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From: Commanding Officer, Naval Submarine Medical Research Laboratory
To: Chief of Naval Operations (N774T), 2000 Navy Pentagon, ATTN: Mr. Joseph S. Johnson, Washington, DC 20350-2000

Subj: RESONANCE WHITE PAPER

Encl: (1) White Paper entitled "A review of the potential for in vivo tissue damage by exposure to underwater sound"

1. Enclosure (1) is forwarded as part of the preparation of the Final Environmental Impact Statement for the Surveillance Towed Array Sonar System Low Frequency Active. The white paper discusses the potential impact of resonance on tissue.

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A review of the potential for *in vivo* tissue damage by exposure to underwater sound

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A. Objective: The principal objective of this review is to determine a best estimate, based on a comprehensive review of the extant literature, of the intensity of sound needed to create tissue damage, with emphasis on the frequency band [100 to 500 Hz]. Two potential mechanisms are examined: 1) direct acoustic impact on tissue, and 2) indirect acoustic impact on tissue surrounding a resonant structure. In general, the resonant structures of interest are expected to be those that are filled with air. These structures are key because it is for these structures that there will be a significant impedance mismatch[1] at the tissue and therefore both mechanisms hypothesized can be involved. That is, the tissue can be directly driven to damage. Alternatively, the air in the structure can resonate, and the tissue can be damaged due to the displacements caused by the resonance exceeding the tolerance of the tissue or due to the resonance forcing the tissue to impact on surrounding hard structures.

B. Background: There have been a number of questions raised regarding the potential damaging effects of resonance on the internal organs of marine mammals. In particular it has been argued that the Surveillance Towed Array Sensor System, Low Frequency Active (SURTASS LFA) sonar induces such resonance. The argued hypothesis is that resonance will increase stress on tissue to the point of damage. This concept results from two physical mechanisms that have been bundled – a) resonance and b) tissue damage. **It is important to note that resonance does not necessarily equal damage and that damage is not always linked to resonance.**

Resonance is a phenomenon whereby the amplitude of vibration is increased due to a match between the frequency of the signal and the elastic properties of the material. The material can be anything from steel to biological tissue to fluids to gas. The key properties for vibration of an elastic structure are its mass and stiffness, as well as the friction (damping) associated with the material. For material embedded in other materials the damping could well arise from the properties of the surrounding medium, including contiguous material and structure.

A familiar form of resonance is that associated with musical instruments. The resonance can come from open cavities, enclosed cavities, strings, and binding materials. For example, when a guitar string is struck, it vibrates best at a particular frequency depending on the material of the

[1] 10 Log [ratio of specific impedance (water/air)] = 35 dB

string and the tension. This frequency of best vibration is the resonant frequency for that string when set up in that fashion. Changing one of the parameters, such as the tension, will alter the resonant frequency.

The above is also true for animals and humans. There are strings, cavities, and binding materials within the body that will resonate when driven at the correct frequency. An obvious example is the vocal cords and speech. Speech has resonant peaks called formants that are a direct consequence of the resonant properties of the vocal cords and the air cavities associated with the vocal tract.

Resonance in biological structures is not always driven internally as in the case of speech. Sounds or vibrations outside the body can make structures within the body resonate. For example, the bass at a concert can make your body vibrate. It is important to note that you can have vibration without resonance. Examples include the vibration felt when a truck rumbles by. Some of the vibration may be due to resonance and some may be due to the high vibration level in the structure upon which the person is standing. As is clear from the examples above, it is easily possible to get resonance without harm.

It is also possible to drive a structure into resonance and induce vibration sufficient to damage the tissue. However, the amount of vibration required to make this happen is a property of the tissue itself and the ability of the structure itself to resonate. Most of the resonant structures in the body are highly damped and cannot generate high multiples of the amplitude of the signal. This is due to the internal organs such as the lung being surrounded by other structures and fluids that do not resonate at the same frequency and thus impede the resonance of the structure.

C. Resonance Studies: There have been some recent studies of resonance in the human body underwater, most notably for the skull (Hanson and Cudahy, 1998) and the lung (Martin et al., 2000). The resonant frequency of the skull is between 600 and 900 Hz underwater. The data available from these studies indicate that there is no effect of depth. Thus, human skull resonance is above the frequency range of the SURTASS LFA system (100 – 500 Hz).

