Noise pollution: acute noise exposure increases susceptibility to disease and chronic exposure reduces host survival

Numair Masud[1], Laura Hayes[1], Davide Crivelli[2], Stephen Grigg[2] and Jo Cable[1]

Schools of Biosciences[1] and Engineering[2], Cardiff University, CF10 3AX, Cardiff, UK.

Correspondence: Jo Cable, Cardiff University, School of Biological Science, CF10 3AX, Cardiff, UK.

Email: cablej@cardiff.ac.uk

Abstract

Anthropogenic noise is a pervasive global pollutant that has been detected in every major habitat on the planet. Detrimental impacts of noise pollution on physiology, immunology and behaviour have been shown in terrestrial vertebrates and invertebrates. Equivalent research on aquatic organisms has until recently been stunted by the misnomer of a silent underwater world. In fish, however, noise pollution can lead to stress, hearing loss, behavioural changes and impacted immunity. But, the functional effects of this impacted immunity on disease resistance due to noise exposure have remained neglected. Parasites that cause transmissible disease are key drivers of ecosystem biodiversity and a significant factor limiting the sustainable expansion of the animal trade. Therefore, understanding how a pervasive stressor is impacting host-parasite interactions will have far reaching implications for global animal health.

Here, we investigated the impact of acute and chronic noise on vertebrate susceptibility to parasitic infections, using a model host-parasite system (guppy-*Gyrodactylus turnbulli*). Hosts experiencing acute noise suffered significantly increased parasite burden compared to those in no noise treatments. In contrast, fish experiencing chronic noise had the lowest parasite

burden. However, these hosts died significantly earlier compared to those exposed to acute and no noise treatments. By revealing the detrimental impacts of acute and chronic noise on host-parasite interactions, we add to the growing body of evidence demonstrating a link between noise pollution and reduced animal health.

Keywords – noise pollution, parasitic disease, host-parasite dynamics, animal welfare

1. Introduction

With species loss occurring 1000 times above the background rate of extinction, there is an urgent need to understand how anthropogenic activity influences ecosystem biodiversity and animal welfare (1). Anthropogenic noise is a global pollutant. It has marked impacts on human health, from reduced cardiovascular function (2-5) to elevated cortisol levels and disrupted sleep patterns (6,7). Indeed, from long term cross-sectional surveys, people report a significant reduction in their quality of life when subject to chronic noise (7). Stress responses to sound pollution have also been shown in non-human vertebrates (reviewed in 6). Bird communities, such as the greater sage-grouse (*Centrocercus urophasianus*), have elevated faecal corticosteroid metabolites and show a decline in male lek attendance when exposed to chronic and intermittent noise (8, 9). Reproductive behaviour, including anuran mate calling, is affected by chronic roadside noise, with frogs for example having to increase song pitch leading to greater energy expenditure (10). More than any other vertebrate system, mouse models have demonstrated that noise can impact behaviour, reproduction, metabolism, the cardiovascular system and immunology (reviewed in 11). Even invertebrates are not exempt from the detrimental impacts of noise pollution (12).

For aquatic organisms, including fish, the potential impact of noise pollution has only recently gained attention, and this is linked to the significant rise in underwater sonar, pile

52 driving, seismic activities and motorised vehicle activity (13). Freshwater fish in particular are
53 a global welfare concern, recognised as the most endangered group of animals on the planet
54 (14,15), in addition to being a major source of animal protein for human consumption (16).
55 Multiple fish species have displayed primary (e.g. cortisol production; 17), secondary (e.g.
56 cellular immune response; 18) and tertiary level impacts (e.g. potential disease resistance; 19)
57 of noise exposure. However, while there have been investigations on a range of tertiary level
58 impacts of noise exposure on fish (e.g. 20, 21), limited work exists on disease resistance in
59 particular.

