REVIEW AND SYNTHESIS

How and why environmental noise impacts animals: an integrative, mechanistic review

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Abstract

The scope and magnitude of anthropogenic noise pollution are often much greater than those of natural noise and are predicted to have an array of deleterious effects on wildlife. Recent work on this topic has focused mainly on behavioural responses of animals exposed to noise. Here, by outlining the effects of acoustic stimuli on animal physiology, development, neural function and genetic effects, we advocate the use of a more mechanistic approach in anthropogenic environments. Specifically, we summarise evidence and hypotheses from research on laboratory, domestic and free-living animals exposed to biotic and abiotic stimuli, studied both observationally and experimentally. We hope that this molecular- and cellular-focused literature, which examines the effects of noise on the neuroendocrine system, reproduction and development, metabolism, cardiovascular health, cognition and sleep, audition, the immune system, and DNA integrity and gene expression, will help researchers better understand results of previous work, as well as identify new avenues of future research in anthropogenic environments. Furthermore, given the interconnectedness of these physiological, cellular and genetic processes, and their effects on behaviour and fitness, we suggest that much can be learned from a more integrative framework of how and why animals are affected by environmental noise.

Keywords

Anthropogenic noise, fitness, human disturbance, noise pollution, physiology, stress.

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INTRODUCTION

Although there are many natural sources of noise, including wind, water and other animals, one increasingly influential source is anthropogenic activity. This pervasive pollutant is expanding in scope and intensity commensurate with human population growth and urban development (Slabbekoorn & Ripmeester 2007). Anthropogenic noises are often louder, more frequent and more common than natural acoustic stimuli (Patricelli & Blickley 2006; Popper & Hastings 2009). Although the bulk of anthropogenic noise research has been conducted in terrestrial habitats, aquatic environments also suffer from noise pollution, which travels faster in the water and attenuates less per unit of distance from the stimulus source (Berg & Stork 2004).

In recent years, there have been several excellent reviews summarising major developments in the field of anthropogenic noise and suggesting future avenues of research (Rabin et al. 2003; Patricelli & Blickley 2006; Warren et al. 2006; Dooling & Popper 2007; Popper & Hastings 2009; Ríos-Chelén 2009; Barber et al. 2010; Slabbekoorn et al. 2010). These papers have focused mainly on the relatively small (although steadily increasing) body of work investigating how animal behaviour, and in some cases reproductive success, is impacted by anthropogenic noise.

However, we feel there is much to be learned from taking a step back and considering research focused on the effects of any environmental noise on wildlife, whether it is anthropogenic or 'natural'. This enables researchers to take advantage of over a half-century of extensive laboratory work conducted on well-characterised focal species such as rats (Rattus norvegicus) and mice (Mus musculus). Although the results of this work may not be broadly applicable to all

species and habitats, they provide a useful starting point for formulating detailed hypotheses about which life history characteristics might be influenced by noise and under what conditions.

Furthermore, a good portion of the general noise literature explores relationships that have not yet been investigated in an anthropogenic context, including the effects of noise stress on various aspects of physiology and development. Understanding how acoustic stimuli impact these fundamental biological processes is vital for elucidating the mechanisms linking environmental noise with animal behaviour (including distribution throughout the landscape) and both proximate and ultimate impacts on fitness traits.

It is our hope that this review will promote interdisciplinary collaboration, allowing us to understand the effects of noise from the level of the gene all the way up to landscape-level patterns and processes. To that end, we have considered a diverse array of literature on captive and wild animals from a variety of taxa. There is an emphasis on terrestrial animals – particularly mammals – because these are the species that have received the most attention. As this is a relatively broad overview, we have sometimes focused on representative results rather than performing an exhaustive review. We have organised our discussion into eight categories that correspond to systems that are impacted by exposure to environmental noise: the neuroendocrine system, reproduction and development, metabolism, cardiovascular health, cognition and sleep, audition and cochlear morphology, the immune system, and DNA integrity and genes. Although we have arranged the discussion into discrete sections associated with each of these categories, we wish to stress that these systems often interact (as we note in many examples). We begin with the neuroendocrine system, perhaps the most macrobiological

category, after which our discussion becomes increasingly microbiological, ultimately focusing on genes and DNA.

THE NEUROENDOCRINE SYSTEM

The hypothalamic–pituitary–adrenal (HPA) axis is an integral part of the endocrine system responsible for maintaining homeostasis. It consists of the hypothalamus, which contains neurosecretory neurons that synthesise hormones such as dopamine and corticotropin-releasing hormone (CRH); the pituitary gland, the anterior portion of which contains cells that produce adrenocorticotropin; and the adrenal gland, comprising the adrenal medulla, which secretes catecholamines (primarily epinephrine, but also norepinephrine, also called adrenalin and noradrenalin, respectively) and the adrenal cortex, which secretes steroid hormones (including cortisol, corticosterone and aldosterone) (Hall 2010). Increased production of these chemical products after exposure to an environmental stimulus is interpreted as a stress response.

Loud noise increases cortisol levels in several species, including lined seahorses (*Hippocampus erectus*) (plasma; Anderson *et al.* 2011), humans (urine; Evans *et al.* 2001), dogs (*Canis lupus familiaris*) (plasma; Gue *et al.* 1987) and goldfish (*Carassius auratus*) (plasma; Smith *et al.* 2004). Likewise, elevated corticosterone levels have been observed in chickens (*Gallus gallus domesticus*) (plasma; Chloupek *et al.* 2009) and mice (serum; Sobrian *et al.* 1997). However, a field study on California spotted owls (*Strix occidentalis occidentalis*) found no change in faecal corticosterone levels in response to presentation of chainsaw noise (Tempel & Gutiérrez 2003) and a laboratory investigation on mice actually reported that noise-stressed animals had lower levels of faecal corticosterone than their control counterparts (Jensen *et al.* 2010). In the latter instance, it was suggested that the main effect of noise was to disrupt normal hormone release patterns, such that the daily peak occurred later. Increases in noradrenaline levels have been reported for humans (Andrén *et al.* 1983), rats (Lenzi *et al.* 2003) and whales (Romano *et al.* 2004), the last of which also experience increases in epinephrine and dopamine. At least one dolphin experienced increases in aldosterone after exposure to water gun noise (Romano *et al.* 2004).

