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## **Anthropogenic Noise as a Stressor in Animals: A Multidisciplinary Perspective**

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Consequences of extreme noise exposure are obvious and usually taken into some consideration in the management of many human activities that affect either human or animal populations. However, the more subtle effects such as masking, annoyance and changes in behavior are often overlooked, especially in animals, because these subtleties can be very difficult to detect. To better understand the possible consequences of exposure to noise, this review draws from the available information on human and animal physiology and psychology, and addresses the importance of context (including physiological and psychological state resulting from any previous stressor exposure) in assessing the true meaning of behavioral responses. The current consensus is that the physiological responses to stressors of various natures are fairly stereotyped across the range of species studied. It is thus expected that exposure to noise can also lead to a physiological stress response in other species either directly or indirectly through annoyance, a secondary stressor. In fact many consequences of exposure to noise can result in a cascade of secondary stressors such as increasing the ambiguity in received signals or causing animals to leave a resourceful area, all with potential negative if not disastrous consequences. The context in which stressors are presented was found to be important not only in affecting behavioral responses, but also in affecting the physiological and psychological responses. Young animals may be particularly sensitive to stressors for a number of reasons including the sensitivity of their still-developing brains. Additionally, short exposure to stressors may result in long-term consequences. Furthermore, physiological acclimation to noise exposure cannot be determined from apparent behavioral reactions alone due to contextual influence, and negative impacts may persist or increase as a consequence of such behavioral changes. Despite the lack of information available to managers, uncertainty analysis and modeling tools can be coupled with adaptive management strategies to support decision making and continuous improvements to managing the impacts of noise on free-ranging animals.

Physiological responses to stressors and the consequences for an individual or a population have been debated in various arenas, partly because they are studied by scientists from widely different disciplines. Here we summarize the knowledge acquired over the recent decades in different disciplines ranging from animal physiology to human psychology. Noise is a ubiquitous stimulus with the potential to act as a stressor, which has been growing in intensity in the oceans over recent decades. Paradoxically however, the effects of noise on the health and wellbeing of humans, terrestrial animals and, most recently, marine animals remain controversial. This paper provides an overview of the physiological responses to various stressors in humans and animals across various scientific fields and their consequences. We also summarize the current state of knowledge about these responses with specific regard to noise in humans and laboratory animals. Then, we extrapolate from this overview to fill some of the gaps concerning the physiological responses induced by noise in humans and free-ranging animals, highlighting marine species as they often rely heavily on acoustical communication as light does not travel far in water (Hatch & Wright, this issue). The importance of the context in which stressors are presented is also emphasized. Finally, we attempt to identify how and to what extent noise affects the health, wellbeing and viability of wildlife populations. Working definitions of several terms related to “stress” used throughout this paper are presented in Wright & Kuczaj (this issue).

Noise levels and exposure to those levels are measured differently in air and water. The reasons for this are varied, complex and beyond the scope of this paper. More information can be found in Clark & Stansfeld (this issue) and Hatch & Wright (this issue).

## Physiological Stress Responses

### *Pathways of response*

Two major systems are known to be involved in stress: the sympathetic nervous system (SNS) and the hypothalamic-pituitary-adrenal (HPA) axis. These systems are activated very rapidly and have broad impacts on diverse aspects of physiological functioning. The concerted effort of these and other critical endocrine and neural systems ultimately comprises an organism's response to a stressor (see Deak, this issue; Romero & Butler, this issue). Indirect measures of SNS activation (e.g., increased heart rate, blood pressure, or hyperthermia) or direct measures of SNS output from the adrenal medulla (plasma concentrations of catecholamines – epinephrine and norepinephrine) and HPA activation (corticosteroid concentrations in plasma, tissue or excrement) are often collectively or individually used to indicate the severity of a stressor. Importantly, “stress responses” can also occur to stimuli that are merely arousing, such as sexual activity (see Deak, this issue). Thus to avoid misinterpretation of physiological and behavioral measures observers should take into consideration baseline information and should verify the presence of a threatening context to determine whether the observed changes actually reflect a stress response and not arousal *per se*.

The SNS response to stressors can be detected within seconds of the perception of a punctate stressor (i.e., one with a sharp onset). However, many stressors are not punctate but rather develop over a long period. In the cases of these building stressors, the SNS activation is often described as a steadily escalating “tone” where general SNS activity increases relatively slowly over the course of hours, days or months, leading to escalated metabolic demand and gradual wear-and-tear on physiological systems that may eventually culminate into physiological failures (see Deak, this issue). These contrasting SNS responses make it particularly difficult to identify a causal relationship between anthropogenic noise and SNS response because anthropogenic noise arises across a wide range of time frames. Noise can be punctate, such as occurs in seismic survey blasts, or noise can gradually increase over a given area and persist for extended periods (if not permanently), such as is the case with the increase in ambient noise throughout the world's oceans resulting from shipping traffic. In the latter situation, the major stressor is unlikely to be the noise itself, unless levels cross some threshold of tolerability, but rather the increasing masking (i.e., the “drowning out” of a signal in the noise) of mating calls, social communication, echolocation of prey and other important signals.

Development of the response by the HPA axis is somewhat slower than that of the SNS response, but its impact is just as profound, albeit on a somewhat more protracted timeline. Immediately upon perception of a stressor a chain of events in the HPA axis triggers the production of glucocorticoids (GCs: e.g., corticosteroid) by the adrenal cortex (see Deak, this issue; Romero & Butler, this issue). The stress hormones are then quickly released into the bloodstream (usually within 3-5 min after activation by stressor onset) where they are rapidly distributed throughout the body to initiate a systemic response to the threat (Romero & Butler,

this issue; Sapolsky, Romero, & Munck, 2000). This can be problematic for researchers as it limits the time during which they can gain valid information on GC levels, as an animal's blood GC levels rises very quickly after the individual perceives the threat of capture, regardless of whether it is yet in hand or not.

