoustic stunning’ in four blue gouramis *Trichogaster trichopterus* (Pallas) exposed for approximately 8 min to a 150 Hz pure tone with a peak pressure of 198 dB re 1 μPa. Three out of four of these fish recovered. The loss of consciousness exhibited by these fish could have been caused by neurotrauma, especially since this species has a bubble of air in the mouth cavity located near the brain that enhances hearing (Yan, 1998; Ladich & Popper, 2004). Thus fish with swim-bladder projections or other air bubbles near the ear (e.g. butterfly fish, some squirrelfish and soldierfish, and other species) could potentially be susceptible to neurotrauma when exposed to high SPLs.

Elsayed (1997) conducted a series of investigations using terrestrial animal models to examine biochemical responses in tissues to blasts. The investigator found two responses that correlate with blast overpressure (1) depletion of antioxidants and (2) lipid pre oxidation. Cernak *et al.* (1996) also examined biochemistry related to neurotrauma in blast injury. They also found lipid pre-oxidation products as well as increased levels of lactate and calcium ions and decreased levels of glucose and magnesium and zinc ions. Changes in lactate and glucose levels indicate changes in metabolism and energy in the damaged tissue, while changes in ion concentrations indicate cellular disruption and damage. Cernak *et al.* (1996) postulate that afferent neural signals from injured organs (such as lungs) could impair central nervous system (CNS) function and contribute to further damage over time. The biochemical mechanisms of acoustic traumas and barotraumas, as well as their acoustic thresholds, however, remain undefined.

Studies by several investigators (*e.g.* Yelverton *et al.*, 1975; Traskey, 1976; Wiley *et al.*, 1981; Hastings, 1990, 1995; Caltrans, 2001; Govoni *et al.*, 2003, 2008) have reported mortality of fish exposed to blasts or other types of high-level sounds. In the only study using pure tones, Hastings (1990, 1995) found that *C. auratus* could not survive 2 h continuous wave (CW) exposures at 250 Hz with a peak sound pressure of 204 dB re 1 μPa, and *T. trichopterus* could not survive 0.5–h CW exposures at 150 Hz and a peak level of 198 dB re 1 μPa.

Yelverton *et al.* (1975), in an important study reported in the grey literature, provides the most definitive study of the gross effects of sound generated by underwater blasts on fishes, although it should be noted that blast pressure waves consist of an extremely high peak pressure (called overpressure) with very rapid rise times (<1 ms) that are somewhat faster than any other sound source. Yelverton *et al.* (1975) exposed eight different species of fish, five physostomes and three physoclists to blasts. The physostomes were top minnow *Gambusia affinis* (Baird & Girard), *C. auratus*, carp *Cyprinus carpio* (L.), *O. mykiss*, and channel catfish *Ictalurus punctatus* (Rafinesque) and the physoclists were guppy *Poecilia reticulata* Peters, *L. macrochirus*, and large mouth bass *Micropterus salmoides* (Lacépède). The test specimens ranged from 0.02 g (*P. reticulata* fry) to 744 g body mass (large *C. carpio*) and included small and large animals from most species. The fish were
exposed to blasts having extremely high peak overpressures with varying signal durations. Yelverton et al. (1975) found a direct correlation between body mass and the acoustic impulse (i.e. time integral of the pressure waveform as defined previously) which caused 50% mortality. Traskey (1976) also reported significant differences between adult fishes and salmonid and clupeid fry in the lethal blast overpressure from buried seismic charges.

While these data are important, there is concern regarding the gross pathological examinations of the fish and the experimental design used by Yelverton et al. (1975). The primary concern is a lack of controls to evaluate fish-handling procedures. Without these controls and professional pathological examinations there is an issue in quantifying damage to fish strictly attributable to exposure to the explosions compared to damage attributable to handling.