Beta-adrenergic stimulation increases glucagon secretion, which, in turn, raises blood glucose levels. In rainbow trout (*Oncorhynchus mykiss*), higher blood glucose levels were observed in individuals housed in tanks with 130-dB (re 1 μPa) noise than in those housed in tanks with 115-dB noise (Wysocki *et al.* 2007). Goldfish exposed to even higher noise levels (*c.* 160–170 dB re 1 μPa) did not experience significant changes in glucose levels, but their cortisol levels were dramatically higher than at pre-noise-exposure (Smith *et al.* 2004). Interestingly, despite the prolonged nature of the environmental noise disturbance, this response only occurred over the short-term, indicating habituation.

Sufficient exposure to noise can also cause physical damage to structures within the HPA axis, which may have both short- and long-term effects on maintenance of homeostasis. Pellegrini *et al.* (1997) exposed rats to 100 dB(A) of noise for 1, 6 and 12 h. Mitochondrial damage was observed in the zona fasciculata (ZF), which along with the zona glomerulosa (ZG) and the zona reticularis (ZR), composes the adrenal cortex. No significant changes were observed in the ZG, but mitochondrial membrane rupture, disturbance of the endoplasmic reticulum and cytoplasm dilution were all observed in the ZR. Although the damage did not appear to become

more severe over time, it did become more widespread. Oliveira *et al.* (2009), who also studied the effects of noise on rats, found time-dependent physical changes in the adrenal cortex, including a decrease in ZF volume and an increase ZR volume. The former trend appears to have been driven by reduced density of ZF lipid droplets, which are responsible for energy storage; the ZF likely utilised its energy stores as it responded to stress by increasing glucocorticoid production. Thus, physiological responses to stress not only change the hormonal environment within the affected individual, but may also deplete energy stores that might be better used for other purposes.

No studies to our knowledge have investigated the long-term impacts of noise stress on the HPA axis, although Oliveira *et al.* (2009) collected data from their rats through 7 months of exposure and some long-term correlative work has been carried out in humans exposed to noise in the workplace (Ising & Kruppa 2004). Research on the effects of other stress response-inducing factors is illuminating. Field work on three species of bird [barn swallows (*Hirundo rustica*), song sparrows (*Melospiza melodia*) and white storks (*Ciconia ciconia*)], suggests that corticosterone levels are negatively associated with immune responses (Saino *et al.* 2003), survival and recruitment (Blas *et al.* 2007; MacDougall-Shackleton *et al.* 2009) and even song syllable diversity (MacDougall-Shackleton *et al.* 2009). Thus, while stress responses may be immediately beneficial, for instance by priming an animal to run away from an oncoming car, they may be detrimental over the long term. Future studies should focus not only on investigating this possibility, but also on determining the relative impacts of infrequent, intermittent and chronic responses.

It is important to note that animals may habituate to stressors over time. Both Magellanic penguins (*Spheniscus magellanicus*) (Fowler 1999) and Galápagos marine iguanas (*Amblyrhynchus cristatus*) (Romero & Wikelski 2002) exhibit lower corticosterone levels in areas more frequently visited by tourists, although interestingly the penguins only had this response at highly trafficked, and not moderately trafficked, sites. Among European blackbird (*Turdus merula*) nestlings that were hand-reared by researchers, corticosterone stress responses were lower in chicks originating from urban sites than in those from the forest. It is unclear whether this was a result of maternal factors, genetic differences or both. It remains to be seen whether similar habituation and adaptation patterns occur in response to noise stressors. Our prediction is that many neuroendocrine responses to noise are highly plastic; thus, ecological control of noise pollution could allow animals to achieve both structural and functional recovery.

REPRODUCTION AND DEVELOPMENT

The impacts of environmental noise can be felt as early as the embryonic stage, by direct (though presumably muted) sound wave activity on the foetus, as well as via physiological impacts on pregnant females. In humans, for example, excessive environmental noise (> 85 dB re 20 μPa) has been correlated with premature birth (American Academy of Pediatrics Committee on Environmental Health 1997). Pregnant female rats exposed to elevated levels of environmental noise gave birth to pups with greater fluctuating asymmetry (a morphological indicator of developmental instability; Møller & Swaddle 1998) in their parietal and long bones, as well as decreased dental calcium concentrations (Gest *et al.* 1986; Siegel & Mooney 1987). Although the exact mechanism behind this response is

not fully understood, these growth abnormalities appear to result from system-wide disruptions of calcium regulation (Siegel & Mooney 1987). Increased mortality was observed in fish eggs and embryos located in environments with ambient noise levels that were only 15 dB re 1 µPa higher than those observed in nature (Banner & Hyatt 1973); among surviving fry, excessive noise was related to slower growth rates, a result also observed in shrimp (Lagardère 1982). Interestingly, and contrary to the aforementioned trends, exposure to environmental noise in the form of 140 dB re 1 µPa classical music enhanced the growth, quality and production of aquacultured gilthead seabream, *Sparus aurata* (Papoutsoglou et al. 2008).

Noise stress appears to be particularly damaging to females, a relationship that likely stems from sex differences in size, hormone expression and the costs of reproductive investment. Reproductive rates were substantially lower among brown shrimp (*Crangon crangon*) that had been exposed to noise (50% vs. 80%) and fewer egg-bearing females were found in the noise treatment (70% vs. 92%), indicating that noise-stressed individuals may not have had the resources necessary for reproduction (Lagardère 1982). A long-term study evaluating daily behavioural and hormonal responses of a captive female giant panda (*Ailuropoda melanoleuca*) found that reproductive state strongly influenced stress level: While the panda demonstrated increases in agitation behaviours and urine cortisol levels on days with louder average amplitude of ambient noise, these results were particularly pronounced during oestrus and lactation (Owen *et al.* 2004). Unfortunately, because only one individual was studied, it is unclear whether these responses can be generalised. At least one **3** study suggests that males may also suffer from sex-specific responses to noise stress: Lower plasma testosterone levels were observed in male mice exposed to 100 dB(A) white noise for 6 h day^{-1} over 6 weeks (Ruffoli *et al.* 2006). This type of hormonal deficiency could be particularly problematic prior to, and during, the breeding season, when testosterone levels influence territoriality and mate-wooing behaviours (e.g. Van Duyse *et al.* 2003) vital to reproductive success.