In general, the more intense the stressor, the greater the amount of GC released. Once the stressor ends, GC levels return to baseline concentrations as a consequence of both the ending of the stimulus and GC negative feedback on the pituitary gland and hypothalamus (see Romero & Butler, this issue). If the stressor persists or occurs at frequent intervals the animal becomes chronically stressed (how frequent depends upon the stressor). This is generally manifested as a long-term increase in GC secretion due to two mechanisms: repeated secretion in response to repeated stressors and a failure of GC negative feedback (Dallman & Bhatnagar, 2001).

### *Consequences of the stress response*

GCs (both independently and in combination with other components of the stress response) cause a variety of behaviors in free-living animals that are heavily context dependent (see Deak, this issue; Romero & Butler, this issue). However, the broad effects of GCs are to shift the animal away from normal life-history behavior to emergency behaviors (see Romero & Butler, this issue). Examples include increasing activity, the scattering of a group, shifting behavior from reproduction to feeding, and abandonment of breeding territories. These behaviors are adaptive in natural environment in the short-term, but may become maladaptive in response to novel human disturbances and/or repeated or chronic exposures.

Detrimental physiological effects can also appear if the stressors remain, or additional stressors are presented, prolonging the GC response over an extended period. A number of pathological effects appear after 2-3 weeks, which are very consistent across species studied (mainly in captivity: see Romero & Bulter, this issue). These include, but are not limited to, diabetes, immune suppression and reproductive malfunction. In fact, the assault on reproductive function is threefold, involving prolonged behavioral changes, such as reorientation of the individual's behavior away from reproduction, psychological effects, such as decreases in libido, and physiological impairment of reproduction (see Deak, this issue; Romero & Butler, this issue). Interestingly, in many human couples seeking artificial conception, the underlying infertility is induced by being stressed (Homan, Davies & Norman, 2007; Wischmann, 2003).

Other long-term consequences of persistent high GC levels include accelerated aging and a slow disintegration of body condition (see Romero & Bulter, this issue). It is clear that accelerated aging in combination with decreased reproductive function presents a double-blow to the fitness of an individual. There are obvious implications for the population if such effects are widespread, but more subtle consequences also exist (see Deak, this issue; Romero & Butler, this issue). For example, if cultural exchange from one generation to the next is limited by the shortened lifespan and premature death of the older generation, certain skills

or valuable information (e.g., regarding a reliable watering hole in times of drought in elephants) may be lost.

One further example of the consequences of persistently elevated GC levels is psychosocial dwarfism (Green, Campbell & David, 1984), a rare but documented inhibition of growth in human children due to altered growth hormone function (see Romero & Butler, this issue). It appears possible (although speculative at this point) that prolonged high levels of GCs may explain why sperm whales (*Physeter macrocephalus*) in the Gulf of Mexico (GoM) are significantly smaller than others elsewhere in the world (Jaquet 2000). Humans have very extensively used the Gulf ever since the discovery of the Mississippi River: activity that has continuously intensified.<sup>1</sup> The apparent dwarfism in the resident sperm whales might be a symptom of the heavily stressed state of the animals due to that activity. This condition would probably not be the result of exposure to noise alone, but rather the cumulative action of noise with various other stressors such as reduced prey availability and contaminants. Genetic differences and other factors might also be involved.

GCs can also have toxic consequences for neurons (i.e., cause neuron death) in the very young brain, which is probably why GC responses to stress are attenuated during the perinatal period (Sapolsky, 1992). Only severe stressors elicit GC release by the newborn during this time, such as parental deprivation or neglect, possibly as a consequence of parental/alloparental poor health (for any reason), or maternal separation, perhaps due to increased foraging times. The period of attenuation extends up to about a week or two postpartum in rats, but its length is not known in many other species, including marine mammals. If the mother is exposed to severe stressors however, GCs may be passed to the offspring through the placenta or in milk, circumventing this attenuating mechanism. The damage caused by exposure of the young brain to GCs produced by the mother alone can have profound and permanent consequences for the offspring, including sensitizing them to stressors, that is increasing their GC response, later in life (Kapoor, Dunn, Kostaki, Andrews & Matthews, 2006). Such changes can last at least to young adulthood and may be permanent, introducing the specter of potential generational effects.

Once this attenuation period ends, the still developing brain may then be very susceptible to neurological damage and re-programming as a result of exposure to high GC levels, whatever the source. Consequently, while reasonably mild stressors can lead to mild and temporary stress responses in adult animals, similar exposure in very young animals, either directly (e.g., brief handling of neonates, for not more than 2-3 minutes per day) or indirectly (e.g., through a “stressed” mother), has the potential to elicit long-term, if not permanent, consequences for the individuals resilience to stressors.

Long-term consequences of a prolonged or repeated stress response may also be present in individuals of any age due to ways that GCs instigate changes in

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<sup>1</sup> For more information on human activity in the GoM see the EPA Gulf of Mexico Program website (<http://www.epa.gov/gmpo/about/facts.html#maritime>), Lynch & Risotto (1985) and Melancon, Bongiovanni & Baud (2003).

the body. In order to have any effects GCs, like other steroids, must first pass through the cell wall. Once inside the cell nucleus, GCs bind with their receptor and they rewrite protein construction priorities (i.e., reprogram the expression of various genes). These revisions can persist long after high GCs levels have ceased circulating in the blood, thus long after the removal of the stressor. This persistence combined with the rapid activation of both the SNS and HPA axis responses means that many of the delayed and/or long-term consequences of stressor exposure are induced as a direct consequence of the initial perception of even a brief punctate stressor.

### ***Effects of combining stressor types***

The brain appears to classify threats as being processive (psychological) or systemic (physiological) in nature (see Deak, this issue). Psychological stressors include threats like predators, while physiological stressors include immediate and severe threats to physiological homeostasis, such as hypoglycemia (low blood sugar, specifically glucose). Importantly, some stressors appear to activate brain systems involved with both classes of stressors and it is these “compound” stressors that appear to produce the most direct outcomes for CNS functioning and overall health (see Deak, this issue).