60 Typically, animal species respond in one of three ways to noise exposure: i) no apparent
61 response to the sound stimulus (e.g. 20); ii) an initial stress response followed by acclimation
62 (e.g. 21); or iii) consistent long-term detrimental health effects (e.g. 12). Reduced resistance to
63 transmissible disease is arguably the most significant long-term welfare concern of noise
64 exposure. This is because if left untreated and/or immune suppression occurs, transmissible
65 disease will impact primary and secondary stress responses and ultimately cause mortality. To
66 date, only two animal studies have assessed the impact of noise on susceptibility to infections
67 (19, 20). Of these, only Wysocki et al. (20) demonstrated that rainbow trout (*Oncorhynchus*
68 *mykiss*) appeared unaffected by chronic 8-month noise exposure and subsequent *Yersinia*
69 *ruckeri* inoculation. Parasites causing transmissible disease are recognised as one of the most
70 significant causes of economic loss, due to host mortality in global animal trade (see 22 and
71 16). For industries such as aquaculture, infectious disease has reached crisis status exacerbated
72 by neglected stressors that compromise host immunity (23). Therefore, the functional
73 importance of stressors such as noise and its relation to disease resistance extends to impacts
74 on valuable human resources.

75 The guppy-*Gyrodactylus turnbulli* host-parasite system has been utilised to understand
76 how anthropogenic stressors impact disease resistance (e.g. nitrate enrichment: 24, animal

transport: 25). This model allows us to monitor individual infection trajectories in real-time, which we do here to assess how acute and chronic noise exposure impacts resistance to transmissible disease. The host is a globally important freshwater fish, the Trinidadian guppy that is an established eco-evolutionary model (e.g. 26). The genus *Gyrodactylus* is a group of hyperprevalent monogenean ectoparasite species of ecological and aquaculture importance (27-29). These so called 'Russian-doll killers' employ progenesis and hyperviviparity allowing parasite numbers to exponentially rise threatening host survival (reviewed in 26). *G. turnbulli* is a primary parasite of guppies and of major concern in the ornamental trade (30). As the conservation status of freshwater fish is critical (31), understanding how anthropogenic noise impacts their resistance to transmissible disease is extremely timely.

2. Methods

2.1.Host and parasite origins and maintenance

Mixed strain ornamental guppies (*Poecilia reticulata,* n=200) were purchased and transported from GuppyFarm UK to Cardiff University in September 2018. All fish were ectoparasite free on arrival, confirmed through three consecutive screens using a dissecting-microscope with fibre optic illumination (32). For experimental infections, the Gt3 strain of *Gyrodactylus turnbulli* was used which originated from a single worm isolated from an ornamental guppy in 1997. This parasite population has since been maintained in culture pots containing at least four naïve fish collectively infected with a minimum of 30 worms. Naïve guppies are added to the culture when worm numbers decrease, and heavily infected fish removed (treated) and replaced to prevent parasite extinction (29). All fish were maintained at 24 ± 1°C under a 12h light/12h dark lighting regime and fed a daily diet of tropical flakes (Aquarian®) along with freshly hatched *Artemia* nauplii every alternate day. Experimental fish were size matched adult females (SL range 14-27 mm).

2.2.Experimental design: acute and chronic noise exposure

To investigate how noise exposure impacts fish resistance to parasitic infections, guppies were allocated to either acute noise (24h, n=24, SL range 16-25 mm) or chronic noise (7 d, n=28, SL range 14-21 mm) treatments prior to parasite exposure. For each treatment, control fish (acute, n=24; chronic, n=28, SL range 14-27 mm) were placed in identical conditions but with no noise exposure. The experimental set-up for both acute and chronic treatments (Figure 1A) involved placing individual guppies in 1 l containers within a glass tank (60 x 30 x 30 cm^3) equidistant from an omnidirectional underwater speaker (UW-30, Illuminate Design Ltd., Witham). Each tank consisted of four 1 l containers per speaker (acute noise =6 replicates; chronic noise =7 replicates). The water level in the tanks was just below the rim of the 1 l containers and sufficient to ensure full submergence of the speakers. This host isolation was necessary to monitor individual infection trajectories as *G. turnbulli* can directly transfer between conspecific fish upon contact (28). To maintain water quality, all experimental fish in 1 l containers underwent complete water changes every alternate day. Underwater speakers were connected to an amplifier and subsequently a laptop to deliver the same sound file into each experimental tank. The speaker played random, intermittent white noise covering the 100-10,000 Hz range (33). These noise files were generated using VCV Rack, an open source additive synthesis software (https://vcvrack.com/), and then randomly enveloped (Figure 1B) to generate individual "bursts" of sound between 0.1 - 10s, interleaved with silence of the same random duration range. In the control (n=13) tanks, the speakers were turned off and disconnected from the main power source. We note, however, the possibility of a confound in relation to the control fish not being exposed to magnetic fields. This is due to electrical currents creating a variable magnetic field to which the voice coil in the underwater speaker responds to generate sound. No noise was transmitted between the noise exposure and control