Indian meal moth (*Plodia interpunctella*) larvae exposed to noise exhibited a 75% reduction in emergence, indicating that acute noise stress can be fatal; however, it is unclear whether this resulted from the experimental use of particular noise levels or extreme sensitivity of larvae during a particular developmental stage (Kirkpatrick & Harein 1965). Muscovy duck (*Cairina moschata*) embryos exhibit behavioural responses to ambient noise stimuli when they are still in the egg (*c.* 75% of the way through the incubation process; Hochel *et al.* 2002). In fact, inter-egg communication between developing embryos is known to facilitate hatching synchrony in several bird species (Woolf *et al.* 1976). Thus, increases in anthropogenic noise might promote size disparities within broods, potentially leading to nutritional deficits and developmental problems in smaller chicks that cannot outcompete their siblings for resources; in extreme cases, this could even lead to starvation and death. Sobrian *et al.* (1997) found that noise-stressed dams gave birth to an increased number of female pups, suggesting that noise disturbance could alter population sex ratios.

Reduced 'reproductive output', ranging from number of juveniles produced to amount of milk production, has been reported in a variety of domestic species in response to sonic booms (reviewed in Bowles *et al.* 1990). However, this literature is conflicting because it often focuses on reports from economically invested farmers or does not adequately control for other relevant factors. Recently, work on urban noise has shown that increased noise levels are associated with smaller clutches and fewer fledglings in great tits (*Parus major*) in Europe (Halfwerk *et al.* 2011) and fewer fledglings among eastern bluebirds (*Sialia sialis*) in North America (Kight 2010). However, the mechanisms behind these effects have yet to be elucidated.

We suspect that noise levels might also negatively correlate with survival of individuals, although decreases in life span are likely to be mediated by different processes at each life stage. However, we could find no work that followed noise-stressed individuals throughout their entire lives. Such data are necessary for calculating recruitment rates, which could, in turn, shed light on the impacts of noise at the population level. Longitudinal data will also be necessary to understand the impact of early life exposure to noise on later life fitness traits, as we predict that if noise affects key developmental processes, the consequences will persist over the long term.

METABOLISM

Animals that respond to noise stressors by increasing vigilance, hiding and/or retreating may correspondingly decrease the amount of time they spend foraging. This could have decrease weight gain, as observed in rats exposed to noise stress for 30 days (Alario *et al.* 1987). Likewise, brown shrimp housed in a noisy aquarium consumed less food, particularly in the first 2 months of noise exposure (Lagardère 1982). At the end of the experiment, noise-stressed individuals had experienced less weight gain and were smaller than their control counterparts. While this was true for both sexes, the result was particularly obvious among females. Another study on shrimp found that noise-exposed individuals excreted higher levels of ammonia and consumed higher levels of oxygen (Regnault & Lagardère 1983). Ammonia is generated via oxidation of the amino group that is removed when proteins are converted to carbohydrates to provide energy; thus, these two results indicate that noise-stressed shrimp were utilising higher levels of energy. The animals had an immediate response to noise stress, which showed no signs of diminishing over time.

Anderson *et al.* (2011) reported decreases in both weight and overall condition of noise-stressed seahorses; unfortunately, although the authors documented an increasing number of distress behaviours among animal exposed to noise, it is unclear whether there was a corresponding decrease in foraging activity. Although it did not appear to impact their weights, noise stress was correlated with poorer foraging performance (as measured by number of food-handling errors and ability to discriminate) among three-spined sticklebacks (*Gasterosteus aculeatus*) (Purser & Radford 2011).

Dogs that were exposed to 80- to 90-dB rock music within an hour of their last gastric migrating motor complex (GMMC) had a longer latency to the next GMMC than control dogs, although the long-term gastric emptying cycle was not impacted by the noise stress (Gue *et al.* 1987). The GMMC is responsible for emptying the stomach of indigestible contents such as bone and fibre; disruptions in this process could lead to transient periods during which the stomach is unnecessarily full, perhaps preventing an animal from receiving hunger cues or decreasing its ability to reach full speed during a chase or getaway.

In their extensive work on noise-stressed rats, Baldwin *et al.* (Baldwin *et al.* 2006; Baldwin & Bell 2007) found that acoustic stress leads to cellular leakage in the mesentery, which suspends the small intestine from the abdominal wall. The increased leakage was due to both a larger number of leakage sites and a larger leakage area per

venule. Because the contents of the smaller intestine are potentially harmful to the body, increased leakage could result in infection. Thus, it is not surprising that the authors also documented greater inflammation of the small intestine and a higher number of degranulated mast cells, indicating local immunological activity against microbial pathogens.

Higher plasma cholesterol and protein levels have been observed in domestic hens (*G. gallus domesticus*) exposed to noise stress (Chloupek *et al.* 2009); this may have resulted from an increased need for these products to fuel the production of hormones used in the stress response. If stressed individuals are rapidly using their stored resources to regulate neuroendocrine and immunological responses to stress, maintenance of normal food intake levels will be especially vital; thus, decreases in feeding activity or metabolic processing under these conditions may be particularly harmful. Given that anthropogenic noise is often accompanied by other environmental characteristics that can decrease food availability, this may be a particularly important area for future research. However, metabolic deficiencies have not been reported uniformly. A study on captive-reared fish, for example, found that although there was obvious treatment-dependent variation early on, there were ultimately no significant differences in mean weight, length, growth, condition, feed conversion or survival of noise-stressed and control individuals (Davidson *et al.* 2009). Thus, again, at least some species appear capable of habituating to noise stressors.

CARDIOVASCULAR HEALTH

During stress reactions, the heart contracts both more rapidly and more forcefully and vasoconstriction occurs throughout much of the body so that blood can be reserved to deliver the oxygen needed to fuel quick movements (e.g. to escape a predator), a process that is aided by vasodilation in the skeletal muscles (Herd 1991). These changes are overwhelmingly meant to aid in behaviours that can be measured on the timescale of seconds, sometimes minutes, or, rarely, hours; frequent or long-term expression of these characteristics may have adverse effects.

As with research on the HPA axis, the bulk of cardiovascular studies have focused on humans, rats and mice. Among humans, exposure to loud (both temporary and long-term), is associated with increases in systolic, diastolic and main arterial pressures, leading to an increase in total peripheral resistance to blood flow (Andrén *et al.* 1983). Chronic exposure to urban noise at home has been associated with elevated resting systolic blood pressure among children, as well as more intense heart rate reactivity in response to the presentation of a novel stressor (Evans *et al.* 2001). However, heart rate data have demonstrated habituation to short-term noise stimuli in both ungulate (Weisenberger *et al.* 1996) and bird (Harms *et al.* 1997) species.