Either exposure to a single very intense acute stressor, or the cumulative impact of numerous stressors across time, can ultimately lead to expression of sickness-like behavior, which is thought to be a symptom of neuroinflammation (Deak, this issue). For example, separation of a young guinea pig from its mother produces psychological stress (separation anxiety) and the offspring immediately begins to run around and vocalize. However, after an hour of exertion (physical stress), the young guinea pig stops that behavior, shuts its eyes, curls up and looks sick (Schiml-Webb, Deak, Greenlee, Maken & Hennessy, 2006). This response can be reversed by giving drugs with potent anti-inflammatory properties (Schiml-Webb, Deak, Greenlee, Maken & Hennessy, 2006). It is possible that the stress response and illness may have co-evolved as both are responses to threats (see Deak, this issue).

Normal aging is associated with greater expression of pro-inflammatory factors in the CNS (see Deak, this issue), so that risk of neuroinflammation increases with age. Repeated stressor exposure also leads to inflammatory responses as well as to accelerated aging as discussed above, creating an escalating combination of effects that can lead to increased incidence of neurodegenerative disorders and other critical problems that normally only arise later in life (see Deak, this issue).

### ***Maladaptation of the stress response***

Generally speaking, physiological responses to acute stressors promote survival in the face of diverse threats and are therefore viewed as being adaptive. Survival is promoted principally through a preferential re-allocation of resources (blood flow, glucose utilization, cognitive and sensory acuity, etc). The increase in

catecholamines associated with the acute fight-or-flight response has distinct energetic and immune consequences for the individual. The effects of GCs are more prolonged in nature and probably evolved as a mechanism to sustain behavioral and physiological responding to stressors of longer duration. The transient expression of sickness-like behavior after stressor cessation probably represents an adaptive period of recuperation that is necessary to reinstate normal levels of cognitive and behavioral function to pre-stress levels (Deak, this issue). With prolonged or repeated stressor exposure, however, neuroinflammatory consequences of stress can become maladaptive, leading to compromised neuronal function, greater susceptibility to infection (Dhabhar & McEwen, 1997), and ultimately reduced reproductive fitness (see Deak, this issue).

Likewise, failure to mount a GC response can lead to the inability of the animal to continue to respond appropriately to a stressor, subsequently resulting in death (see Romero & Butler, this issue). This failure might be due to overstimulation from either chronic or intense acute stressors that could have shutdown GC production through negative feedback, and possibly also depleted some of the various precursor molecules and biosynthetic enzymes necessary to produce the GC molecule. Alternatively, a prolonged response or exposure to a persistent stressor, such as pollutants, may have caused damage to the adrenocortical tissue where GCs are produced (Hontela, Rasmussen, Audet, & Chevalier, 1992; Martineau, this issue). Functional abnormalities of chronic stress are not restricted to GC effects. They can also result from catecholamines. For example, long-term activation of the fight-or-flight response across the life span can lead to coronary dysfunction and disease (see Romero & Butler, this issue), an effect that may involve vascular inflammation as an intermediate mechanism (Black, 2002, 2003).

In general, the physiological stress response and the consequences thereof described above are highly conserved between species, including fish, birds and mammals, although the exact basal levels of GCs and other stress hormones are fairly variable from one individual, population or species to another (see Deak, this issue; Martineau, this issue; Romero & Butler, this issue). However, not all stimuli are actually stressors. The distinction is largely a matter of perception by the animal/human. Experience immediately prior to a stimulus plays an important role in the nature and intensity of an animal's response to that stimulus. For example, a very slowly increasing stimulus is easily acclimated to and only becomes a stressor once it exceeds some threshold. Similarly, the stress response is initiated only when events are worse than those expected by an animal (Levine, Goldman & Coover, 1972). Conversely, if a stimulus decreases in frequency or magnitude, the individual perceives an improvement in situation and the stress response will decline, even if the individual is still being subjected to an unpleasant stimulus. Complicating the matter further, the expectation of an unpleasant stimulus may in itself initiate the stress response. Furthermore, acute stressors that normally last a short time (such as predator attacks, dominance interactions and storms) may become chronic stressors if they occur often enough or persist.



## Context and Behavioral Responses

Context is thus extremely important in the overall expression of a response to a potential stressor. Innumerable factors combine to form the context: environmental factors, such as season; recent history of incidence of the particular stimulus including intervals (i.e., prior experience); maturity, age, sex and other life history factors; inter- and intra-specific variation (genetic and propensity) including individual sensitivities, resilience and personality; condition (e.g., well-fed or hungry); other stressors currently acting upon an individual (e.g., infection, chemical exposure, etc.); predictability of stressor exposure; behavioral context (e.g., what the animal is doing when subjected to the stimuli); current psychological state (e.g., anxious, optimistic); and social structure.

### *Behavioral responses as an indicator for stress effects*

While many of the above contextual factors may influence the onset and/or magnitude of a physiological stress response, the response itself is reasonably consistent once activated. However, an observed response does not necessarily reflect the magnitude of the impact actually experienced by the animal (Beale, this issue; Beale & Monaghan, 2004; Bejder, Samuels, Whitehead & Gales, 2006; Gill, Norris & Sutherland, 2001; Harrington & Veitch, 1992; Lusseau 2004; NMFS, 1996; Stillman & Goss-Custard 2002; Todd, Stevick, Lien, Marques & Ketten, 1996).