The resonant frequency of the male human lung close to the surface is 30 – 40 Hz (Martin, et al, 2000). As depth increases to 120 feet (36.6 meters), the resonant frequency increases to 80 Hz. This is in contrast to the calculations of Tepley (2001) suggesting a resonant frequency for the lung of 60 Hz for females at 20 feet. The data that Tepley references (which are for male divers) show a resonant frequency for the lung near 60 Hz at 65.6 ft (20 m). The measured data also contradict Tepley's assertion that the lung resonant frequency will be 141 Hz for male divers at 50 feet. As indicated, the resonant frequency for male divers (Martin, et al, 2000) was approximately 60 Hz at 66 feet, an error by a factor of more than two. This suggests that Tepley is using the wrong equations or his selection of values for parameters are in error. The increase in resonant frequency with increasing depth reflects the fact that the density of the gas increases (in effect the gas becomes stiffer) while the volume of the lung stays close to constant. The resonant frequency increases more slowly the deeper the depth, so the resonant frequency of the lung will not reach 100 Hz until depths of 300 feet (91.4 m) (using equations from Martin et al., 2000) or greater. It should be noted that at the resonant frequency of the lung, which should theoretically be the frequency that will cause damage at the lowest intensity, the lung will not experience damage until the intensity exceeds 180 dB sound pressure level (SPL) re 1 micropascal (Cudahy

et al., 1999). The relation between lung resonance, intensity and tissue damage is discussed in more detail below.

Resonant frequency can also change depending on the volume of a gas-filled space; i.e., the smaller the volume, the higher the resonant frequency. For example, the resonant frequency of a mouse lung underwater is around 350 Hz, whereas the human lung resonant frequency at the same depth is around 35 – 40 Hz. Higher resonant frequencies have been observed in human skull measurements, but preliminary calculations suggest that resonance at these frequencies is not very highly tuned (unpublished data). That is, the amplitude does not increase as much at these higher frequencies as it does at the resonant frequency of the lung.

Results such as those just described were obtained with terrestrial mammals that were artificially placed underwater. It must be noted that marine mammals evolved to cope with the underwater medium, where they routinely accommodate vastly greater changes in pressure than any terrestrial vertebrate. For example, the pressure difference between the top of Mt. Everest and the bottom of Death Valley corresponds to a change in underwater depth of only 23 ft (7 m). This is a fraction of the routine dive depths for most species of marine mammals.

This ability of marine mammals to adapt to dramatic fluctuations in pressure is supported by abundant anatomical evidence.. For example, marine mammal lungs are reinforced with more extensive connective tissues than their terrestrial relatives. These extensive connective tissues, combined with the probable collapse of the lung alveoli at the depths at which LFA signals can be heard, make it very unlikely that significant lung resonance effects could be realized. Alveolar collapse is not the only change in the lungs. The trachea can also collapse because cartilage armor rings are often incomplete. Air that does not escape the alveoli is quickly absorbed during diving due to the high partial pressure of the gas (Berta and Sumich, 1999). Complete lung collapse occurs at depths of 82-164 ft (25-50) meters for Weddell seals (Falke et al., 1985), 246 ft (75 m) for the bottlenose dolphin (Ridgway and Howard, 1979), and probably occurs in the first 164-328 ft (50-100 m) for most marine mammals (Berta and Sumich, 1999).

The nasal air sacs are too small to be relevant to LFA transmissions. Furthermore, these nasal diverticuli are clearly involved in sound production (Heyning and Mead, 1990). The pressure fluctuations that accompany the emission of echolocation clicks or communicative sounds must be substantial, so these tissues should also be relatively resistant to damage from external sound sources.

D. Potential for Tissue Damage: The following summary review of potential damaging effects of sound was therefore broken into two major parts: 1) direct damage to tissue, and 2) tissue damage in air-filled spaces. The results are primarily based on the Gerth and Thalmann (1999) presentation at the Underwater Sound Conference of January 25, 1999, and summary test data (along with more recent analysis) on animal sound exposure from the SURTASS LFA EIS Technical Report Number 3 (Cudahy et al., 1999).

1. Direct Tissue Damage: Three **mechanisms are examined and discussed below; transluminal effects on the vascular system, cavitation damage and direct shear damage to tissue.**

a. Transluminal damage – The intensity for the onset of transluminal (hydraulic) damage is on the order of 190 dB or greater (Gerth and Thalmann, 1999). This result is

extrapolated from the effects of the Valsalva maneuver on hydraulic pressure in the vascular system, and is summarized below. Comparing an estimate of the sound pressure level creating a peak pressure in the arterial vasculature to the peak pressure of 75 mmHg due to the Valsalva maneuver yields approximately 190 dB as a safe level. This level is conservatively founded on the basis of variations during normal everyday activities on the order of 70 mmHg occurring in cerebral transmural pressure.

b. Cavitation damage – Vascular damage thresholds from cavitation in individuals exposed to low frequency sound are in the 240 dB regime (Gerth and Thalmann, 1999). This is 25 dB above any levels that the LFA source is capable of producing.

c. Tissue shear damage – The intensity for shear tissue damage is on the order of 190 dB or greater (Gerth and Thalmann, 1999). This is based on estimates of tissue displacement (in micrometers, $m \times 10^{-6}$) of 14.3 and 1.43 for 100 and 1000 Hz in air-filled spaces. This range of displacements would suggest the potential for damage of capillaries at the lowest LFA frequencies at 190 dB or higher, but larger vessels would be unaffected.