Detailed morphological research in rats has uncovered the variety of physical damage that can accrue in the heart during reactions to noise stress. One common finding has been mitochondrial damage in myocardial cells, as well as swollen sarcoplasmic reticulum and dilation of the intercalated discs (Gesi *et al.* 2002b), all of which may result from increased calcium entry driven by catecholamine-induced increases in cytosolic calcium concentration. Damage has been observed in both the atria and the ventricles (Soldani *et al.* 1997; Lenzi *et al.* 2003), although the former may be more impacted by noise stress than the latter, especially at the mitochondrial level; structural damage in the ventricles is only observed after more prolonged noise exposure (Soldani *et al.* 1997). Paparelli *et al.* (1992) found that the density of noradrenergic fibres was significantly higher in both the aortas and atria of young rats exposed to 12 h of 100 dB(A) noise stress, leading to increased responses to a β-adrenergic agonist; in other words, both the morphology and the function of cardiac tissue had changed in response to an acoustic stimulus. Significant decreases in peripheral benzodiazepine receptors (PBR; Salvetti *et al.* 2000), which are involved in responses to uncontrollable stress (Drugan & Holmes 1991), have also been observed in rats. PBRs are found primarily on the mitochondria and appear to play a role in the mitochondrial permeability transition (MPT)-pore, which may facilitate cellular pathology resulting from trauma. Interestingly, MPT-pores can be induced by both calcium and free radicals, both of which can be increased by exposure to noise stress. At least one study has found more cardiac damage in males than in females (atrial; Gesi *et al.* 2002a), again emphasising the importance of sex in mediating responses to noise stressors.

A comparative study on rats and mice (*M. musculus*) found that mice were less sensitive to noise stresses than rats, possibly because mice have zonal cardiac noradrenaline receptor patterning, rather than the diffuse pattern observed in rats (Gesi *et al.* 2002b). Expression patterns of hormone receptors, at both the cellular and tissue levels, may therefore be a useful clue in determining whether, and to what extent, animals will respond to environmental noise disturbances.

Like rats and humans, largemouth bass (*Micropterus salmoides*) responded to noise stress with increases in cardiac output and heart rate, as well as decreases in stroke volume (Graham & Cooke 2008). The magnitude of their responses positively corresponded to the intensity of the disturbance (e.g. noise from canoe paddling, a trolling motor and a combustion engine), while the amount of time required to return to baseline levels was negatively corresponded to intensity. Unfortunately, the fish in this study were not exposed to recordings of noise stimuli, but to the actual objects themselves (e.g. a paddle and two motors); thus, their responses may have been influenced by variations in appearance, as well as noise. Additional work appears necessary for verifying that fish exhibit a cardiovascular stress response to noise alone. Across all species, it is unclear whether, and what type of, cardiac damage might result from chronic exposure to environmental noise stressors.

COGNITION AND SLEEP

Chronic noise exposure in industrial workers and individuals living near major transportation routes has been associated with depression and feelings of aggression (Stansfeld & Matheson 2003; Ising & Kruppa 2004). Noise may also be fear-inducing, as evidenced by a more prominent tonic immobility response in noise-stressed hens (Campo *et al.* 2005; Chloupek *et al.* 2009). Children exposed to higher ambient noise levels in their homes self-reported higher stress levels than those from quieter environments (Evans *et al.* 2001); noise-stressed girls appeared particularly likely to suffer from feelings of reduced motivation, highlighting yet again sex differences in response to the acoustic environment. We therefore predict that personality traits may be affected by noise pollution, which could alter behavioural interactions and population dynamics.

Serotonin expression is one mechanism that may be responsible for psychological responses to noise. For example, serotonin expression

was increased in rats that had been prenatally exposed to 95-dB supersonic sound, but was decreased in individuals prenatally exposed to 65-dB music (Kim *et al.* 2004). Unfortunately, it is difficult to directly compare these two treatments, as they varied in both type and intensity of noise. However, this example raises the interesting issue of music; why it should be beneficial, while other environmental noises are generally disruptive, is an intriguing question.

Increased noise levels have been associated with decreases in intentional, incidental and recognition memory in children (Lercher *et al.* 2002), a result that has also been paralleled in rats (Rabat 2007). In addition, noise-stressed children have been shown to have deficits in speech perception and reading ability (Hygge *et al.* 2002); although scores of the latter improved once the noise had ceased, scores of the former did not. These results are somewhat similar to those of several avian song-learning studies investigating the effects of hearing impairment on memory and vocal ability (e.g., Marler *et al.* 1973). In one notable study, zebra finch (*Taeniopygia guttata*) juveniles were exposed to shorter- or longer-term treatments of extreme (> 110 dB re 20 μPa) environmental noise (Funabiki & Konishi 2003). Once released from the noise exposure, individuals of both groups were able to recover some of their vocal skills, but not all; in no case were noise-stressed individuals able to reproduce 'normal,' species-appropriate vocalisations.

Unfortunately, in studies such as these it can be difficult to assess the relative impacts of noise as a physiological stressor, a distraction (Chan *et al.* 2010; Purser & Radford 2011), and/or a deafening agent. One study of cognitive deficits in rats has attempted to disentangle these effects (Cui *et al.* 2009). Briefly, rats were trained to use visual cues to locate a submerged platform in one quadrant of a circular pool. Individuals that experienced loud noise conditions during the learning phase of the experiment took longer to find the platform and spent less time in the target quadrant. A variety of corresponding neural assays indicated that these delayed responses resulted from learning deficits related to shifts in neuron structure, neurotransmitter balance and neuronal receptor subunit expression. Likewise, it has also found that offspring of noise-stressed female rats performed worse on spatial tests and had higher error rates (Kim *et al.* 2006); it is currently unclear whether these responses resulted from direct effects of noise on the foetus or were mediated by maternal stress responses during pregnancy. Surprisingly, rats presented with 70-dB white noise during maze-learning trials not only made fewer errors and required less time to complete the maze, but also performed less rearing (a sign of stress; Prior 2006). Thus, it appears that more work is needed to determine the conditions under which noise might act as a beneficial stimulant of brain activity and to document whether this positive effect is widespread across the animal kingdom.