For instance, behavioral reactions may be influenced by the psychological state of the individual. All behavioral decisions (whether conscious or not) are the product of information processing systems within the animal's brain. Stressors, including noise, and their associated emotional states, such as anxiety and depression, may influence this processing in a number of ways. First, anxiety is essentially an early warning system for the fight-or-flight response, and as such is associated with a suite of adaptive changes in cognition. Attention shifts towards awareness of possible threats and ambiguous information is interpreted more pessimistically (see Bateson this volume). These effects may be subtle and reversible, but may significantly affect the actions of an animal while they persist. For example, captive European starlings (*Sturnus vulgaris*) exposed to the stressor of being housed in barren cages may become more pessimistic and risk-averse in their interpretation of cues associated with food rewards. This pessimism is seen in a shift towards preferring safe foraging options, avoiding riskier but potentially more rewarding sites (Bateson & Matheson, 2007; Matheson, Asher & Bateson, 2008). Similar biases induced by other stressors could therefore result in changes in the spatial or temporal pattern of foraging behavior, with knock-on consequences for the fitness of the animals exposed. These changes in behavior also have the potential to place animals in situations where additional stressors could occur, such as food deprivation, or arrival in a novel environment due to avoidance efforts.

Physical condition can also influence behavioral responses. For example, well-fed animals may take fewer risks than their hungry counterparts, preferring a certain food reward over a more variable (i.e. risky) alternative (Caraco et al., 1990). Consequently, these individuals may also appear to be more sensitive to disruption, fleeing from a disturbance source at much greater distances. Conversely, a starving or sick animal may not display any observable response, as they may simply not be able to afford to react behaviorally: this is the only good feeding habitat in the area. Similarly, the well-fed animal may eventually be forced to return to its foraging ground when it becomes hungry, regardless of the potential threats. In this case, the change in behavior reflects a change in the physiological status of the animal.

However, such apparent increases in tolerance have often been used to argue that animals are “habituating” to the source and are thus no longer impacted by it (see below). On the contrary, any individuals (such as the hungry animal described above) remaining in a location in the face of potential danger may be subjected to one or more potential stressors. They may therefore display a number of physiological and epidemiological responses consistent with a stress response. For example, kittiwakes (*Rissa tridactyla*) in Scotland show an increase in heart rate in response to human disturbance. This cardiac reaction has been estimated to increase daily energy expenditure by around 7.5-10% for some individuals, despite a long history of exposure to disturbances in the area (see Beale, this issue). This increase in daily energy expenditure is sufficient to result in eventual abandonment of nesting attempts once energy reserves drop below a critical level.

In summary, a lack of behavioral response could be either because there is no stress felt, or because the animal can’t afford, or is not able, to respond overtly. Likewise, a strong behavioral response to a stressor, or a high level of observed response in a population, may mean that the stimulus is a particularly horrible stressor that is to be avoided at all costs, or it may imply that there is very little, if any, cost of responding to the stimulus, even though it may amount to no more than a minor irritation. Thus, given that animals make decisions (consciously or subconsciously) about how and/or whether or not to respond to a stimulus on the basis of their current context, this context must be known to biologists in order to accurately interpret the response intensity to a given stressor. As acquiring this knowledge is fraught with enormous difficulties in practice, it may not be possible at all to make such a determination simply from behavioral observations (see Beale, this issue). However, if such information is cautiously coupled with additional data (e.g., through the application of resource-use models), behavioral measures may allow the absolute minimum cost associated with responding to a stressor to be assessed (see Beale, this issue). Also, behavioral reactions observed in longitudinal studies can be, to some extent, placed in the context in which they occur (such as population abundance trends, residency patterns, season, etc.: e.g., Bejder et al., 2006; Lusseau 2005). These multi-scale approaches can also provide a framework to infer the synergistic costs of multiple stressors (natural and anthropogenic).

Likewise if the context in which decisions are made is not changed between two stressor exposures, behavioral measures can be used directly to

measure the relative degree to which the stressors affect individuals (see Beale, this issue). However, maintaining similarity of context is challenging. Many factors, such as passing predators, changes in prey abundance and distribution (even on a very local scale), and recent experience of any and all other stressors, can be difficult to measure especially in the marine environment. If such experimental approaches are not feasible, these contextual factors need then to be included as model co-factors or accounted for in other ways. One exception occurs if the behavioral responses to a given type of stimulus remain great regardless of the context, which would indicate unambiguously that the species involved attempts to avoid that type of noise at all costs.

### **Acclimation**

The term “habituation” is often used loosely to describe animals “getting used to” a stimulus, with various broad implications. However, “habituation” is often invoked without reference to the literature and seemingly in conflict with the use of the term in the biomedical or psychological literature (see Bejder et al., 2006). To avoid confusion, we shall use the term “acclimation” or “acclimatization”, meaning that an animal no longer produces a physiological stress response in reaction to a stimulus (Romero, 2004; Wright & Kuczaj, this issue and references therein). Animals can only truly acclimate in this way to stimuli that they perceive to be the same from one instance to the next, as well as non-life threatening (Romero, 2004; Wright & Kuczaj, this issue and references therein).

Acclimation is more likely to occur with frequently repeated, predictable exposures and can be lost if enough time passes between exposure events. This may explain why laboratory results for acclimation are more consistent than observations in the wild, as what appears to be repeated exposure in the “real world” may not be predictable or perceived as precisely the same by the animal. Chronic stimuli obviously meet the exposure frequency criteria required for an animal to acclimate, however animals may still lose acclimation if the exposure ends and there is enough time before the next exposure begins. The magnitude of exposure is also a consideration, because, in general, the greater the stress response initiated by a stressor, the less likely an animal is to acclimate to it, to the point where animals never acclimate to serious stressors.

In summary, animals will acclimate quicker to stimuli that are perceived to be smaller potential threats than those representing larger possible threats. However, acclimation only eliminates or reduces the stress response. It does not prevent other effects produced by a stimulus, such as hearing loss and masking that result from noise, as well as any stress response that these effects might subsequently induce. Similarly, acclimation also opens the possibility for sensitization, where the animal may produce an enhanced stress response when exposed to a new or different stressor.

Additionally, some uncertainties remain even within the narrower definition of “acclimation” as some humans can continue to perceive a noise as annoying or stressful without physiological responses or vice versa. Also, it’s not

clear exactly how similar a sound must be for animals to cease to be able to tell them apart: e.g., different boats may sound very different.