2. Tissue Damage in air-filled spaces: Shear damage due to resonance would apply primarily to the lungs of a marine mammal, with caveats as discussed below. It should be noted that many of the sinus cavities in marine mammals do not appear to be air-filled nor connected to the auditory spaces. Furthermore, resonant frequencies for such cavities are probably greater than 500 Hz based on their size.

Critical issues to consider in examining resonance effects is the tuning of the resonance and the damping due to contiguous body structures. The degree of tuning (defined as Q, with high Q indicating sharper tuning) that has been measured *in vivo* in the lungs (of pigs and humans) is a Q from 3 to 5, ($3<Q<5$, Martin, et al, 2000). There are no data to support a Q of 10 as a good estimate of the degree of tuning in an air-filled space. In general, the internal organs of mammals are highly damped. Examining fishes, extensive measurements of the Q of swimbladders at resonance (covering a wide range of species and sizes) support an *in vivo* range of Q from 1.0 to 6.1. (Love, 1978). Thus, an educated estimate of the Q for other gas-filled structures, which are much less free to move than the lung, would generally be very small, even less than the [$1<Q<6$] range encompassing both lung and fish swimbladder measured results.

Extrapolations from human data are best at very shallow (10 feet or less) depths where the lung of the marine mammal is fully expanded. At greater depths the contraction of the lungs and the different supporting structure will start to play a large role in determining the resonance frequency and the amount of damping by the lung.

For the non-shallow-water lung resonance case, the best estimate is from the mouse data (over 400 animals tested, see LFA EIS Technical Report 3, Table 1 [Cudahy et al., 1999]). Those data indicate that lung damage would start at about 181 dB. Lower thresholds were originally estimated for a small number of guinea pigs (approximately 174 dB). Additional mice and guinea pigs have been tested since that report. These data did not significantly change the resonant frequencies or damage risk thresholds for mice cited in that report but did result in small changes to the damage risk threshold for guinea pigs. These data indicate that the inception of damage threshold is 184 dB for the mice and above 180 dB for the guinea pigs. The total number

of mice tested for lung damage is over 400, but the number of guinea pigs is less than 10, thus we have much more confidence in the values based on the mice data. This level is at primary lung resonance underwater, so no correction factor for Q is required. It should be noted that the exposure times in these tests were significantly longer in duration than would be experienced by an LFA signal (5 minutes vs. 60-100 seconds for LFA). All of these data are from terrestrial mammals that are clearly not well adapted in lung mechanism or musculature to an underwater environment for either acoustic pressure exposure or hydraulic pressure exposure.

E. Summary: As discussed above, each of the *in vivo* and theoretical studies related to potential tissue damage from underwater sound support a damage threshold on the order of 180 to 190 dB. The *in vivo* results are based on underwater measurements of terrestrial mammals, including humans, as well as extrapolation from in-air results. As the direct and controlled measurement of damage levels in marine mammals is tacitly proscribed, this body of data must serve as the basis for the current assessment. Much of the results provided and discussed here are clearly directly relevant, and independent of terrestrial vs. marine environment.

However, where they are not, it seems very plausible that marine mammals that have evolved in an ambient hydrostatic pressure environment spanning several orders of magnitude [$1{:}10^3$] of dynamic range would be pre-disposed to have an innately more rugged physiology for handling pressure changes than terrestrial animals.

With regard to more sudden changes in pressure, many marine mammals breach (i.e., leap out of the water). The drastic pressure change that accompanies the transitions from water to air and back would favor animals with relatively robust tissues, including their ears. By contrast, human springboard and platform divers suffer permanent hearing loss from their sport unless they use special protection.

Further, the limiting values of resonant shear stress were obtained on animals (guinea pigs and mice) with significantly lower musculature and body mass than the marine mammals of interest to this study.

Marine mammals also encountered much louder sounds in their evolutionary histories than ancestral mice and rats did. Undersea seismic events produce enormously powerful sounds, for which the only terrestrial analog might be an explosive volcanic eruption. It would be rare for a rat to be close to such an eruption, or survive it, but pelagic marine mammals probably pass by seismically active areas many times in their lives. Lightning strikes produce extremely loud sounds in air and water, but these sounds carry much farther underwater. Finally, marine mammals produce the loudest sounds in the animal kingdom. Their bodies must have evolved to deal with their own sounds, and with the sounds of their close neighbors.

Finally, it is important to note that research under the auspices of the Office of Naval Research continues on these issues, with the specific objective of more closely defining the threshold at which physiological harm can begin. In the interim, the continued use of 180 dB as a mitigation guideline appears to be fully validated by all controlled testing and theoretical extrapolation accomplished to date.

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