A significant amount of research, particularly in humans, has documented the impacts of noise on sleep. 'Natural' environmental noise has more deleterious effects than manufactured white noise, as it is both temporally and spectrally more variable (as reviewed in Rabat 2007). Sleep perturbations may occur in response to even relatively low amplitudes of environmental noise, leading to variations in slow-wave ('deep') and/or paradoxical ('rapid-eye-movement,' or 'REM') sleep; species differ in their reports of whether, and how much, either category of sleep is impacted by noise. However, both sleep types can become permanently altered by repeated noise-induced sleep disruptions; in turn, chronic sleep problems can lead to other physiological pathologies, as well as cognitive deficits (Spreng 2000; Rabat 2007). Sleep patterns are also likely to be influenced by excess light, a common accompaniment to noise pollution in anthropogenic environments.

AUDITION AND COCHLEAR MORPHOLOGY

Hearing impairment and deafness are two of the most obvious effects of extreme environmental noise on sensory systems. Because these responses have been extensively reviewed in the literature for a variety of species (e.g. Dooling & Popper 2007), we will only present a summary here. Briefly, these maladies result from damage of the cochlea and/or its related neural structures (McCauley 2003; Hu & Zheng 2008). In fish, vibrations from extreme noise may also impact the swim bladder, leading to tears and ruptures; this can be particularly devastating because the swim bladder is used not only in the reception and resonance of sounds, but also in buoyancy control (Popper & Hastings 2009). In both terrestrial and aquatic animals, auditory injuries may stem from single, extreme acoustic traumas (e.g. noises occurring beyond the pain threshold), or from chronic exposure to dangerous levels of noise. While the former category has received much attention – particularly in aquatic habitats (as reviewed in Popper & Hastings 2009) – the latter category has generally been overlooked. Many habitats, such as those found along roads, receive prolonged exposure to lower-amplitude noises (Parris & Schneider 2008); thus, chronic, low-level noise disturbance is likely to impact a variety of organisms.

Exposure to noise stress usually increases an animal's hearing threshold (Chang & Merzenich 2003; Shi & Nuttall 2003; Smith *et al.* 2004; Song *et al.* 2008; Codarin *et al.* 2009); in some cases, this may be temporary (a temporary threshold shift, or TTS), while in other cases, it may be permanent (a permanent threshold shift, or PTS). In many instances, the strength of the TTS corresponds with the duration of exposure to the noise stressor (Smith *et al.* 2004). The length of time required to recover from a TTS varies according to the temporal and spectral characteristics of the noise stressor, as well as the auditory sensitivity of the affected animal (Clark 1991).

In addition to having direct, physical impacts on hearing apparati, noise stress can also affect auditory processing in the brain. Studies of sound perception and auditory cortex development in rats have shown that exposure to noise leads to poorer acoustic processing and delayed neural maturation (Chang & Merzenich 2003; Sun *et al.* 2011). Once individuals are no longer exposed to noise stimuli, auditory development appears to proceed in a normal fashion, eventually allowing attainment of age-appropriate neural function. Retarded neural development as a result of noise exposure appears to extend the plastic phase of auditory tuning; thus, in species that disperse, individuals that relocate from noisy to quiet environments may be less adversely impacted by noise than those that stay. This provides yet another example of plastic and even reversible, responses to noise.

IMMUNE SYSTEM

As mentioned above, activity of noise stresses on the HPA axis can lead to downstream effects on the immune system. This is even true across generations. Sobrian *et al.* (1997) repeatedly exposed pregnant female mice to an 85- to 95-dB alarm bell and then measured the immune function of their pups in comparison to that of control pups. Juveniles from mothers in the noise treatment had smaller thymus weights shortly after birth, as well as lower serum IgG levels,

indicating impairment of the secondary immune response. Prenatally stressed juveniles did not respond to immunological stressors as strongly as control juveniles did; furthermore, these effects appeared to be mediated by sex, with females generally more impaired than males.

Rats exposed to short periods of loud noise (85 dB re 20 µPa) for 3 weeks displayed significant decreases in their humoral immune responses (including increases in immunoglobulin levels, decreased numbers of T cells and decreases in phagocytic activity) within the first week of the study, but reached an asymptotic response state within 3 weeks of noise exposure (Van Raaij et al. 1996). In some individuals, immune responses even improved between the first and third weeks of the study.

Release of corticosterone affects the heterophil-to-lymphocyte ratio (H : L); generally, H : L increases in response to stress, either because H increases while L decreases or simply because H increases (Gross & Siegel 1983). For example, H : L was higher in noise-stressed hens than in controls because the stressed birds had higher H levels (Campo et al. 2005). Likewise, seahorses exposed to loud aquarium noise had higher H : L as a result of larger H values; noise-stressed individuals were also more likely to be infected with metazoan liver parasites (Anderson et al. 2011).

Rats exposed to 130-dB infrasonic noise experienced increased activation of microglial cells, macrophages in the brain and spinal cord that defend the central nervous system against immunological challenges (Du et al. 2010). The activated cells upregulated their expression of corticotrophin-releasing hormone receptors (CRH-R1), highlighting the interconnectedness of neuroendocrine and immunological responses to noise stresses (Du et al. 2010). Among rats intermittently exposed to 85-dB white noise for 3 weeks, the effects of acoustic stress varied with length of exposure (Van Raaij et al. 1996). For instance, activity of splenic natural killer cells was higher after both 24 h and 7 days of exposure, but had been suppressed after 3 weeks of exposure. Because similar patterns were not observed for all immune variables measured in the study, the authors suggest that different components of the immune system may be differentially impacted by chronic exposure to noise.

Recently, Moreno-Rueda (2010) found that there was an immunological cost of increased begging among house sparrow (*Passer domesticus*) nestlings. Although this work was not conducted in an anthropogenic noise context, it provides a tantalising glimpse of the possible repercussions of noise-induced communication breakdown between adults and their young (e.g. Leonard & Horn 2008).

DNA INTEGRITY AND GENES

Acoustic stressors can impact genes in two principal ways: by setting off chemical cascades that can lead to DNA damage and/or by altering gene expression.

The neural activity required to process environmental noise leads to an increased number of free radicals, which are known to cause carcinogenic mutations (Samson et al. 2005). Levels of cochlear reactive oxygen species (ROS) may also rise in noise-stressed animals. Like free radicals, ROSs cause damage to DNA, as well as to proteins and lipids. Cochlear ROS levels were quadrupled in mice that had been exposed to PTS-generating noise and these values did not decrease over time (Ohlemiller et al. 1999). ROS-induced damage has been observed in the adrenal glands (Frenzilli et al. 2004) and hearts (Lenzi et al. 2003) of noise-stressed rats. The cardiac damage was

persistent for at least 24 h after noise stress, an unusual pattern given that DNA breaks are usually fixed within hours of their occurrence. Shi et al. (2003) recently proposed a potential mechanism linking noise to ROS, at least in the mouse cochlear stria vascularis: In response to noise, increased activity of inducible nitric oxide (NO) synthase (iNOS) generates NO, which causes oxidative stress, generating an excess of ROS, leading to DNA damage. It is unclear whether similar patterns exist in free-living animals exposed to subacute and/or chronic levels of noise. In addition to exploring this possibility, future work should attempt to measure the length of time over which DNA damage persists and whether it has phenotypic consequences. Exploring direct damage to germ line cells may be particularly fruitful, as even temporary changes to these cells could alter their fates.