At the same time, there are very few studies known to have involved a professional fish pathologist to do full necropsy and histopathology after noise exposure (Govoni et al., 2003, 2008; Abbott et al., 2005; Popper et al., 2007). Although Govoni et al. (2003, 2008) found damage from blasts in small juvenile and larval fish based on full necropsy and histopathology, Popper et al. (2007) showed that there were virtually no effects on any body system in O. mykiss as a result of exposure to SURTASS LFA sonar. Abbott et al. (2005) examined fish following their exposure to 4 min of driving of concrete piles at the Port of Oakland. While these investigators found some pathology, it did not differ between sound-exposed and control groups, suggesting that any pathology may have been from prior effects or disease. Indeed, these results highlight the necessity of involving a professional fish pathologist in studies of this type because it is exceedingly easy to impose artefacts in the tissue as a result of mishandling or poor tissue preparation.

Significantly, the pile driving studies by Caltrans (2001), Abbott & Bing-Sawyer (2002) and Caltrans (2004) did not involve a professional fish pathologist. In addition, Abbott & Bing-Sawyer (2002) sacrificed fish by putting them in plastic bags and placing them on ice, whereas Caltrans (2004) sacrificed fishes in other ways but then froze the animals. While differences were reported between exposed and control animals in pile-driving studies [though one study (Caltrans, 2001) showed damage in 70% of controls that were attributed to handling], it is possible that handling post stress from exposure to sound resulted in the reported pathology, rather than being caused directly by exposure to pile-driving signal per se. Moreover, it is widely known that freezing damages tissues and cells and such damage could make recognizing actual effects of exposure to sound difficult because it would ‘mask’ effects of sound exposure in both experimental and control animals.

EGGS AND LARVAE

In considering fishes, it is important to not only think in terms of adults but also in terms of fish eggs and larvae. Whereas it is possible that some (though not all) species of fish would swim away from a sound source, thereby decreasing exposure to sound, larvae and eggs are often at the mercy of currents and move very slowly, if at all. Eggs are often stationary and thus could be exposed to extensive anthropogenic sound if it is presented in the area, including sound transmitted through water (i.e.
eggs within the water column) or substrate (e.g. eggs deposited within substrate, such as salmonid redds).

Data on effects of sound on developing eggs and larvae are very limited. There is some suggestion in the literature that developing larvae have different levels of sensitivity to mechanical stimulation at different stages of development (Piper et al., 1982; Jensen & Alderdice, 1983, 1989; Dwyer et al., 1993). However, virtually all these studies used explosions or large mechanical shocks. For example, Jensen & Alderdice (1989, but also see Jensen & Alderdice, 1983) did controlled drops of trays of five salmonid species from different heights and then examined for mortality over time. Jensen and Alderdice found that there was greatest mortality to eggs dropped before they had started to divide (activation), whereas other investigators found no effects. Post et al. (1974) dropped O. mykiss eggs in a way designed to mimic the effects of the seismic effects of nearby nuclear blasts and found no effects on eggs of any ages, whereas Smirnov (1959) mechanically agitated O. keta salmon eggs (no description of this agitation was provided other than that it was a mechanical stimulus of some sort) and found different levels of mortality at different ages post the start of cell division.

Banner & Hyatt (1973) conducted the only peer-reviewed study on the effect of sound on eggs and development in fish, and it was never followed up with additional investigations. They found an increased in mortality eggs and embryos of the sheephead minnow Cyprinodon variegatus Lacépède (subgenus not specified) in 20 l glass aquaria exposed to broadband noise (100–1000 Hz) that was about 15 dB above ambient sound level. The sound did not affect hatched fry of C. variegatus, and neither eggs nor fry of longnose killifish Fundulus similis (Baird and Girard) were affected. Banner & Hyatt (1973) also found that the larval growth was significantly less in the noise-exposed larvae of both species than in the larvae raised in ambient noise. [Interestingly, these findings with parallel studies show that shrimp exposed to noise has slower growth than controls not exposed to noise (Legardère, 1982).] While these results are of interest, they were from only two species subject to relatively low noise levels and for a limited time period.

Other than the blast study by Govoni et al. (2003, 2008) on small juvenile and larval L. rhomboides and L. xanthurus, which correlated injury with the impulse metric, very little is actually known about effects of sound of any type on eggs and larvae, and this remains an important issue for future research. Indeed, there are several issues that must be considered with regard to the effects on eggs and larvae of exposure to sound. These include (1) immediate effects as measured by mortality; (2) long-term effects, even after the termination of the insonification, as measured by mortality; (3) long-term effects from which recovery is possible if the fish is not subject to predation or other factors that kill it during the recovery time; (4) effects on egg development and viability, (5) effects on short and long-term growth of the developing larvae and young fish in the presence of sound and, or after termination of sound; (6) effects of exposure to sound on the development and function of various organ systems; (7) effects on larval behaviour and longer-term survival; and (8) potential differences in effects at different stages of development.