### **Determining Cumulative Effects**

We have already discussed above the potential for one stressor to influence the impacts of a subsequently applied stressor through the alteration of the context of exposure. Accurate prediction of all the potential cumulative and synergistic effects requires a reasonable knowledge of all the various contextual factors for each exposure and is thus not an easy proposition. However, at the most basic level it seems reasonable to conclude that the addition of new stressors is likely to increase the stress response, a concept that has some support in the literature (see review by Dallman & Bhatnagar, 2001).

The cumulative effects of multiple stressors can be estimated in this way through use of the concept of allostasis (see summary in Wright & Kuczaj, this issue; and discussion in Romero, 2004: Box 1 and references within), which suggests that all the various energetic demands that would be placed on an individual can be added up to see if that individual would be able to cope with them (i.e., maintain an allostatic load) or not (i.e., go into allostatic overload). Allostasis is currently a contentious idea in the biomedical world, a debate that goes beyond the scope of this paper. Suffice to say that the use of the concept of allostasis to investigate the cumulative effects of various stressors requires a working knowledge of the size of the energetic demands generated by each stimulus, which is clearly lacking for many species. This is not to say that energetic models cannot be useful in the management of the cumulative effects of various stressors on species where such data are limited, for example marine mammals (e.g., Lusseau, 2004). Rather energetic models are indicators of minimum possible energetic costs because of the various assumptions involved and the limited knowledge of the possible non-linear synergistic interactions between stressors.

Initial efforts to begin considering such non-linear synergistic interaction could be based on the two broad categories of stressors defined earlier, psychological, or processive, and physiological, or systemic, stressors. These categories should be considered because the simultaneous exposure to stressors belonging to each category increases the likelihood of having a severe impact on the individual. For instance, rats exposed to either simple restraint or hypoglycemic challenge show no evidence of neuroinflammation, while rats exposed to both challenges showed profound neuroinflammation (Deak, Bordner, McElderry, Bellamy, Barnum, & Blandino, 2005). Given that neuroinflammation may be a harbinger of adverse long-term health outcomes of stressor exposure, these data indicate that a categorically distinct, synergistic response can be provoked when otherwise innocuous events are combined. This may have profound implications for animals in captivity, which may be exposed to a wide variety of both physiological and psychological stressors such as confinement in a small environment, handling (especially in marine species, where handling is often

accompanied with at least partial removal from water), and the noise and activities of the public, staff, and/or researchers.

Even if both the different types of stressors and their cumulative energetic demands are accounted for, it may still not be possible to predict the overall effect of multiple stressors on an individual because lab-based studies have shown that multiple stressors interact in unpredictable ways to alter GC release, either increasing or decreasing circulating GC levels (see Dallman & Bhatnagar, 2001). Context or the influence of context may also vary unpredictably. Consequently, efforts to determine cumulative and synergistic effects of multiple stressors on animals, though important to pursue, should be undertaken cautiously.

### **Noise-Induced Stress Responses**

Some of the known effects of noise in animals include audiogenic seizures and increases in serum cholesterol levels (Clough, 1982), intestinal inflammation (Baldwin, Primeau, & Johnson, 2006), and increased adrenal weights due to overproduction of adrenal hormones caused by a prolonged stress response (Ulrich-Lai, et al., 2006). Stress responses induced by loud or sharp noises have even lead to cannibalism of neonates, as well as a generally decreased reproductive performance in mice (Michael Rand, pers. comm.).

The stress response with its various effects and impacts has been studied to some extent in rats and humans exposed to noise. For example, laboratory rats exposed daily to short periods of white noise exhibited a variety of conditions consistent with the onset of a physiological stress response after around 2 weeks, becoming more pronounced at 3 weeks (Baldwin, this issue). These conditions included inflammation of the intestinal mucosa and the mesenteric microvessels, degranulation of mast cells in the intestinal mucosa, migration of eosinophils into the wall of the intestine, and oxidative damage. Additionally, exposed rats groomed excessively and had redness around eyes and neck. After a recovery period of 3 weeks, the noise-exposed rats displayed some characteristics similar to unexposed controls, but other characteristics remained similar to pre-recovery conditions, indicating that some pathological effects continued to persist even after removing the noise exposure (Baldwin, this issue).

In humans, noise causes a number of predictable short-term physiological responses such as changes in hormone levels. However, little is known about how these might combine to have long-term consequences on health (see Clark & Stansfeld, this issue). Furthermore, specific evidence of chronic noise effects on adrenaline, noradrenaline and cortisol levels in humans is weak and inconclusive, suffering from various experimental difficulties (see Clark & Stansfeld, this issue). However, there is stronger evidence for a positive association between chronic noise exposure and both hypertension (i.e., raised blood pressure) and coronary heart disease (CHD), including some significant increases in myocardial infarction (i.e., heart attacks) associated with exposure to occupational, road traffic and aircraft noise.

There are indications that some of these effects on health may be mediated through annoyance, itself a psychological stressor (see Clark & Stansfeld, this

issue). In addition, noise exposure (or the annoyance it causes) has been associated with increased reporting of psychological and somatic symptoms in affected populations, but not with more serious clinically diagnosable psychiatric disorders such as anxiety and depressive disorders. This suggests that noise is probably not associated with serious psychological illness, but may affect well-being and quality of life (see Clark & Stansfeld, this issue). However, there have been no longitudinal studies in this area.

Noise may disturb sleep in humans as well, which may in turn have consequences for performance, mood and health. However, it appears that, with regards to sleep disturbance, naïve exposure (i.e., no prior experience) is a very important factor. Regardless of the mechanisms involved, these various effects may contribute to the increase in mortality observed in one study of industrial noise, with additional job-related stressors potentially acting cumulatively with the noise (see Clark & Stansfeld, this issue).

The greater expression of noise-related impacts in workers with higher job-related stressors is one example of the importance of contextual factors and cumulative exposure on the strength of response and ultimate outcomes from exposure to noise or any other stressor. Various other contextual factors are also important in humans in ways that are similar to the influence of prior experience on the physiological stress response of animals (see Clark & Stansfeld, this issue). For example, individuals with poor psychological health prior to exposure to noise reported greater annoyance (Tarnopolsky, Barker, Wiggins, & McLean, 1978), showing that individual psychological traits determine how annoying noise is.