Environmental noise is known to impact expression of several genes, predominantly in the brain. Noise-exposed rats that performed poorly on spatial tasks were found to have decreased expression of N-methyl-D-aspartic (NMDA) receptors, which are sometimes called the 'smart' receptors because of their role in synaptic plasticity and memory (Cui et al. 2009). Increased expression of the NR2B protein, an NMDA receptor subunit, was responsible for the extended period of plasticity that enabled noise-exposed rats to develop age-appropriate auditory functioning after removal from a noisy environment (Sun et al. 2011).

Acoustic stress impacts expression of benzodiazepine receptors, allosteric modulatory sites on $GABA_A$ receptors, which bind gamma-aminobutyric acid (GABA), the primary inhibitory neurotransmitter in the central nervous system. Specifically, higher levels of benzodiazepine receptors were observed in the cerebral cortex in noise-stressed rats (Lai & Carino 1990), but not in the hippocampus or cerebellum, indicating that noise affected gene expression in a region-selective manner. Noise-stressed rats also were found to have increased levels of diazepam binding inhibitor (Ferrarese et al. 1991), which is involved in the displacement of benzodiazepines, in both the hippocampus and adrenal gland. Juvenile rats exposed to prenatal noise disturbance had higher levels of tryptophan hydroxylase (TPH) expression than controls (Kim et al. 2004); since TPH is the rate-limiting step in serotonin synthesis, these same rats had correspondingly higher levels of serotonin. As previously mentioned, experimental noise has been shown to cause upregulation of CRH and its receptor neurons in the paraventricular nucleus, a neuronal nucleus in the hypothalamus. This, in turn, is thought to induce expression and release of tumour necrosis factor-alpha, which at low levels, can strengthen the stress response, but at high levels, can lead to neuroinflammation and apoptosis (Du et al. 2010).

Research into the effects of environmental noise at the genetic level has only just begun. Previous efforts have focused primarily on stress response-related genes in the brain and CNS. However, given the widespread downstream impacts of stress, gene expression is also likely to be affected in other systems and structures. Changes in the expression of genes, both singly and in suites, are likely to impact an animal's physiology and behaviour, as documented in an increasing number of anthropogenic areas (e.g. Romero & Wikelski 2002; Jiménez et al. 2011). Thus, an overdue focus on genetic responses to environmental noise is likely to greatly expand our understanding of how noise pollution influences biological systems. In particular, we feel that a priority would be to understand influences of noise on the germ line cells, and in key systems such as the HPA axis and cerebral cortex.

CONCLUSIONS

We have shown that environmental noise can lead to DNA damage, alterations in gene expression and changes to a myriad of cellular processes related to appropriate neural, developmental, immunological and physiological functioning. In addition, previous authors have discussed ways in which noise can impact animal behaviour and community ecology (Francis *et al.* 2009; Barber *et al.* 2010). We wish to stress that each of the systems and functions that can be influenced by noise – DNA integrity and genes, cell structure and signalling, physiological systems, behavioural ecology and community ecology – can also influence each other (Fig. 1). In other words, anthropogenic noise is likely to have both diverse and complex impacts on wildlife, as it can influence multiple biological systems both directly and indirectly. Thus, we encourage ecologists who wish to understand how and why animals respond to noise in particular ways to develop integrated study designs allowing them to investigate not just macrobiological processes such as behaviour or species diversity (e.g. the right side of the continuum in Fig. 1), but also the cellular and genetic mechanisms that can drive them (e.g. the left side of the continuum). Integrated studies that span this continuum are integral to developing predictions about how noise will affect wildlife and are vital for making informed mitigation and management decisions.

As emphasised in Fig. 1, many gene and cell responses may not be reversible, especially if noise perturbations occur at key developmental stages and/or in the germ line. However, physiological and behavioural responses are notoriously plastic (e.g. Beckers & Schul 2008) and may have relatively fewer long-term consequences for individuals and populations. Therefore, we believe that researchers interested in the detrimental effects of noise pollution on wildlife should attempt to link genetic and cellular responses to physiological and behavioural ecological mechanisms. This integrative framework will not only help us understand how animals are affected by noise, but may also give us insights into how we can reduce the harmful effects of anthropogenic noise on populations. If we can understand the mechanisms behind various responses, we will have greater opportunities for minimising future damage.

Some of the results discussed in this review suggest that, while some types and levels of noise may be harmful, others may enhance (Papoutsoglou *et al.* 2008), or even play an integral role in (Sun *et al.* 2011), development. Thus, researchers may need to develop nonlinear predictions of how biological systems respond to noise. This is an important consideration in experimental design, as well as quantitative analyses and modelling.

Despite our enthusiasm for the framework proposed here, we feel it is important to point out two substantial caveats related to the current state of the field. First, the literature reviewed here describes how captive mammals respond to noises ranging from 65 to 130 dB re 20 µPa. Although exposure to noise levels at the lower end of this spectrum may not be uncommon in some anthropogenic habitats, only a small minority of animals will encounter amplitudes at the middle and upper end of the scale. While we feel that there is much to learn from experimental studies that have utilised these extreme noise levels, given that they offer a suite of possible explanations for previously observed behavioural and fitness responses to human-generated noise, much additional work is still needed to determine which of the patterns and mechanisms discussed here are directly applicable to wildlife.

Second, we also note that the current literature is taxonomically restricted. Future research efforts should attempt to broaden our understanding of the effects of noise in a more diverse array of taxa, including those in aquatic environments; reptiles, amphibians and invertebrates have been particularly underrepresented. As noise research is conducted on an increasing diversity of focal species and populations, comparative studies may allow us to determine why different species sometimes react differently to the same noise stimuli. These data, in turn, should allow us to predict sensitivities in closely related animals, including those that cannot easily be studied in the field (e.g. endangered species). Broadening our taxonomic reach will also help future efforts in predicting ecosystem responses to anthropogenic noise.