Several other sets of data are worth noting. A non-peer-reviewed study on the effects of 100, 800 and 5600 Hz sounds from 105 to 167 dB re 1 μPa on eggs and embryos in Lake Pend Oreille (Idaho, U.S.A) reported normal survival or hatching (Bennett et al., 1994). However, few data were provided that could be used to
evaluate the results. Kostyuchenko (1973) worked with marine fishes, to determine the effects of seismic air-gun sounds on eggs. The investigator reported damage to eggs at up to 20 m from the source. Similarly, Booman et al. (1996) investigated the effects of seismic air guns on eggs, larvae and fry and found significant mortality in G. morhua, saithe Pollockis virens (L.) and Atlantic herring Clupea harengus L. at a variety of ages but only when the specimens were within about 5 m of the source, and the most substantial effects were to fish that were within 1.4 m of the source. These authors also reported damage to neuromasts (sensory structures with sensory hair cells) of the lateral line system and to other organ systems; however, data are limited to just a few species and need replication. At such close distances to the air-gun array, the hydrodynamic motion (i.e. bulk oscillatory fluid motion) would be huge, but the received sound pressure and fluid motion were not measured in this study.

A recent unpublished report examined the effects of a mid frequency sonar (1.5–6.5 kHz) on eggs and larvae of several species (Jørgensen et al., 2005; Kvadsheim & Sevaldsen, 2005). While the study dealt with continuous pure tones, it did involve a paradigm that included full pathology on the tissue of the experimental animals. Larval and juvenile fish were exposed to simulated sonar signals to investigate potential effects on survival, development and behaviour. The study used C. harengus (standard lengths 2–5 cm), G. morhua (standard length 2 and 6 cm), P. virens (standard length 4 cm) and spotted wolffish Anarhichas minor Olafsen (standard length 4 cm) at different developmental stages.

Fish were placed in plastic bags 3 m from the sonar source and exposed to between 4 and 100 pulses of 1 s duration of pure tones at 1.5, 4 and 6.5 kHz. SPLs at the location of the fish ranged from 150 to 189 dB (re 1 μPa). There were no effects on fish behaviour during or after exposure to sound (other than some startle or panic movements by C. harengus for sounds at 1.5 kHz) and there were no effects on behaviour, growth (length and weight), or survival of fish kept as long as 34 days post-exposure. All exposed animals were compared to controls that received similar treatment except for actual exposure to the sound. Excellent pathology of internal organs showed no damage as a result of sound exposure. The only exception to almost full survival was exposure of two groups of C. harengus tested at an SPL of 189 dB re 1 μPa, where there was post-exposure mortality of 20–30%. While these were statistically significant losses, it is important to note that this sound level was only tested once and so it is unknown if this increased mortality was due to the level of the test signal or to other unknown factors.

In a follow-up unpublished analysis of these data, Kvadsheim & Sevaldsen (2005) sought to understand whether the mid-frequency CW signals used by Jørgensen et al. (2005) would have a significant effect on eggs and larvae in the wild exposed to this sonar. The investigators concluded that the extent of damage or death induced by the sonar would be below the level of loss of eggs and larvae from natural causes, and so no concerns should be raised with regard to sonar effects. Kvadsheim & Sevaldsen (2005) suggest that what might be of concern is when the CW signal is at the resonance frequency of the swim bladders of small clupeids. If this is the case, the investigators predict (based on minimal data that is in need of replication) that such sounds might increase the mortality of small clupeids that have swim bladders that would resonate.

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In summary, the few studies of the effects on eggs, larvae and fry are insufficient to reach any conclusions with respect to the way sound would affect survival. Moreover, most of the studies were done with seismic air guns or mechanical shock and these are stimuli that are very different than those produced by pile driving. The results suggesting some damage and death need to be followed up in a way that would be relevant to pile driving and the characteristic sound transmitted through water and substrate.

LATERAL LINE

The lateral line system consists of a set of receptors found on the surface of the body in all fishes (Dijkgraaf, 1963; Coombs and Montgomery, 1999; Webb et al., 2008b) that detect water motion within a few body lengths of the fish (e.g. Denton & Gray, 1989). The sensitivity of the lateral line system is generally below a few hundred hertz, and there appears to be a dichotomy of function between lateral line receptors that lie below the surface of the skin in canals and those that lie on the surface of the body (Montgomery et al., 1997).

The only study on the effect of exposure to sound on the lateral line system suggests no effect on these sensory cells (Hastings et al., 1996). While not directly relevant to potential effects due to sound, Denton & Gray (1993) showed that mechanical stimulation of the lateral line of clupeids may cause damage by decoupling the cupulae from the neuromasts. Loss of the attachment between the cupula and neuromast would result in dysfunction of the lateral line. In their study, Denton and Gray did not consider the hair cells themselves, nor did Hastings et al. (1996) look at any effects on coupling of the cupula.

The work from Denton & Gray (1993) suggests, however, that potential damage to the lateral line is possible when fish are located in close proximity to an intense sound source, and future studies of the lateral line should consider the coupling issue because damage to the coupling would have a significant effect on the function of the lateral line, just as would damage to the sensory cells themselves. This is reinforced by the very limited work on the lateral line by Booman et al. (1996).

CONCLUSIONS

While there is a growing concern about the effects of anthropogenic sounds on fishes, an evaluation of both the peer-reviewed and grey literature leaves the strong impression that we still know very little about the effects of such sounds on fish (and even less so on invertebrates). Moreover, it is clear that the available literature is equivocal in what it teaches and leaves great gaps that need to be filled before meaningful noise exposure metrics or reliable noise exposure criteria can be developed. Indeed, there is some indication that some sounds, under some conditions, with some species, may cause some kind(s) of effects. But, extrapolation to the same sounds under other conditions, or to other fish species, or to other effects, is not possible.

The basic need right now is for a set of systematic studies that examine effects of highly quantified sounds of various types (e.g. different types of pile driving, different
seismic sources, etc.) on a wide range of morphologically and taxonomically diverse species of interest. While it is clearly impossible to examine effects of all sound sources on even a tiny fraction of the 29,000+ extant species, systematic studies are needed that examine fishes that represent species that differ with regard to (as examples) (1) hearing capabilities and ear and lateral line structures; (2) life styles (e.g. bottom v. mid water fishes), species of different sizes (as well as eggs and larvae); and (3) behavioural responses to potential ‘fright’ stimuli (e.g. fishes that stay in one location v. species that swim away).

There are also issues with the nature of the exposure including the number of exposures of individual animals, the intervals between exposures, and the spectrum of the received signal. Additionally, there are methodological issues in experimental design that include not only the need for excellent controls and pathology but also careful signal measures and use of proper metrics that not only define the received sound field but also allow for comparisons of signals of different types (e.g. pile driving and seismic air guns).

Finally, and perhaps most importantly, there is a need for behavioural studies that actually examine the responses of wild fish to anthropogenic sounds. Almost all studies to date have involved caged fish. And even in the few studies where free-living fish have been observed by underwater video (Wardle et al., 2001) or sonar (Slotte et al., 2004), not enough is known about how fish, individually and over a large region around a sound source, react to the source. Most importantly, it is not possible to extrapolate in any way from studies of caged fish to wild animals. Fish in cages are highly restricted in movements, not only by cage walls but also often by crowding. It is highly likely that fish ‘sense’ the limits of their (caged) environment and this strongly alters the responses of the fish to a potentially noxious stimulus. Whereas in the wild a fish may respond to a loud sound by rapidly swimming away, this is impossible in a cage, and the fish may sense that they cannot move far and thus show no response whatsoever.

In conclusion, the extent of data needed to enable investigators, regulators and industry to understand the effects of sound on fish is rather staggering. However, without such data, obtained in some systematic way and with excellent controls and peer review, it will be impossible to develop clear sound-exposure metrics and criteria that could help in making predictions on potential effects of sounds on fish.

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