tanks and the same noise levels were recorded within each 1 l container in each experimental tank, confirmed through hydrophone (Reson TC 4013) recordings and data acquisition system (Picoscope 5443B). The white noise emitted from the speaker was altered by reflections due to tank geometry, and the mechanical characteristics of the medium, the tank wall and tank contents. Figure 2 shows the resulting power spectrum, measured at mid depth of the 1 l fish containers and averaged over ~10s. While we are aware that fish can respond to particle motion (34), we could not measure this as we were unable to source a suitable accelerometer. However, these sound pressures are in line with mild sound levels recorded in concrete raceways, earthen ponds and indoor aquaculture systems (35).

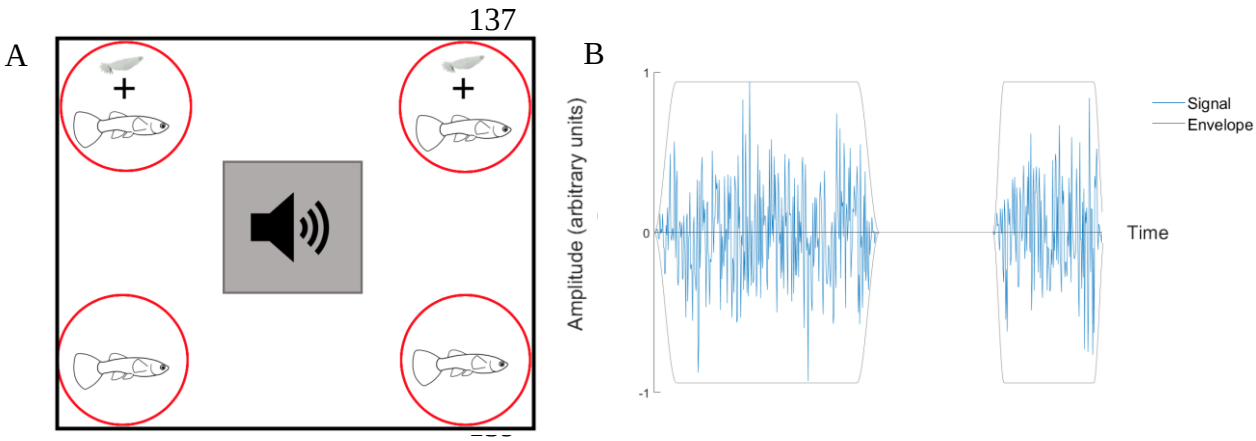

Figure 1 . (A) Schematic of general experimental design. Guppies were exposed to one of three treatments: acute noise (n=24), chronic noise (n=28) or no noise, controls (n=52). This was followed by experimental infections of half the fish with *Gyrodactylus turnbulli* parasites (shown here as grey worms, not to scale). Sound treatment design shown here, the black rectangle represents a glass tank (60 x 30 x 30 cm^3) with an underwater speaker (grey filled square; turned off in the no noise controls). Each red circle with a female guppy represents 1 l containers in which hosts were isolated for the duration of acute and chronic noise exposure as well as control treatment. (B) White noise enveloped (i.e. turning a continuous sound into bursts of shorter sounds of random length, followed by silence of random length) to generate 'bursts' of noise that was used for both the acute and chronic treatments.

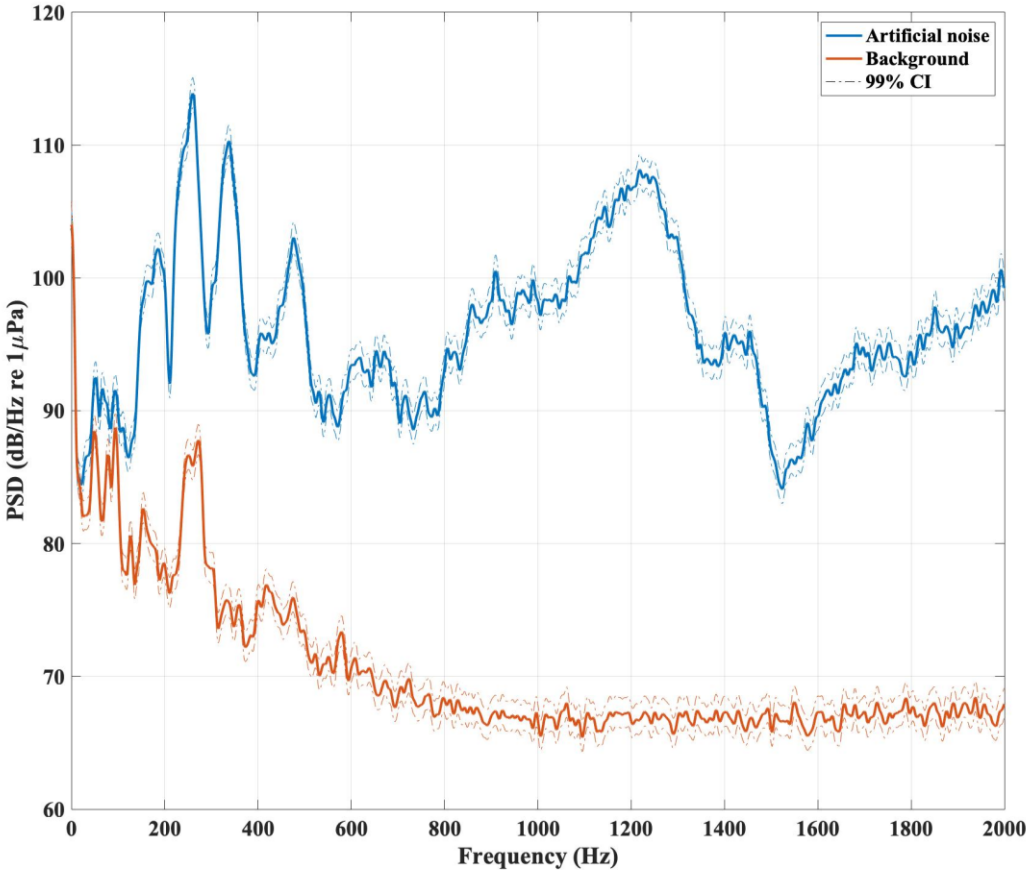

Figure 2. Power spectral density of the noise hosts were exposed to compared with the background noise inside a tank. Average of 10 distributions over 1s intervals (frequency resolution =1Hz) and 99% CI.

2.3.Experimental infections

Guppies were experimentally infected after acute (24h) or during chronic noise exposure (day 7) (Figure 3). For the chronic noise treatment, hosts that were infected with parasites continued to experience noise during infection trajectories. Thus, chronic exposure fish, experienced noise for a total of 24 days. Experimental infections involved lightly anaesthetizing individual guppies with 0.02% MS-222, and each fish was infected with two gyrodactylid worms. Parasite transfer was conducted following standard methods of King & Cable (36). Briefly, two worms from heavily infected donor fish were transferred to the caudal fin of recipient hosts by placing the anaesthetized donor fish in close proximity to an anaesthetized naïve host, monitored

continuously using a dissecting microscope with fibre optic illumination. Parasite infections were then monitored every 48h by anaesthetizing fish and counting the total number of gyrodactylids over the first 17 d of infections; a timeline determined from our previous *G. turnbulli* infections (e.g. 37).

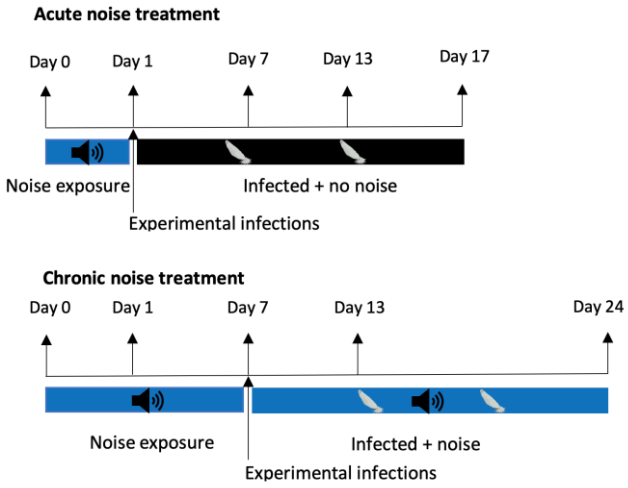

Figure 3. Timeline of when hosts were exposed to noise and experimental infection for both acute and chronic noise treatments.

To determine whether there was any immediate impact of noise exposure on *G. turnbulli* reproduction on the host, we infected n=10 size matched female guppies from the same mixed ornamental stock with 15 parasites each and exposed them to 24h of noise as detailed above. Control fish (n=10) were also infected but not exposed to noise. Over the 24h time period, fish were removed and screened at two different time points (2h and 24h) to record parasite infrapopulation numbers.

Mortality was recorded for all treatments and any fish that survived parasite infection studies were treated with Levamisole (Norbrook®) according to Schelkle et al. (38). Post-

187 treatment fish were monitored for 3 weeks and no mortalities occurred during this time. Fish
188 mortality only occurred during infections.
189
190 *2.4.Statistical analysis*
191 All statistical analyses were conducted using RStudio v2.1 (39) and final models were
192 all selected based on the lowest Akaike's information criterion (AIC) value. Peak parasite
193 burden is the maximum number of parasites at a given time point, defined here as peak day
194 (40). To quantify total infection trajectory over the 17 days, we calculated Area Under the
195 Curve (AUC) using the trapezoid rule (41). To analyse peak parasite burden, peak day and
196 AUC we used a Generalised Linear Model (GLM) with a negative binomial error family and a
197 log link function in the R MASS package. Explanatory variables for the GLM were treatment
198 (no noise, acute noise, chronic noise) standard length and mortality day. All GLM error
199 families were chosen based on the lowest dispersion parameter, theta (42).
200 A Generalised Linear Mixed Model (GLMM) with a negative binomial error family
201 and log link function was used to analyse intrinsic rate of parasite increase. A GLMM was
202 utilised as parasite data was recorded for each fish at different time points and therefore to
203 prevent pseudoreplication, Fish ID was treated as a random factor. Standard length and
204 treatment (no noise, acute noise, chronic noise) were treated as explanatory variables. As
205 experimental fish were placed in n=6 (acute treatment) and n=7 (chronic treatment) tanks, tank
206 number was also treated as a fixed factor to rule out batch effect. For all models used in
207 analysis, no batch effect was found for either noise exposure treatments ($P > 0.05$ for all
208 models). Model refinement was conducted by removing standard length in the GLM and
209 GLMM used to analyse AUC and intrinsic rates of parasite increase as it was a non-significant
210 explanatory variable (AUC: $Z=0.72$, $SE=0.01$, $P>0.05$; intrinsic rate of parasite increase:
211 $Z=0.719$, $SE=0.04$, $P>0.05$).

For analysing the *in vivo* impact of 24h noise exposure on parasite infrapopulations, a GLMM with a Poisson error family was utilised to analyse parasite count over time to prevent pseudo-replication as fish were screened at two different time points. The explanatory variable for this model being 'treatment' (i.e. noise exposure versus no-sound) and fish ID being a fixed factor. A further GLMM with a negative binomial error family and log link function was used to analyse parasite counts on hosts that survived till day 17 (days 13-17). Here, the explanatory variables were standard length and treatment. Finally, A GLM with poisson error family and log link function was used to analysing death day, where the explanatory variable was treatment as fish mortality only occurred if they were infected.

3. Results

Guppies exposed to acute noise and subsequently infected had significantly greater parasite burdens over time as measured through AUC compared to no noise controls (GLM: Z=0.08, SE=-4.14, P<0.001; Figure 4A). Fish exposed to acute noise also had significantly higher peak parasite burdens compared to controls (GLM, Z=-6.44, SE=0.09, P<0.001; Figure 4B). In contrast, guppies exposed to chronic noise had significantly reduced peak parasite burden and infection trajectories compared to controls (GLM, peak parasite burden: Z=-8.4, SE=0.07, P<0.001; AUC: Z=-9.9, SE=0.06, P<0.001). Fish exposed to chronic noise also showed a reduced intrinsic rate of parasite increase compared to control guppies (GLMM: Z=-3.554, SE=0.10219, P<0.01). When parasites were directly exposed *in vivo* to noise, the number of worms alive did not significantly change over the 24h time period (GLMM: Z=-0.43, SE=0.06, P>0.05) suggesting no immediate impact of sound exposure on the parasites.

Day on which host mortality occurred was significantly associated with peak parasite count and AUC for both acute and chronic noise treatments (AUC: Z=52.23, SE= 0.007, P<0.001, peak count: Z=39.8, SE=0.008, P<0.001) indicating that mortality of hosts influenced overall

infection trajectories. Furthermore, when analysing parasite counts on only those hosts that survived until day 17 of the infection, no significant difference was seen between chronic noise treatments and controls (parasite count from day 13-17: GLMM-Z=-1.4, SE=0.88, $P>0.05$). This reflects the fact that guppies experiencing chronic noise treatment died significantly earlier than fish experiencing acute or no noise (chronic treatment average death day=12, compared to average death day=14 for acute and control fish, GLM: Z=3.08, SE=0.03723, $P<0.01$).

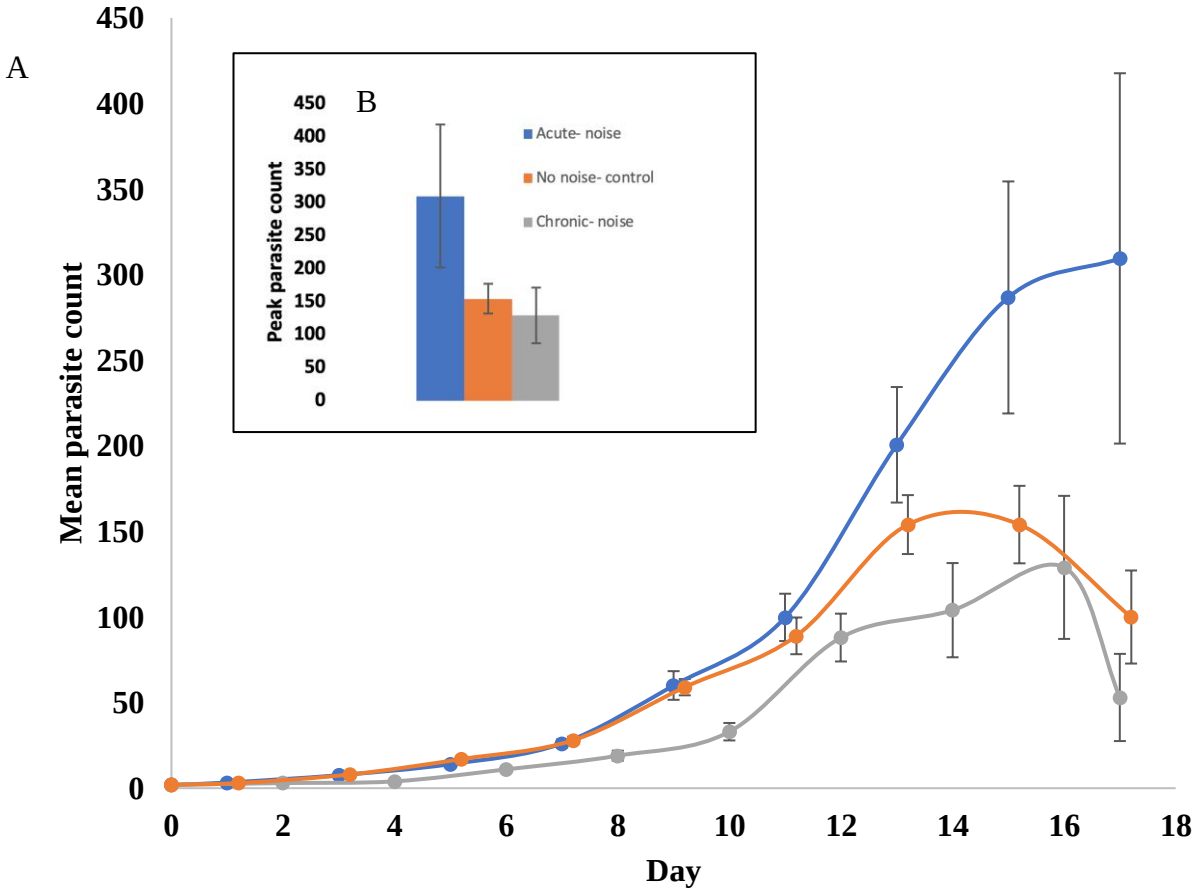

Figure 4. Mean count (A) and peak parasite burden (B) of *Gyrodactylus turnbulli* infections in guppies (*Poecilia reticulata*) exposed to either acute, chronic or no noise (controls). Standard error bars slightly transposed to one side to prevent overlap.

4. Discussion

Anthropogenic noise pollution is now a recognised welfare concern, with international regulations (e.g. 43) aiming to restrict potential detrimental health impacts. Regulations in the European Parliament, for example, have imposed restrictions on noise levels for motorised vehicles as well as introducing silencing systems, in recognition that noise can have wide ranging health impacts. Here we show fish experiencing acute noise suffered increased disease susceptibility. In contrast, chronic noise exposure significantly reduced parasite burden, but fish were prone to earlier mortality. It is likely that acute noise caused a stress response without providing sufficient time for the immune system to respond before pathogenic challenge. Acute stress has been linked to increased lymphocyte trafficking and expressions of protein cytokines from leukocytes, whereas chronic stress is associated with reduced leukocyte function (44,45). Chronic stress is generally accepted as detrimental to immunity and acute stress as potentially adaptive (44). However, our understanding of how stressors impact the immune system is largely based on *in vitro* investigations (reviewed in 46), although we are seeing an increase in studies on how stressors influence disease resistance. This is unsurprising considering the economic cost of disease for animal husbandry (16,22) and certainly for farmed aquatic species classic stressors, such as stocking density and water quality, are now known to significantly impact immune responses and disease resistance (47,48). In addition to stressors associated with husbandry practices, those linked to environmental change are also being investigated in relation to disease resistance (49). There is particular concern regarding such stressors that cross critical thresholds, termed ‘planetary boundaries’ (50), that induce physiological stress leading to system dysfunctions that includes increased disease susceptibility (51). Noise pollution, however, that may be contributing to the breach of planetary boundaries has previously been neglected in terms of disease resistance. Therefore, *in vivo* experiments,

275 combined with immunological expression studies, are needed to determine how noise has
276 functional impacts on disease resistance.

277 Chronic noise exposure can activate the immune system, with gilthead sea bream
278 (*Sparus aurata*), for example, showing significantly higher total oxidant status, lysozyme
279 activity and antiprotease activity in response to 40 days of chronic aquaculture noise compared
280 to no noise controls (18). Chronic noise exposure in mice can cause immune alterations but
281 this is dependent on strain type, with T-cell dependant antibody production and *ex vivo* T-cell
282 proliferation significantly reduced in C57Bl/6 but not BALB/c mice (52). In comparison to a
283 classic stressor (physical restraint) also applied to the C57Bl/6 mice, chronic noise had greater
284 impact on antibody production and immune cell proliferation (see 50). In our study, chronic
285 noise exposure was linked to significantly earlier host mortality and reduced pathogen burdens
286 compared to fish from acute noise and control treatments which strongly indicates that chronic
287 noise reduces pathogen tolerance. We cannot exclude the fact that direct exposure to sound
288 might also disrupt parasite development or cause them to actively move off the host. However,
289 our *in vivo* investigations suggest that, at least over 24h, noise exposure has no immediate
290 impact on parasite infrapopulations. The only research showing that sound exposure can
291 directly impact ectoparasites utilised ultrasonic waves that are of frequencies several orders of
292 magnitude higher (e.g. 53) than those used in the current study. Furthermore, at ultrasonic
293 frequencies, sound only impacted ectoparasitic lice when they were within close range of the
294 emitted sound (see 53).

295 Animal food industries, including aquaculture, are projected to see a further rise in
296 disease burden linked to increased stressors (16, 54). Here, for the first time we reveal the
297 detrimental impact of noise exposure on disease resistance and mortality. With animal
298 husbandry focussed on increasing output to meet human food chain demands, increased
299 automation and machinery use is exposing animals to further noise (11, 13). We are aware that

our study has isolated noise as an individual stressor under laboratory conditions and that animals face multiple stressors during routine husbandry. Future work must consider how noise pollution in conjunction with other common anthropogenic stressors, for instance enrichment use (55), transport (25) and manual handling (56), impact animal health. Currently, there are no effective treatments for many of the diseases that plague animal industries and the renewed emphasis on 'prevention rather than cure' means that now more than ever identifying key stressors associated with increased disease burden is an important goal towards developing sustainable preventive measures.

Ethics: All animal work was approved by Cardiff Universities Animal Ethics Committee and conducted under UK Home Office Licence (PPL 303424) following ARRIVE guidelines.

Data accessibility statement: Data is publicly available at Dryad (57) and can be accessed via the following URL:

https://datadryad.org/stash/share/2tUsOMtU2XzWEjRAqisdcxFzOVagUWU3ls8FNB4YXt4

Author contributions: NM and JC designed the study and drafted the manuscript. NM and LH conducted all experimental infections and associated data analyses. DC and SG collected and analysed the noise data. All authors commented on the final manuscript.

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