It is important to remember that many species can detect ultra- and infrasound noise, which may have significant detrimental impacts at the levels produced in anthropogenic environments. When investigating the effects of noise on physiology, behaviour and fitness, it is helpful to determine which aspects of the acoustic stress (e.g.
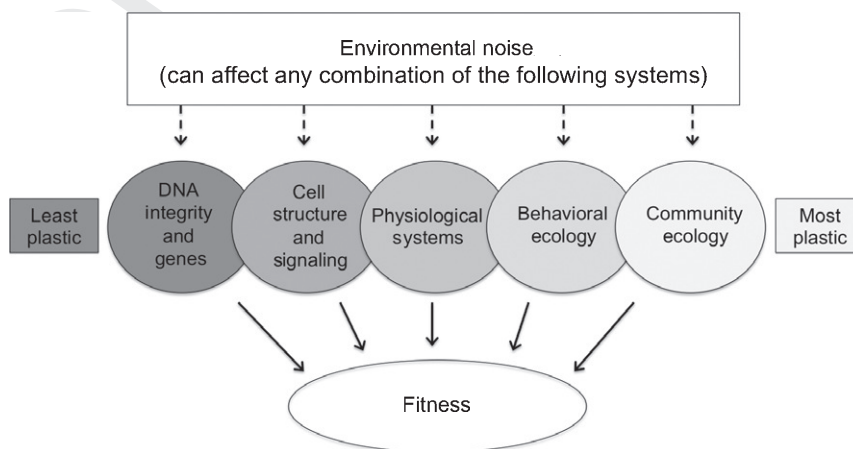

Figure 1 A conceptual framework of how environmental noise can affect biological systems, from DNA repair and gene expression (far left) through to community ecology (far right), which will then influence fitness traits. Because the interconnectedness of the systems will vary among taxa, we have not included arrows to link them. However, we do note that the systems at the right side of the continuum (behavioural and ecological processes) will tend to react more plastically to noise stimuli, allowing for more possibilities of recovery from noise perturbations, than systems towards the left end of the continuum (genetic and cellular processes).

duration, amplitude, spectral and temporal frequency and predictability) elicit various responses. This information is likely to be important on a mechanistic level, as well as for suggesting useful conservation and management strategies. Noise traits probably also influence whether or not animals will habituate during a single sustained acoustic stimulus, or across multiple stimuli repeated over time. Moreover, understanding these details may facilitate the development of management plans. However, likelihood of habituation will be related to morphological factors such as hearing sensitivity, protein distribution and gene expression, and will therefore be, to some extent, species-, or at the very least, family specific.

Perhaps the most important unanswered question in anthropogenic noise research – and in anthropogenic disturbance research, in general – is how repeated exposure over a lifetime cumulatively impacts an individual, both over the short- (e.g. condition, survival) and long- (e.g. reproductive success) terms. To this end, we advocate more comprehensive, long-term work, such as that conducted on great tits in Holland (Halfwerk & Slabbekoorn 2009; Halfwerk *et al.* 2011); introduction of physiological, cellular and genetic investigations into such a system would be extremely informative, providing a thorough understanding of responses from the molecular to the population level. In addition, use of individual marking should be encouraged to generate data on survival and return rates (e.g. MacDougall-Shackleton *et al.* 2009), which, in turn, can be used to create population and metapopulation models. However, this sort of research is not practical in all species and habitats. Conservationists and ecologists are often wary of extrapolating from controlled laboratory experiments. We contend that if we can implicate particular genetic, cellular and/or physiological mechanisms in noise-stress responses, we can become more comfortable with cross-species and cross-environment extrapolation, as these mechanisms are more likely to be conserved across species the further we delve into molecular mechanisms. Hence, our framework may be directly useful for interpreting how endangered species or those that are difficult to study in the field, will be affected by environmental noise.

Another advantage of laboratory research is the ability to more easily isolate responses to acoustic stimuli, rather than corollaries such as light pollution, habitat structure and human activity. That said, it is important to keep in mind that these various influences may interact synergistically or obstructively to influence animal physiology, behaviour and reproduction; higher-level effects will be easier to examine once we have achieved a better baseline understanding of the influence of each stimulus individually.

A decrease in human expansion is unlikely to occur in the near future, making it increasingly important to understand the implications of anthropogenic stressors, such as noise, on wildlife. We are only just beginning to discover the variety of ways in which human noise pollution impacts behaviour and fitness. A more in-depth investigation of physiological, developmental, cellular and genetic responses to noise is vital for understanding how molecular processes interact within the body and how these interactions, in turn, lead to altered behaviours and fitness outcomes.

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USING e-ANNOTATION TOOLS FOR ELECTRONIC PROOF CORRECTION

Required software to e-Annotate PDFs: <u>Adobe Acrobat Professional</u> or <u>Adobe Reader</u> (version 8.0 or above). (Note that this document uses screenshots from <u>Adobe Reader X</u>)
The latest version of Acrobat Reader can be downloaded for free at: <u>http://get.adobe.com/reader/</u>

Once you have Acrobat Reader open on your computer, click on the Comment tab at the right of the toolbar:

| | | | | | | 1 / 27 | | | 70.4% | | | | | | Tools | Comment | Share |
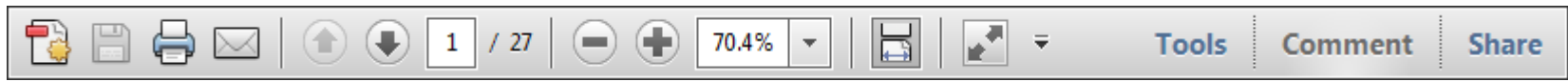
This will open up a panel down the right side of the document. The majority of tools you will use for annotating your proof will be in the Annotations section, pictured opposite. We've picked out some of these tools below:
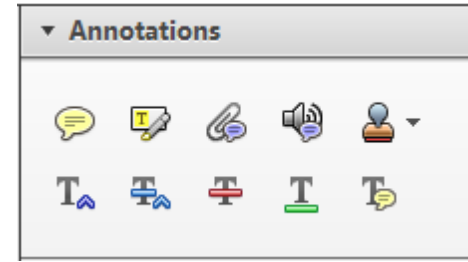
▾ Annotations

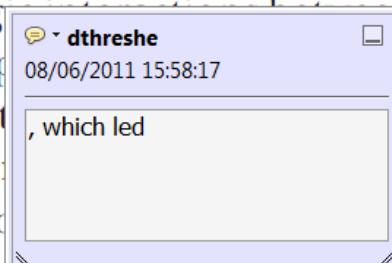### 1. Replace (Ins) Tool – for replacing text.