Children may be more vulnerable to the effects of environmental stress as they have less cognitive capacity to understand and anticipate environmental stressors, in addition to lacking well-developed coping repertoires (Stansfeld, Haines, & Brown, 2000). Studies have consistently found that chronic noise negatively affects children's learning and cognitive abilities, and are beginning to indicate an effect on hyperactivity, although evidence for an increase in psychological symptoms is mixed and inconclusive (see Clark & Stansfeld, this issue). Recovery of some of these deficits may be possible if noise exposure ends, but noise could potentially impair child development, resulting in lifelong effects on both educational attainment and health. Longer exposures are known to cause larger and more persistent effects on physical health and are also likely to generate larger cognitive deficits and bigger effects on psychological health (see Clark and Stansfeld, this issue). Furthermore, the consequences for educational attainment are more likely to be long-lived or permanent if exposure overlaps with the closure of any learning window or opportunity (e.g., until a child leaves school).

### *Acclimation to noise*

Given the above considerations on acclimation to stressors in general, apparent behavioral tolerance of noise cannot be automatically interpreted as true physiological acclimation. Instead, apparent behavioral tolerance could be the result of different contexts, such as an overwhelming need for an individual or a population to remain in the area, the absence of alternative habitats, the prohibitive

costs associated with avoidance, or even that the animal might already have reduced hearing at the frequencies of the stimuli. Learning alone (i.e., without an associated reduction in physiological response) might also simulate acclimation to noise. In addition to the above mechanisms, an apparent increase in behavioral tolerance at the population level can arise if the most sensitive animals in the population have already left the area (e.g., Bejder, Samuels, Whitehead, & Gales, 2006). One other possibility is that rapid “natural” selection may have taken place, through the death of either the most sensitive individuals and/or the ones that are most prone to maladaptive alarm/escape responses (for some possible examples of these in marine mammals see Wright et al., this issue, b). The possible long-term costs and benefits of behavioral tolerance as a result of any of these mechanisms are unknown, although the action of either selection or emigration will clearly reduce the number of animals in the local population.

If an animal spends a considerable amount of time reacting to human disturbance, it may be fatigued and not willing or able to evade a potential threat and thus may appear to have acclimated when in fact it has not. Likewise, the apparently quick development of tolerance to disturbances in humans (e.g., aircraft noise in most people sleeping near airports) may not translate into free-ranging animals because animals must remain aware of predators, while humans in contrast are largely spared threats of this kind. Humans also benefit from prior knowledge that the noises can be reliably associated with passing aircraft or road traffic and that these things are unlikely to indicate an imminent threat.

The matter is complicated further still by the concept of “tuning out”, a type of filter for chronic, but changing, noise as is seen in humans (see Clark & Stansfeld, this issue). Consider that many patrons in a bustling restaurant largely filter out the general noise of employee activity and the conversations of other diners. This filtering does not prevent other effects, such as masking and hearing loss. Furthermore, it is not clear how much people or animals might perceive the noise as changing. For example, many of the abovementioned diners would look up if they hear a waiter breaking a plate or a glass.

### ***Masking, psychology and behavior***

Acoustic signals become ambiguous when they are hard to discriminate from other sounds. Increased environmental noise thus augments the ambiguity of incoming information by either reducing hearing capacity through hearing damage (temporary or permanent) or through masking by increasing background noise levels. Hearing damage persists after exposure (even if only temporarily) and affected animals can do little to compensate for the loss during that time. On the other hand, animals can employ several strategies to limit the ambiguity created by masking (see Bateson, this issue).

One option, physical avoidance, is to leave the noisy area for somewhere quieter. Avoidance strategies are not likely to be feasible for the majority of chronic or high-incidence noises. This is especially true for marine life exposed to ambient noise generated by shipping, which dominates background noise at low frequencies in many of the world’s oceans, particularly in the northern hemisphere.

A second option, available if noise is not continuous, is to cease communicating during periods when noise levels are highest. For example, urban European robins (*Erithacus rubecula*) switch to nocturnal singing in areas with high daytime noise (Fuller, Warren & Gaston, 2007). However, such evasive behaviors could again place animals in situations where they will encounter new stressors. In the above example, nocturnal singing could lead to an increased risk of predation by exposure to, or attraction of, nocturnal predators. In any case, temporal and spatial avoidance strategies can only be employed if the temporal distribution of the noise is predictable.

A third tactic available to animals is to change one or more characteristics of their acoustic signals, such as length, frequency, amplitude, or other acoustic features, to increase their transmission probability in a noisy environment. Beluga whales (*Delphinapterus leucas*) for instance can increase the amplitude of their signals in response to increasing background noise, a response known as the Lombard effect (Scheifele, Andrew, Cooper, Darre, Musiek, & Max, 2005). Humans speaking loudly in noisy situations are employing this option, but will eventually become hoarse and may temporarily lose their voice. It is not known what kind of consequences long-term use of signal-change strategies may have for animals, however increasing the amplitude of a sound uses more energy and therefore carries some additional cost.

The fundamental ability of an animal to actually alter its signals may also be limited, physiologically, anatomically, or by age. Many songbirds, such as the chaffinch (*Fringilla coelebs*), have a narrow window of time in early life in which their brains are particularly receptive to acquisition of new vocal patterns such as song. A few species, such as mockingbirds (*Mimidae*) and European starlings, continue to learn new vocal patterns after this period, while other singers show only limited variation from the parental song after early learning (for a review see Catchpole & Slater, 1995). Similarly, there are indications that bottlenose dolphins (*Tursiops truncatus*) may be able to learn at any time (e.g., Watwood, Owen, Tyack, & Wells, 2005) and male humpback whales (*Megaptera novaeangliae*) are known to change their songs repeatedly throughout their lives (see Noad, Cato, Bryden, Jenner & Jenner, 2000; Payne, Tyack, & Payne, 1983). However, very little is known about the abilities of most other marine species, especially marine mammals that use low frequencies, to acquire new vocal patterns throughout their lifetimes.

In any case, while altered signals may propagate further or be more distinct in the face of increases in ambient noise than unaltered ones, the potential usefulness of signal alteration is limited by the extent to which signals continue to be recognized by the intended receiver. This is especially important when the calls are involved in species recognition, perhaps for mating or maintaining social structure, which may further reduce the extent that these calls can be changed. Alteration of signals may also be problematic in species that communicate over long distances (such as mysticetes – baleen whales), because two animals may be subjected to very different ambient noise profiles. This means that the optimum signaling strategy in the immediate acoustic environment of the signaler may be



very different from the best option given the noise profile in the immediate area of the signal receiver.

Signal alterations are also not an option for animals that hunt using passive acoustics (i.e., eaves-dropping on their prey). Consequently, there will be many occasions when the only option available to an animal will be to alter its responses to incoming sounds. For example, animals can alter their thresholds for responding to incoming sounds that they receive, be they communication signals or sounds made incidentally by prey, predators, or con-specifics (see Bateson, this issue). If increases in masking noise make it harder to discriminate important signals from other irrelevant sounds, then animals may adapt to this situation in different ways, including:

- 1) Lowering their threshold for a sound to be identified as a particular type of signal, thus increasing their probability of falsely identifying signals as related to mates, prey and/or predators. Possible results include chasing after objects or organisms that are neither prey nor a mate, or fleeing from things that are not a predator (or other threat). This has consequences in terms of increased energetic costs.
- 2) Increasing their threshold for a sound to be identified as a particular type of signal, thus decreasing their probability of identifying a signal related to a mate, prey and/or a predator. Possible results include increased missed opportunity costs (e.g., passing up on possible prey and potential mates) or increasing the risk of predation if predators are missed.

In summary, animals have a range of options available for mitigating the adverse effects of environmental noise on their use of acoustic information. However, it is important to assess the potential fitness costs of any observed adaptation. Costs may arise from increased energetic expenditure, increased risk of predation, or lost opportunities for feeding or mating. All of these sources of cost could potentially be associated with increased risks of a physiological stress response occurring as animals struggle to adapt to function in a noisy environment.

### **Management Issues**

The stress effects from noise that are of the greatest interest to managers are those that ultimately have consequences for survival and fecundity rates (*vital rates*). Population level impacts are potentially catastrophic but highly uncertain, providing some grounds for a precautionary approach. However, as uncertainty is pervasive in ecology and conservation management, various tools have been developed that attempt to characterize and deal with such uncertainty in decision making processes (see Wintle, this issue). In particular, adaptive management and Bayesian modeling approaches offer some promise (see below and Wintle, this issue).

### ***Management under uncertainty: A general framework***

Adaptive management can be loosely defined as management with a plan for learning (Wintle, this issue). The sequential actions in the process of adaptive management should have the dual purpose of achieving management goals and facilitating learning about both the system under management and the relative performance of management actions. Effective adaptive management requires simultaneous implementation of multiple competing hypotheses and/or management actions that are iteratively updated through concurrent assessment and evaluation with monitoring data. Hypothesis generation and modeling may be based on existing data and/or expert opinion.

Adaptive management is appealing as it explicitly acknowledges that the decision being made is subject to uncertainty and may change in the next time step depending on what is discovered (i.e., learned) in the intervening period. Notably, the completion of an experiment is not required before a change to management can be instituted. This allows a more rapid response that is particularly well suited for managing systems in which changes take a long time to become apparent.

### ***Population modeling and scenario analysis***

Adaptive management of anthropogenic impacts on any species requires the construction of a model (or competing models) of species' responses to those impacts and any management intervention. Population models have been used in both terrestrial and marine systems to evaluate the long-term population consequences of competing management options (Akçakaya, Radeloff, Mladenoff & He, 2004; Taylor & Plater, 2001; Wade, 1998; Wintle, Bekessy, Pearce, Veneir, & Chisholm, 2005). Predictions of population models must be treated with caution as most population models require numerous assumptions and are themselves subject to substantial uncertainty. Despite the prevalence of uncertainty, modeling may be useful in challenging stakeholders and managers to clearly state their belief about species population dynamics and the magnitude and mechanisms of anthropogenic impacts. Models represent testable hypotheses that may be improved and updated as new data or knowledge comes to hand. As data are gathered, updated models may begin to provide predictions that are more broadly trusted by managers and stakeholders. In data-poor situations, it is important to make the most of available expertise or "collateral" data.

### ***Bayesian approaches to inference***

It is not easy or cheap to collect ecological data and definitive results are rare. Bayesian inference provides a coherent approach to synthesizing and making the most of disparate ecological data and/or expert opinion. McCarthy (2007 and summarized in Wintle, this issue) utilized a novel Bayesian approach to estimate the mortality rate of powerful owls (*Ninox strenua*) by combining very sparse observation data with predictions from a regression of body mass on mortality rate data for a range of other raptors. This approach provides a sound template for

analyses of other species that are characteristically difficult to study, including marine mammals. Expert opinion can (and should) be used in ecological studies, however it is very important that it is integrated in analyses appropriately (see Martin, Kuhnert, Mengersen, & Possingham, 2005 and McCarthy, 2007 on soliciting subjective priors for Bayesian estimates).

Once parameters have been estimated, population models may then be used to evaluate the long-term population consequences of competing management options (Akçakaya, Radeloff, Mladenoff, & He, 2004; Wintle, Bekessy, Pearce, Veneir, & Chisholm, 2005). However, any predictions arising from such a model would, at first, be compromised by substantial uncertainty in the parameter estimates. To address this, sensitivity analyses should be undertaken to identify the parameters and assumptions in the model that most strongly affect its predictions. These assumptions should then become the focus for adaptive management plans for learning.

### **Conclusions**

It is clear that the debate surrounding physiological stress responses, behavior, welfare and anthropogenic noise are going to continue for some time. To provide some focus we offer the following points as particularly noteworthy findings and recommend that scientists and managers take them into consideration when planning research and in assessments of environmental impact of noise.

1. Noise can act as a stressor. A single source of noise can result in a range of interwoven stressors. The various potential impacts of signal masking by noise illustrate this. The cascade of interwoven stressors that can be triggered by noise and masking includes separation anxiety, anxiety arising from ambiguous information, and hypoglycemia from loss of foraging opportunities, which can all in turn lead to other consequences as discussed earlier. Even when the noise itself may not lead directly to effects arising from the stress response, animals may create their own stressors through maladaptive efforts to avoid the noise. Similarly, physical injuries resulting from noise exposure may also act as additional stressors.
2. Short-term stress responses cannot be presumed to have only short-term consequences, especially when considering cumulative effects.
3. There is great potential for synergistic effects to arise through exposure of an animal to noise cumulatively with other stressors.
4. Context, especially the predictability of the stimulus and available response choices, is a very important (and possibly the most important) factor in mediating the overall stress response. For example, very young animals and fetuses are likely to be particularly susceptible to stressors, due to the effects of stress hormones on the developing brain. Thus, while

single or infrequent exposures alone may not produce long-term effects in adults, they may produce long-term consequences in young, still-developing animals. Unfortunately, such impacts will be very hard to detect in wild animals, especially in species that are hard to observe constantly, such as marine mammals.

5. It is impossible to determine the physiological and psychological responses of an animal to a stressor based on behavioral observations alone. Changes in an individual animal's behavior (or lack thereof) cannot be related to actual physiological and psychological impact without extensive investigation of the context. Behavioral changes in context are best understood and controlled in captive situations where exposure rates, environmental conditions and other factors are documented over long periods of time. However, the extrapolation of results from captive animals to the responses of wild animals should be done very cautiously given the large contextual differences (i.e., captivity and training vs. wild and free ranging) and the potential for high ambient noise levels to alter the baseline in the captive environment. Such contextual information is not generally available when assessing the possible correlations between acoustic stimuli and behavioral change in the wild. Collecting this information presents a considerable challenge, especially in the marine environment, although it is not impossible. Impact assessment studies need to specifically incorporate long-term and large-scale contextual information in their experimental design. Current short-term studies are generally failing to correctly assess the impacts of noise. Studies that have incorporated contextual information have led to a better understanding of disturbance impacts in other human-wildlife interactions. Without such contextual information it cannot be assumed that lack of a behavioral response means that no physiological stress response has occurred, or conversely that a behavioral response indicates the occurrence of a physiological stress response. In the latter case there may still be negative consequences for the animal if the behavioral response is maladaptive, involves a detrimental increase in energetic expenditure or exposes it to other threats.
6. By definition, acclimation requires consistency between non-severe repetitive exposures (including context) to sounds that are (near-) identical as perceived by the receiver. Conversely, repetitive exposure to different types of sounds (in frequency, intensity and other acoustic characteristics) cannot result in acclimation. Furthermore, animals cannot and will never acclimate to (contextually) severe stressors as these always, by definition, represent a threat. These reasons probably explain why few studies have shown acclimation occurring in the wild. Therefore, it should be assumed that animals have not acclimated to a sound, until proven otherwise. Although humans might be able to "tune out" more generalized noise sources such as road noise, health effects of exposure to such noise can

still arise (see Clark & Stansfeld, this issue). Tuning out can have its own detrimental consequences as individuals may over-generalize that ability to other sources, which may result in that individual ignoring sounds that are important to them, such as those produced by a predator.

7. While physiological acclimation to noise in the wild appears likely to be uncommon, it is clear that many animals have the capacity to learn to react behaviorally in a specific way to a generalized set of sounds. For instance, a whale might learn not to react behaviorally to noise from all types of engines because they have proven to be non-threatening to date. As the specific repeated experience required to induce physiological acclimation has not occurred, the whale may still initiate a stress-response to the sounds of a passing ship, priming the animal to react in the case that this particular noise is different. To date, however, the evidence that non-human animals have genuinely learned to reduce or eliminate behavioral responses to human disturbance is largely anecdotal. Regardless, generalized learning may also explain similar reductions in behavioral responsiveness to a given stressor at the population level. However, it is difficult to separate the action of such learning from a number of other possible mechanisms, including the mortality or displacement of the most susceptible individuals, gradual changes in the context in which a population find itself, and selection for adaptive responses occurring over several generations.
8. The considerable effects of relatively short periods of noise in the lab must be taken into consideration when interpreting the results of experiments undertaken with animals in captivity (see Baldwin, this issue). Most animals in captivity will have been exposed to relatively high levels of noise on a regular basis, due to feeding or other husbandry activities, machinery noise or other general facility operations. These effects, in addition to the increased sensitivity of developing brains to the effects of GCs, may partially explain why attempts to breed some animal species in captivity have not been successful.
9. Epidemiological studies in humans have been more consistent in demonstrating effects of noise on health and psychological wellbeing than on the physiological stress response. This might be explained if the epidemiological effects arise from cumulative effects over a long timeframe. Also, inconsistencies in the studies of the human physiological stress response to noise exposure may be due to (unknown) contextual elements that have not been accounted for.
10. Managing the impacts of noise on animal populations is likely to require an adaptive strategy to address the substantial uncertainties arising from a poorly understood stressor, especially in data-poor species such as many marine mammals. In situations of severe uncertainty, models can be useful

decision tools, not only because they make assumptions explicit, but also as they allow the stakeholders to explore the importance of those various assumptions. Adaptive management of noise impacts should be accompanied by well-planned long-term studies that address key uncertainties about the population level impacts of noise on the species concerned. Careful extrapolation of data from other species using appropriate analytical methods may provide a basis for developing actions to reduce noise impacts. Such actions would be refined as better, species-specific data come to hand.

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