Strikes a line through text and opens up a text box where replacement text can be entered.

How to use it

- Highlight a word or sentence.
- Click on the Replace (Ins) icon in the Annotations section.
- Type the replacement text into the blue box that appears.

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2. Strikethrough (Del) Tool – for deleting text.

Strikes a red line through text that is to be deleted.

How to use it

- Highlight a word or sentence.
- Click on the Strikethrough (Del) icon in the Annotations section.

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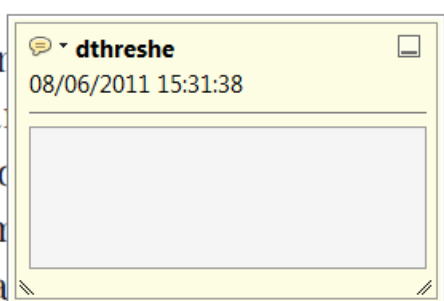### 3. Add note to text Tool – for highlighting a section to be changed to bold or italic.

Highlights text in yellow and opens up a text box where comments can be entered.

How to use it

- Highlight the relevant section of text.
- Click on the Add note to text icon in the Annotations section.
- Type instruction on what should be changed regarding the text into the yellow box that appears.

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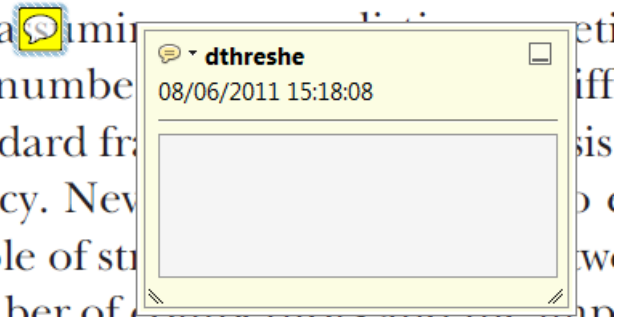### 4. Add sticky note Tool – for making notes at specific points in the text.

Marks a point in the proof where a comment needs to be highlighted.

How to use it

- Click on the Add sticky note icon in the Annotations section.
- Click at the point in the proof where the comment should be inserted.
- Type the comment into the yellow box that appears.

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# USING e-ANNOTATION TOOLS FOR ELECTRONIC PROOF CORRECTION

5. Attach File Tool – for inserting large amounts of text or replacement figures.

Inserts an icon linking to the attached file in the appropriate pace in the text.

How to use it

- Click on the Attach File icon in the Annotations section.
- Click on the proof to where you'd like the attached file to be linked.
- Select the file to be attached from your computer or network.
- Select the colour and type of icon that will appear in the proof. Click OK.

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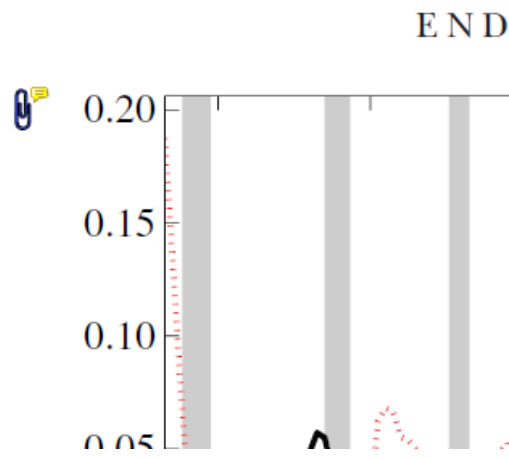
## 6. Add stamp Tool – for approving a proof if no corrections are required.

Inserts a selected stamp onto an appropriate place in the proof.

How to use it

- Click on the Add stamp icon in the Annotations section.
- Select the stamp you want to use. (The Approved stamp is usually available directly in the menu that appears).
- Click on the proof where you'd like the stamp to appear. (Where a proof is to be approved as it is, this would normally be on the first page).

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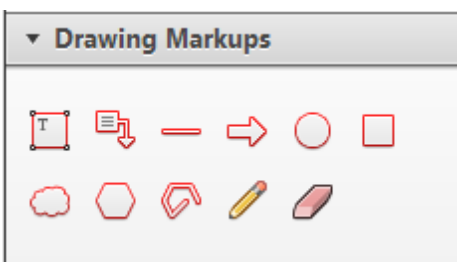
7. Drawing Markups Tools – for drawing shapes, lines and freeform annotations on proofs and commenting on these marks.

Allows shapes, lines and freeform annotations to be drawn on proofs and for comment to be made on these marks..

How to use it

- Click on one of the shapes in the Drawing Markups section.
- Click on the proof at the relevant point and draw the selected shape with the cursor.
- To add a comment to the drawn shape, move the cursor over the shape until an arrowhead appears.
- Double click on the shape and type any text in the red box that appears.

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Move to the bottom of the left column of panels.
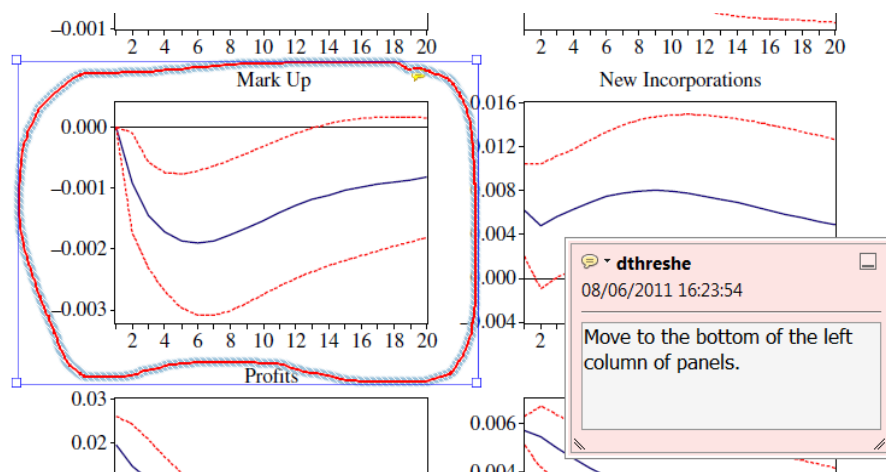
For further information on how to annotate proofs, click on the Help menu to reveal a list of further options: