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Title

Population level effects of acoustic disturbance in Atlantic cod: a size-structured analysis based on energy-budgets.

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Author contributions

FHS contributed to the concept of the study and the design of the model, conducted the model analysis, analysed the data for the parameterization of the model, interpreted the results and wrote the manuscript.

TvK contributed to the concept of the study, provided feedback on the results and interpretation, and participated in the writing process.

HS contributed to the concept of the study, provided feedback on the results and interpretation, and participated in the writing process.

AMdR conceived the concept of the study, developed the method for the model analysis, provided feedback on the results and interpretation, and participated in the writing process.

Abstract

Anthropogenic underwater noise may negatively affect marine animals. Yet, while fish are highly sensitive to sounds, effects of acoustic disturbances on fish have not been extensively studied at the population level. In this study, we use a size-structured model based on energy budgets to analyse potential population-level effects of anthropogenic noise on Atlantic cod (*Gadus morhua*). Using the model framework, we assess the impact of four possible effect pathways of disturbance on the cod population growth rate. Through increased stress, changes in foraging and movement behaviour, and effects on the auditory system, anthropogenic noise can lead to: 1. increased energy expenditure, 2. reduced food intake, 3. increased mortality and 4. reduced reproductive output. Our results show that population growth rates are particularly sensitive to changes in energy expenditure and food intake because they indirectly affect the age of maturation, survival, and fecundity. Sub-lethal effects of sound exposure may thus affect populations of cod and fishes with similar life histories more than lethal effects of sound exposure. Moreover, anthropogenic noise may negatively affect populations when causing persistent increases of energy expenditure or decreases of food intake. Effects of specific acoustic pollutants on energy acquisition and expenditure should therefore be further investigated.

Introduction

Anthropogenic noise forms a potential threat to fish [1,2] since fish rely on advanced hearing and sound production systems for orientation and communication [3]. Although the extent varies geographically, ambient noise levels have increased considerably over the past 40 years [4,5]. This increase has been related to an increase of anthropogenic activities of which the most important are probably shipping and seismic surveys (explorations for oil and gas) [5]. Also anthropogenic activities such as drilling (oil and gas), operation of wind farms, pile driving (wind farm construction), the use of sonar (fisheries & navy) and underwater explosions produce sounds underwater. Because low-frequency sounds spread easily underwater and attenuate slowly over large distances [6,7], acoustic disturbances can lead to moderately elevated sound levels over large areas. Exposure to loud sounds, such as produced during pile driving, may cause serious (lethal) injuries in animals that are close by [8]. More often, sound exposure leads to non-lethal effects [9]. In experimental studies, anthropogenic noise has been found to increase stress, reduce foraging, reduce sound perception and increase movement in fish [9].

The non-lethal effects of sound exposure on fish seem subtle, but small changes in behaviour can lead to significant reductions in growth and reproduction [10,11]. Non-lethal effects of acoustic disturbance can be assessed using the "Population Consequences of Disturbance approach" (PCoD) framework, which was originally developed for marine mammals [11,12]. The PCoD framework translates changes in physiology or behaviour into changes in vital rates (e.g. reproduction, mortality and growth) to estimate population-level effects. Population-level effects form the basis of many current policy decisions regarding disturbance mitigation and nature conservation, such as, for example, the Birds and Habitats Directives of the European Union (Council directives 92/43/EEC [13] and 2009/147/EC [14]). However, there is currently no assessment method to estimate population-level effects of acoustic disturbances on fish.

In this study, we use a model to evaluate the population-level consequences of changes in individual-level processes that might result from lethal and non-lethal effects of sound exposure for Atlantic cod (*Gadus morhua*). The size-structured life history model for cod is based on individual energy budgets. The advantage of using such a mechanistic model is that effects of changes in food intake or energy expenditure are, through both direct and indirect effects, translated to changes in the vital rates. This type of model is considered suitable for estimating population-level effects of non-lethal disturbances [11,15]. Using the model, we explore the sensitivity of the cod population growth rate to changes in four different processes that can be affected by sound disturbance. The population growth rate is a relevant metric for population consequences of disturbances because it indicates when disturbance leads to negative population growth [16].

The effect of sound exposure on fish is not thoroughly understood and quantitative data on the relationship between sound exposure and vital rates is unavailable. Yet, a number of effect pathways have been suggested (Table S1), including increased stress, changes in foraging and movement behaviour, and effects on the auditory system. These effects may lead to changes in energy expenditure, food intake, mortality and reproduction (Table S1). We use the size-structured life history model introduced above, to examine the relative importance of these four potential effect pathways. This work lays the foundation for an assessment framework for anthropogenic noise effects on Atlantic cod populations. As understanding of the effects of acoustic disturbance on cod develops further, the model can be used to study population consequences of specific anthropogenic sources of noise pollution. The current analysis shows which mechanisms potentially lead to the largest population-level effects. The outcomes give an indication of how acoustic disturbances may affect cod most and provide guidance for future experimental and empirical research.

Model description

Population model framework

To analyse the effect of acoustic disturbances on fish, we conduct a demographic analysis of a size-structured life history model of cod. The life history model is based on the model previously described by van Leeuwen et al. [17]. Our model is adjusted to use a constant, size-dependent feeding level representing individual-level food availability. We do not consider starvation conditions; we assume a feeding level which is sufficiently high to cover the metabolic rate for fish of all body sizes. The energy budget is affected by two of the disturbance pathways that we test. As soon as the net-energy drops below zero at any point in the life history before maturation occurs, the model calculations stop. Without maturation, the population growth rate is undefined as reproduction does not take place. In other respects, we follow the model structure previously described by van Leeuwen et al. [17]. Here, we describe the model in general terms and the functions related to the implementation of acoustic disturbance. Additional details are provided in Appendix A.

From the moment an individual starts feeding actively, the model continuously tracks its age and body size. The model uses size-dependent functions for energy uptake, storage and expenditure. Energy uptake depends on the feeding level, which is defined as the food uptake rate as a fraction of the maximum feeding rate given an individual's body size. The feeding level is assumed to be size-dependent but constant in time. Reproduction is modelled as a discrete process occurring once per year. Following the demographic analysis method described in de Roos [18], we calculated population growth rates for exponentially growing populations based on Lotka's integral equation. Using this analysis method and the cod life history model, we tested the sensitivity of the population growth rate to changes in energy expenditure, food intake, mortality and reproductive output. Since fish populations are generally spread out over large areas, we expect that a given acoustic disturbance often only affects part of the population. Therefore, we tested how the disturbance of a fraction of the population, rather than the entire population, affects the population growth rate.

Accounting for acoustic disturbance

There is no quantitative empirical information available regarding sound exposure levels of cod in the field or the effects of sound exposure on cod. An overview of experimental studies with fish shows that anthropogenic noise may lead to increased stress, changes in foraging and movement behaviour, and effects on the auditory system (Table S1). The (combined) effects of anthropogenic noise may lead to increased energy expenditure, reduced food intake, an increased mortality rate, and a reduced reproductive output (Appendix A, Table S1). We therefore assessed the potential negative effects of sound exposure on cod by analysing the consequences of relative changes in its energy expenditure, food intake, mortality rate, and reproductive output on its population growth. Due to a lack of detailed information, the effects of sound exposure are assumed continuous through time and independent of age or size. As we have no quantitative information regarding the values of the disturbance parameters described below, we tested the effect of a range of values (Figure 2).

The food ingestion rate $I(l)$ depends on length l. It is defined as the ratio between the feeding level $F(l)$ and the time the individual needs to digest a unit mass of food $G(l)$ (the inverse $1/G(l)$ equals an individual's maximum feeding rate):

$$I(l) = \frac{1}{G(l)}(1 - \psi_I)F(l).$$

Food ingestion decreases proportionally with a sound exposure foraging effect parameter ψ_I. Reduced food intake as a result of sound exposure is thus defined as a proportional reduction of the standard food intake.

Ingested food is assimilated to energy, with efficiency σ. The energy is first used to cover the metabolic maintenance requirements. The net-energy $N(l, w)$ thus equals:

$$N(l, w) = \sigma\, I(l) - (1 + \psi_T)\, T(w).$$

The standard energy expenditure for metabolic maintenance $T(w)$ depends on the total body weight w. The term $\psi_T T(w)$ represents the increase in energy expenditure due to acoustic

disturbance. These costs increase proportionally with the sound exposure energy expenditure effect parameter ψ_T relative to the standard energy expenditure.

Each individual suffers from background mortality μ_0, size-dependent background mortality D_s, and, fisheries mortality D_v. These result in the following equation for the per capita mortality rate:

$$D(l) = (1 + \psi_D)\mu_0 + D_s(l) + D_v(l).$$

The term $\psi_D\ \mu_0$, background mortality multiplied by the acoustic disturbance mortality effect parameter ψ_D, represents the increase in mortality due to acoustic disturbance.

For mature individuals with sufficient energy storage (see Appendix A), spawning occurs at the end of each year n at day Υ, at time points $\zeta_n = (n\Upsilon + \Upsilon)$:

$$B = (1 - \psi_B)\frac{\sigma_r\, y_g(\zeta_n^-)}{m(l_b)},$$

$$R_0(\zeta_n^+) = R_0(\zeta_n^-) + B\, s(\zeta_n^-),$$

$$y_g(\zeta_n^+) = 0.0.$$

Here, ζ_n^- and ζ_n^+ respectively represent the time points just prior to and following reproduction. The number of offspring B that the individual produces depends on the mass of the gonads y_g prior to spawning, the mass at the size of birth $m(l_b)$ and the gonad-to-offspring conversion efficiency σ_r. The number of offspring produced decreases proportionally with the acoustic disturbance reproductive failure effect parameter ψ_B. To calculate the expected cumulative lifetime reproductive output R_0, the number of offspring is multiplied by the survival probability s of the individual and added to the offspring the individual has produced so far. After spawning the gonadal mass is depleted, while all other variables are unchanged.

Analytical method

Individual life histories were modelled with a mix of continuous time ordinary differential equations (ODEs) and discrete time recurrence relations (see Appendix A). The computation of

the population growth rate follows the approach presented by de Roos [18]. This method finds the population growth rate $\tilde{r}$ by calculating the value of r that satisfies the equation:

$$L(A_n, r) = \sum_{i=1}^{n} (R_0(A_i) - R_0(A_{i-1}))e^{-rA_i} = 1.$$

This is equivalent to the discrete-time Euler-Lotka equation for computation of the population growth rate, r. As in the discrete-time Euler-Lotka equation, the summed quantity $L(A_n, r)$ discounts the expected offspring produced at every age with the growth-rate dependent factor e^{-rA_i}. The expected cumulative lifetime reproductive output $R_0(A_i)$ represents reproduction up to and including reproduction occurring at age A_i, which depends on the survival probability up to age A_i. The increase in lifetime reproductive output R_0 from age A_{i-1} to age A_i is computed by integration of the continuous-time ODE system for life history processes and application of recurrence relations for discrete events related to reproduction (see Appendix A). The maximum age A_n is defined as the moment at which the survival probability of the individual is lower than 10^{-9}.

When a fraction p_s of the population experiences a disturbance, the population growth rate r is equal to the value for which the dominant eigenvalue of the following matrix is 1 (see [18], for the theoretical background):

$$\begin{array}{c} \\ \text{Unstressed offspring} \\ \text{Stressed offspring} \end{array} \begin{array}{c} \begin{array}{cc} \text{Unstressed parent} & \text{Stressed parent} \end{array} \\ \begin{pmatrix} (1-p_s)\,L_{ns}(A_n, r) & (1-p_s)\,L_s(A_n, r) \\ p_s\,L_{ns}(A_n, r) & p_s\,L_s(A_n, r) \end{pmatrix} \end{array}.$$

Stressed individuals, which experience a disturbance, produce an expected number $L_s(A_n, r)$ of offspring during their lives. The analogous quantity for unstressed individuals is given by $L_{ns}(A_n, r)$. Of these newly produced offspring, a fraction p_s will experience a disturbance, while a fraction $(1 - p_s)$ will not. The resultant population growth rate r of a partly stressed population hence satisfies the condition:

190 $$\begin{vmatrix} (1-p_s)\,L_{ns}(A_n,r)-1 & (1-p_s)\,L_s(A_n,r) \\ p_s\,L_{ns}(A_n,r) & p_s\,L_s(A_n,r)-1 \end{vmatrix} = 0.$$

191 We used the R package deSolve [19] to solve the system of ODEs and recurrence relations. The
192 population growth rate calculations were executed using a C-based, open source software
193 package that solves generic systems of nonlinear equations
194 (https://bitbucket.org/amderoos/findcurve). We made the model implementation files publicly
195 available online (files available from the journals office).

196 *Parameterization of the model*

197 Parameters and their values are listed in Table S2; details regarding parameter derivation are
198 described in Appendix A. The parameter values used by van Leeuwen et al. [17] are based on
199 Atlantic cod in the Baltic Sea. We adjusted length at maturation, adult condition target and size-
200 dependent functions for the maintenance rate, digestion time and fisheries retention (Figure S1)
201 on the basis of available literature data on Atlantic cod in the North Sea. Otherwise, parameter
202 values are as given in van Leeuwen et al. [17].

203 The feeding level $F(l)$ is assumed constant in time, but body size-dependent (Figure S1). The
204 high feeding-level function corresponds to a situation with unlimited food (Appendix A). Under
205 these conditions, growth depends only on the parameters of maximum feeding and energy
206 expenditure. These were derived from experimental data from the literature (Appendix A). We
207 chose the shape and parameters of an intermediate and a low feeding level function to match
208 observed growth patterns of Atlantic cod in the North Sea (Figure 1). Together with the high
209 feeding level function, the intermediate and low feeding level functions cover the range of
210 observed growth patterns of Atlantic cod in the North Sea (Figure S1, Figure1).

211 *Data of Atlantic cod*

212 We used lengths at age from North Sea IBTS-survey data for Atlantic cod between 1970 – 2018
213 (Figure 1, [20]). We adjusted the ages for the quarter of the year in which the survey took place
214 (quarter 1, no adjustment; quarter 2, +0.25 year; quarter 3, +0.5 year; quarter 4, +0.75 year).

Fecundity-length relationships are based on field data of Atlantic cod in the North Sea in several different years (Figure 1, [21]).

Results

For unlimited food (high feeding level), we compared model output to length-at-age and fecundity-length data for Atlantic cod in the North Sea. The model growth curve for the high feeding level corresponds well to the high end of the length-at-age data range (Figure 1). This indicates that maximum growth in the model is similar to that in field observations. Fecundity in the model is similar to field observations for small-sized cod but deviates for large-sized cod (Figure 1).

Without acoustic disturbance, the population growth rate is estimated to be 0.0125 for high, 0.0072 for intermediate and 0.0048 for low feeding levels (Figure 2). The population growth rates thus predict undisturbed populations to grow for all three feeding levels. The population growth rates are negatively affected through all sound exposure effect pathways. They are more strongly affected by increased energy expenditure and a lower food intake than by additional mortality and lower reproductive output (Figure 2). For the highest feeding level, the population growth rate becomes negative with a ~60% increase in energy expenditure or a ~35% reduction of the food intake. This switch occurs at ~450% additional mortality and a ~99.9% reduction of the reproductive output (Figure 2). For intermediate and low feeding levels, the population growth rate is lower overall. As a result, it becomes negative already at lower disturbance levels (Figure 2). For example, for the low feeding level, a negative population growth rate already occurs at a ~20% reduction in food intake (Figure 2B).

These results are based on a situation where the entire population is affected equally. We also test the effect of the proportion of the population that is disturbed (Figure 3). Increasing the proportion affected decreases the population growth rate. The shape of this relationship depends on the strength of the disturbance. For a weak disturbance, for example a 10% increase of the energy expenditure, the population growth rate shows a slow decrease with the

proportion affected (Figure 3). For strong disturbances, the population growth rate initially decreases slowly. When 50% or more of the population is affected, it decreases more rapidly. The shape of the relationship between the population growth rate and proportion affected is independent of the sound exposure effect pathway (energy expenditure, food intake, mortality or reproductive failure, results not shown).

The individual-level life history trajectories can be used to explain the different effects of the sound exposure effect pathways on the population growth rates (Figure 4). A reduction of the population growth rate results from a decrease of the cumulative lifetime reproductive output. The cumulative lifetime reproductive output is more strongly affected by a 30% decrease of the food intake than by 30% additional mortality (Figure 4A, B). It depends on age at maturation, survival and the annual reproductive output. Somatic growth is inhibited by a lower food intake but unaffected by additional mortality (Figure 4C, D, note that the black line lies on top of the green line). As a result, maturation is delayed from year 2 to 5 for individuals with a lower food intake (Figure 4C, D). Survival is reduced by both a lower food intake and additional mortality (Figure 4E, F). For the lower food intake, individuals grow more slowly and are subject for longer to high mortality in the smallest size range (Figure S1D). Finally, the energy in the gonads, and thus the annual reproductive output, is reduced by a lower food intake but unaffected by mortality (Figure 4G, H). In summary, changes in food intake directly affect the individual growth curve and indirectly affect the age at maturation, the survival up to maturation and the annual reproductive output. On the other hand, mortality and reproductive failure directly reduce respectively survival and the annual reproductive output, while both have no further indirect effects. The effect of increased energy expenditure is similar to a reduction in food intake: both lead to a reduction of the net-energy availability and affect the individual growth curve.

Discussion

Our study uses a size-structured life history model to evaluate population-level consequences of changes in individual-level processes that might result from noise pollution for Atlantic cod. The model framework incorporates energetics and, with the exception of the fecundity of large cod, matches patterns of maximum growth and reproductive output observed for cod in the field. Based on experimental studies with fish, anthropogenic noise may directly lead to higher energy expenditure, lower food intake, higher mortality and lower reproductive output (Table S1). Of these four possible effect pathways, a higher energy expenditure and a lower food intake have a strong effect on the population growth rate in particular. This is because indirect effects lead to an increased age at maturation, a decreased survival up to maturation and a decreased annual reproductive output. The population growth rate decreases most rapidly in response to disturbances that affect at least 50% of the population.

In this study, we test the relative importance of gradual changes in four processes that could be affected by acoustic disturbance. We have chosen this approach because there is still insufficient empirical information available to relate sound exposure explicitly to changes in life-history parameters. The actual importance of each of the pathways is, of course, determined by how strongly each of them is affected by sound exposure. For example, despite the fact that the population growth rate is more sensitive to changes in food intake, a large increase in mortality per-unit-disturbance may cause a stronger effect on the population growth rate than a small decrease in food intake per-unit-disturbance. When dose-response relationships that estimate effects of sound exposure for cod become available, the modelling approach we have developed can be used to estimate the effects of sound exposure on cod populations. Our results suggest that the strongest population level effects will, through effects on energetics, stem from the sub-lethal effects of sound exposure on individuals.

Empirical sound exposure studies

In our model, the population growth rate is most sensitive to sound exposure effects through increased energy expenditure and a lower food intake. The energy expenditure and food intake of fish are likely affected by anthropogenic noise through stress and changes in foraging and movement behaviour (Table S1). Stress increases the metabolic rate [22]. Foraging success would be affected by sound exposure when it distracts fish from or masks acoustic stimuli of prey [23,24]. Alternatively, foraging may be affected by sound exposure indirectly through shifts in behaviour [23] or lower appetite due to stress [25,26]. Changes in movement behaviour in response to anthropogenic noise include changes such as higher activity and swimming speed as well as partial disintegration of schools [27–29], which all cost energy [30,31].

At the same time, the population growth rate is relatively insensitive to direct additional mortality and reduced reproductive output. At the lowest feeding level, the population growth rate becomes negative only when mortality reaches ~250% compared to natural mortality. Fish mortality after sound exposure has mostly been studied for pile driving [e.g. 8,32,33]. It is generally thought that mortal injuries after sound exposure occur in relatively few individuals, situated close to the sound source. Mortality after sound exposure might also occur further away from the source, through additional predation mortality due to masking [34]. For example, predation risk was found to increase for Ambon damselfish (*Pomacentrus amboinensis*) exposed to boat noise [35]. However, in a recent meta-analysis of sound experiments with fish, predation mortality showed no significant relation with anthropogenic noise [9]. Reproductive output may also be directly affected by sound exposure, as the mating success of cod depends on auditory cues [36]. However, our results show that the population growth rate is only significantly reduced by a strong decrease of the reproductive output.

The high sound exposure levels needed for direct mortality are likely to occur only in limited areas directly around loud sound sources. Since sound attenuates over large distances underwater, low to moderate sound exposure levels will be experienced by many individuals

during sound disturbances. These scale differences imply that the sub-lethal effects of sound exposure are likely to occur in a larger part of the population than lethal effects. The most influential sound exposure effect pathways at the population level could thus also be the pathways that occur on a larger scale at the individual level.

In summary, empirical support exists for the effect of sound exposure on fish through all of the four pathways that we investigated. Our understanding is far from complete [37], also because different fish species react differently to anthropogenic noise [38]. While the effect of sound exposure on Atlantic cod specifically has received little attention, available studies of cod indicate that sound exposure may affect foraging activity and movement [39], cortisol levels [40] and larval growth [41]. A more exact quantification of the effects of sound exposure on cod is needed to allow assessments of the impact of noise pollution on cod populations.

Theoretical sound exposure studies

Previous theoretical studies applied a bioenergetics approach to study population consequences of sound exposure for several species of marine mammals [e.g. 15,42–44]. Our study is the first to develop such methodology for a species of fish. A similar approach was used by Hin, Hardwood and de Roos [15] to study the effect of sound disturbance on the population growth rate of pilot whales (*Globicephala melas*). Together with the work described here, this illustrates the usefulness of our methodology; an energy-budget model continuously tracks the effect of sound exposure on growth, reproduction and survival throughout the life history of an individual. Subsequently, it expresses the significance of these effects on the population level in the form of changes in the cumulative lifetime reproductive output and population growth rate. The approach appears to be generally applicable across different taxa.

Future model improvements

Our model contains size-dependent functions for feeding and energy expenditure that are parameterized on the basis of empirical data. Our model predictions match maximum growth observations of Atlantic cod quite well. Yet, like many other theoretical models [45], our model underestimates the fecundity of large fish. This is either due to an underestimation of the

342 feeding rate, or, an overestimation of the energetic or reproduction costs for these large-sized
343 individuals. As a consequence, our model may underestimate the population growth rate of cod
344 and the sensitivity of the population growth rate to lower food intake and increased energy
345 expenditure. A lower food intake and increased energy expenditure reduce early stage survival
346 and thus the occurrence of large-sized individuals.

347 Our model could be further refined by incorporating temporal variation, in terms of life history
348 stages, seasonality and sound exposure. Life history is likely to modulate the effects of sound
349 exposure, since cod undergo morphological, diet and habitat changes over their lifetime. If the
350 effects of sound exposure or sound exposure levels change between life stages, this could affect
351 our results but it is impossible to say how. Seasonal variation in sound exposure can be
352 important when the food availability displays seasonal variation and sound exposure decreases
353 food intake. For example, for pilot whales, sound exposure is expected to have a stronger effect
354 during a period with low food availability [15]. Furthermore, sound exposure may affect species
355 that cod depend on as a food source [46,47]. The effect of changes in food availability can be
356 assessed by changing the feeding level function in the current framework. Finally, the model
357 assumes processes to be density independent. A more complex, density-dependent model
358 framework, which is available for cod, includes multiple food sources and feedbacks between the
359 food sources and the cod population [17]. However, this level of model complexity is unsuited
360 for a first exploration of potential effects with unknown magnitude.

361 *Perspectives for future studies*

362 During spawning, cod aggregate in specific areas [48] and male cod produce mating grunts
363 during courting [36]. Sound exposure of cod during the spawning period could thus potentially
364 result in failure of reproduction for part of the population. It is often thought that reproduction
365 is the most sensitive part of cod life history [49]. At the same time, our analysis shows that, for
366 cod, reproductive failure per se does not have a strong effect at the population level. Our work
367 highlights that subtle effects of sound exposure on fish, e.g. on their behaviour and physiology,

most easily reduce population growth rates. This finding has important ramifications for future experimental and empirical work, as well as for management aimed at mitigating effects of sound exposure. This work calls for elucidation of the relationship between sound exposure and individual-level effects for cod and other fish species. Only then, can our model framework be used to properly assess the effects of marine underwater noise disturbance.

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Appendix A

Acoustic disturbance

Based on a meta-analysis of sound exposure experiments [1], we summarize known effects of anthropogenic noise for fish in Table S1. Subsequently, we identify, based on documented responses of fish to changes in physiology or behaviour, potential direct consequences for individual-level processes in the model (energy expenditure, food intake, mortality and reproductive output; Table S1). It should be noted that, while many acoustic disturbances are of an impulsive nature (seismic surveys, pile driving), our choice of 'potential effects' relate to regular exposure to such pulses, not the effects of individual pulses. We give a broad overview of how acoustic disturbances may affect individual-level processes (Table S1) to motivate the link between acoustic disturbances and changes in individual level processes.

Across different fish species, anthropogenic noise has been shown to increase stress and affect foraging behaviour, movement behaviour and the auditory system [1]. Stress has been linked to increased energy expenditure, decreased food intake, increased mortality and decreased reproductive output (Table S1). Changes in foraging behaviour may result in increased energy expenditure or lower food intake, while short-term consequences for mortality or reproductive output seem unlikely (Table S1). Changes in movement behaviour may lead to increased energy expenditure (Table S1) but have not been documented to affect food intake or reproductive output. Changes in the auditory system of fish may lead to decreased food intake, increased mortality and decreased reproduction (for example, through the negative consequences of elevated hearing thresholds; Table S1). Although it has often been argued that displacement from foraging areas and spawning grounds affects fish [2], these effects play on a larger scale than the experiments reviewed by Cox et al. [1] and we did not include this link in our table. Yet, we do analyse the effect of reduced reproductive output on the population growth rate based on another link in the table. In summary, anthropogenic noise could eventually lead to increased energy expenditure, reduced food intake, increased mortality and reduced reproductive output for individual cod (Table S1).

539 *Model formulation*

540 The body mass of an individual is assumed to consist of structural mass x (muscles and bones),
541 reserves y_r (lipids and fat) and gonads y_g. Total individual body mass w is hence given by:

542 $$w = x + y_r + y_g.$$

543 The mass-length relationship equals:

544 $$x = \frac{\gamma\, l^{\delta}}{(1 + q_j)},$$

545 which relates the structural mass x to the condition parameter q_j and the length l with scaling
546 parameters γ and δ.

547 Food ingestion follows a Holling type-II functional response. Unlike van Leeuwen et al. [3], we do
548 not explicitly consider multiple food resources. Instead, the food ingestion rate $I(l)$ is defined as
549 the ratio between the feeding level $F(l)$ and the time the individual needs to digest a unit mass
550 of food $G(l)$ (the inverse $1/G(l)$ equals an individual's maximum feeding rate):

551 $$I(l) = \frac{1}{G(l)}(1 - \psi_I)F(l).$$

552 The feeding level $F(l)$ is assumed constant in time, but body size-dependent (figure S1B). Food
553 ingestion decreases proportionally with acoustic foraging disturbance multiplier ψ_I.Digestion
554 time scales with length as:

555 $$G(l) = \varepsilon\, l^{\theta},$$

556 with scaling constant ε and exponent θ.

557 The net-energy $N(l, w)$ equals:

558 $$N(l, w) = \sigma\, I(l) - (1 + \psi_T)\, T(w).$$

559 Ingested food is assimilated to energy with efficiency σ. The energy is then first used to cover the
560 energy expenditure for metabolic maintenance. The standard metabolic maintenance
561 requirements are represented by term $T(w)$. The energy expenditure increases proportionally
562 with the acoustic disturbance energy expenditure multiplier ψ_T relative to the standard
563 metabolic maintenance requirements. The standard metabolic requirements depend on the total
564 body weight, including both structural and reversible mass:

565 $$T(w) = \alpha\, w^{\beta},$$

566 with scaling constant α and exponent β.

567 To assess the population growth rate, we consider a situation with ample food availability. The
568 net-energy is always taken to be positive and we do not consider starvation conditions.

569 A proportion k of the net-energy is invested in growth in structural mass; the remaining
570 proportion $1 - k$ of the net-energy is invested in reversible and gonad mass (see below). The
571 proportion k is defined as:

572 $$k\big(l, x, y_r, y_g\big) = \begin{cases} \dfrac{1}{q(l)+1}, & \dfrac{y_r + y_g}{x} > q(l) \\ \dfrac{1}{q(l)+1}\left(\dfrac{y_r + y_g}{q(l)\,x}\right)^2, & \text{otherwise.} \end{cases}$$

573 $$q(l) = \begin{cases} q_j, & l < l_m; \\ q_a, & \text{otherwise.} \end{cases}$$

574 The proportion k depends on the ratio between the reversible $(y_r + y_g)$ and structural (x) mass.
575 The proportion k targets for a constant ratio $q(l)$ between reversible and structural mass (note
576 that $k = 1/(q(l)+1)$ when $y_r + y_g = q(l)x$). When the actual ratio between reversible and
577 structural mass drops below the target condition level $q(l)$, $k(l, x, y_r, y_g)$ decreases with a
578 decrease in the ratio between the reversible and structural mass. This slows down growth in
579 structural mass and favours restoring reserve mass to reach the target condition. The value of
580 $q(l)$ depends on the size of the individual, it is equal to q_j before, and equal to q_a after they have

become sexually mature. Sexual maturity is reached when an individual reaches the size at maturation l_m. Since $q_j < q_a$, adults accumulate more reserves than juveniles, a surplus they allocate to reproduction.

For each individual, mortality is a sum of background mortality μ_0, size-dependent background mortality D_s, and, fisheries D_v mortality, resulting in the following equation for the per capita mortality rate:

$$D(l) = (1 + \psi_D)\mu_0 + D_s(l) + D_v(l).$$

The term $\psi_D\ \mu_0$, background mortality multiplied by acoustic disturbance mortality multiplier ψ_D, represents the increase in mortality due to acoustic disturbance. The size-dependent background mortality $D_s(l)$ decreases exponentially with body size:

$$D_s(l) = \mu_s e^{-(\frac{l}{l_s})^{\delta}},$$

with mortality constant μ_s and characteristic size l_s. Fisheries mortality $D_v(l)$ depends on body size following a double sigmoid function (see Figure S1C):

$$D_v(l) = \mu_v\big(X(l, l_v, l_{vh}) - (1-\rho)X(l, l_{vd}, l_{vdh})\big),$$

in which $X(l, l_1, l_2)$ is defined as:

$$X(l, l_1, l_2) = \begin{cases} 0.0, & nx \le 0.0 \\ \frac{1}{6}nx^3, & 0.0 < nx \le 1.0 \\ (-\frac{3}{2}nx + \frac{3}{2}nx^2 - \frac{1}{3}nx^3 + 0.5), & 1.0 < nx \le 2.0 \\ (\frac{9}{2}nx - \frac{3}{2}nx^2 + \frac{1}{6}nx^3 - 3.5), & 2.0 < nx \le 3.0 \\ 1.0, & \text{otherwise} \end{cases} \quad \text{with } nx = 1.5\,\frac{(l - l_1)}{(l_2 - l_1)}.$$

The fisheries mortality depends on mortality constant μ_v, the average fisheries retention in the largest size classes' ρ and sigmoid function $X(l, l_1, l_2)$. The sigmoid function depends on the body length l, the length at the start l_1 and at 50% l_2 of the s-shaped part of the function. For the increasing part of $D_v\ (l)$, the sigmoid is defined by the length at the start (l_v) and at 50% (l_{vh}) of

fisheries retention. For the decreasing part of $D_v(l)$, the sigmoid depends on the length at the start (l_{vd}) and at 50% (l_{vdh}) of the fisheries mortality decline (Figure S1C).

The individual life history is a mix of continuous and discrete time processes that operate within each year. The individual-level functions for feeding, energy allocation and mortality lead to a set of ODEs that describe the continuous-time changes of the individual throughout its life. The age a, structural mass x, length l and survival probability s change continuously with time τ as:

$$\frac{da}{d\tau} = 1.0,$$

$$\frac{dx}{d\tau} = \kappa\, N,$$

$$\frac{dl}{d\tau} = \frac{dx}{d\tau} \Big/ \frac{dx}{dl} = kN\, \frac{l}{\delta\, x},$$

$$\frac{ds}{d\tau} = -D(l)s.$$

The dynamics of the reversible mass of reserves y_r and gonads y_g depend on the time of the year and the sexual maturity of the individual. During the first period, with length τ_r, of each year, the energy invested in reversible and gonad mass is stored in the reserves only and the gonads remain empty. Within each year n with length Υ, these dynamics during the intervals $(n\Upsilon) \leq \tau < (n\Upsilon + \tau_r)$ are hence described by:

$$\frac{dy_r}{d\tau} = (1-k)\,\eta$$

$$\frac{dy_g}{d\tau} = 0.0.$$

At day τ_r within each year (that is, at all time-points $\varrho_n = (n\Upsilon + \tau_r)$), the individual decides whether it will spawn at the end of the year. This decision depends on the reserve mass in relation to the target body condition:

$$y_g(\varrho_n^+) = \begin{cases} 0.0, & y_r(\varrho_n^-) \leq q_j x(\varrho_n^-) \\ y_r(\varrho_n^-) - q_j x(\varrho_n^-), & y_r(\varrho_n^-) > q_j x(\varrho_n^-) \end{cases}$$

623 $$y_r(\varrho_n^+) = \begin{cases} y_r(\varrho_n^-), & y_r(\varrho_n^-) \le q_j x(\varrho_n^-) \\ q_j x(\varrho_n^-), & y_r(\varrho_n^-) > q_j x(\varrho_n^-) \end{cases}.$$

624 If the ratio of reserves over structural mass is smaller than or equal to q_j, the individual will not
625 spawn because any investment in reproduction would lower the body condition below the
626 target condition q_j. In this case, all variables stay the same. On the other hand, if the ratio of
627 reserves over structural mass is larger than q_j, the individual will spawn. In this case, the
628 surplus of reserve mass, in excess of the target body condition q_j, is transferred to the gonads.
629 All other variables stay the same.

630 Depending on the decisions about spawning at time points ϱ_n, the dynamics that occur
631 subsequently until the end of the year, during intervals $(n\Upsilon + \tau_r) \le \tau < (n\Upsilon + \Upsilon)$, are:

632 for years without spawning:

633 $$\frac{dy_r}{d\tau} = (1-k)N,$$

634 $$\frac{dy_g}{d\tau} = 0.0,$$

635 for years with spawning:

636 $$\frac{dy_r}{d\tau} = \begin{cases} (1-k)\,N\,, & l < l_m, \\ 0.0, & l \ge l_m, \end{cases}$$

637 $$\frac{dy_g}{d\tau} = \begin{cases} 0.0, & l < l_m, \\ (1-k)N, & l \ge l_m. \end{cases}$$

638 If no spawning occurs, energy invested in reversible mass is stored in reserves by all individuals.
639 In years with spawning, energy invested in reversible mass is stored in the gonads by
640 individuals with a length larger than l_m. while energy invested in reversible mass is stored in
641 the reserves by immature individuals, with length smaller than l_m .

642 Spawning occurs at the end of each year at day Υ, at the time points $\zeta_n = (n\Upsilon + \Upsilon)$:

643 $$B \quad = (1-\psi_B)\frac{\sigma_r\, y_g(\zeta_n^-)}{m(l_b)},$$

$$R_0(\zeta_n^+) = R_0(\zeta_n^-) + B\, s(\zeta_n^-),$$

$$y_g(\zeta_n^+) = 0.0.$$

The number of offspring B that an individual produces depends on the mass of the gonads, the mass $m(l_b)$ at the size of birth l_b and the gonad-to-offspring conversion efficiency σ_r. The number of offspring produced decreases proportionally with the reproductive failure multiplier ψ_B due to acoustic disturbance. To calculate the lifetime reproductive output R_0, the number of offspring B is multiplied by the survival probability of the individual and added to the offspring the individual has produced so far. The gonadal mass is set to zero. All other variables do not change.

The initial state of an individual in the model is defined at the moment at which the individual starts feeding actively. At this moment, the individual age is a_b and body size is l_b. Its survival probability up to that moment depends on the mortality in the egg and larval phase μ_e. The individuals reserves contain a portion of the total mass such that, while the gonads are empty, the ratio between reversible and structural mass is equal to the target ratio q_j. This results in the following state of the model variables for newborns:

$$a(0) = a_b,$$

$$x(0) = \frac{1}{(1+q_j)} m(l_b),$$

$$l(0) = l_{b,}$$

$$s(0) = e^{-\mu_e a_b},$$

$$h(0) = 0.0,$$

$$y_r(0) = \frac{q_j}{(1+q_j)} m(l_b),$$

$$y_g(0) = 0.0.$$

Feeding level functions

For the intermediate and low feeding-levels, we assume sigmoid function:

$$F = f_1 + f_2 X(l, l_1, l_2).$$

The feeding-level starts at level f_1 and gradually shifts following function X with length to $f_1 + f_2$. The shift starts at l_1 and at length l_2 the shift is halfway. With $l_1 = 0.39$ cm and $l_2 = 30$ cm, we define the intermediate feeding level function as $F_{int} = 0.7 + 0.25\, X(l, 0.39{,}30)$ and the low feeding level function as $F_{low} = 0.55 + 0.35\, X(l, 0.39{,}30)$ (Figure S1B).

Model parameters

Parameters and their values are listed in Table S2. The parameter values used by van Leeuwen et al. [3] are based on Baltic Sea cod. To represent Atlantic cod in the North Sea, we adjust the length at maturation (l_m), adult target condition (q_a) and the size-dependent functions for fisheries retention, energy expenditure for metabolic maintenance, and digestion time (Figure S1A-C). The derivation of these parameter values is described below. Otherwise, we use the parameter values as given in van Leeuwen et al. [3].

The metabolic rate and the digestion time are temperature dependent. The average annual North Sea SST is ~10 ˚C [4]. Assuming the sea bottom is about 2 degrees colder, we use 8 ˚C for the derivation of the size-dependent metabolic rate and the digestion time. The metabolic rate is derived from oxygen consumption of cod reared in light at 8 ˚C [5]. We convert dry-to-wet body-mass with conversion factor 5 [see data in 5], oxygen to grams energy using an oxy-calorific coefficient of 13.6 kJ $\cdot$ g^{-1} O_2 [6] and energy-to-wet-weight ratio of 7 kJ/g [7]. To compress annual activity into $Y = 250$ days (Table S2), we rescale the metabolic rate parameters to $\beta = 0.9124$ and $\alpha = 0.022$ (Figure S1A). The digestion time G is derived from data of maximum growth in body mass in Atlantic cod, fed ad lib in captivity [8]. For a situation with maximum feeding, the growth in body mass equals the weight increase (N) in the model; $N = \sigma \frac{1}{G} - T$. We derive digestion time scaling parameters $\varepsilon = 270.651$ and $\theta = -2.389$ from the growth rate between $l = 0.39 - 150$ cm at 8 ˚C, using a year with length $Y = 250$ days, $\sigma = 0.6$ (Table S2) and the metabolic rate above (Table S2, Figure 1B) .

Fisheries retention is defined as the ratio between the age-specific annual fisheries mortality rate and the maximum fisheries mortality rate in the same year. We use data from the ICES fisheries assessments between 2000-2016 [9]. Each age class is converted to a mean annual size-at-age based on the mean size per age class in the years 2000-2016 of the cod IBTS-survey data [10]. We use a double s-shaped function for the relationship between fisheries retention and body size and fitted the curve to the data points manually (Figure S1C). Fisheries retention starts at $l_v = 10$ cm and reaches 50% of its maximum at $l_{vh} = 34$ cm. From $l_{vd} = 58$ cm, fisheries retention gradually decreases to the average retention in the last age class (6+ years old) $\rho = 0.55$ and reaches 50% of this level at $l_{vdh} = 78$ cm. Over the last ten years, the maximum fisheries mortality for North Sea cod ranged between 0.35 (2016) and 0.75 (2006; [9]). We use a daily mortality rate of $\frac{0.31}{\Upsilon} = 0.00124\ \mathrm{d}^{-1}$, which is the currently advised maximum cod fisheries mortality [11].

The typical size at maturation of Atlantic cod varies across regions; we use $l_m = 62$ cm, which is the length where 50% of Atlantic cod in the North Sea are reported mature by Thorsen et al. [12]. The adult target body condition is taken as $q_a = 1.2$ based on a mean 24% somatic weight loss after spawning in Atlantic cod from the Gulf of St. Lawrence [13].

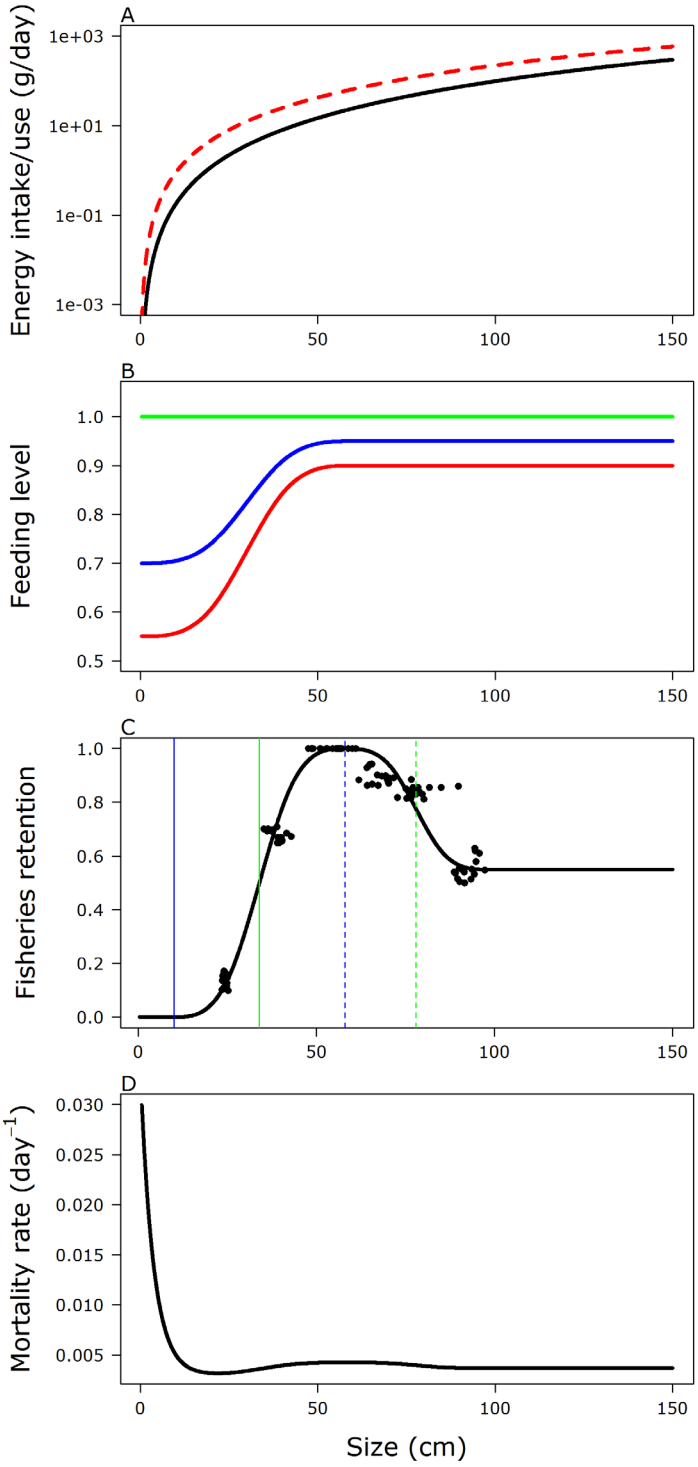

Figure S1 (A) Maintenance costs (black solid line) and maximum ingestion rate $\frac{1}{G}$ (red dashed line), (B) feeding level in case of high (green), intermediate (blue) and low (red) food availability, (C) fisheries retention data (black dots; 2000-2016, 65), the length at the start of the increase l_v (blue solid line), at 50% of the increase l_{vh} (green solid line) , at the start of the decline l_{vd} (blue dashed line) and 50% of the decline l_{vdh} (green dashed line) of fisheries retention and fisheries retention implemented in the model (black line) and (D) mortality rate as a function of body size implemented in the model.

Table S1 Potential consequences of anthropogenic noise for individual level processes. The observed effects of anthropogenic noise on fish (row names on the left) were chosen based on a meta-analysis of anthropogenic noise experiments with fish by Cox et al. [1]. We show an overview of potential consequences of the observed effects of anthropogenic noise for individual level processes (column names on top) that have been documented for fish. Colours of the cells indicate the quality of evidence (orange – documented consequences for individual level process; yellow – logical consequence but no consequences have been documented, or, only anecdotal studies of consequences for individual level process; blue – no short-term consequences for individual level process have been documented)

Observations \ Consequences	Energy expenditure	Food intake	Mortality	Reproductive output
Stress (Higher levels of cortisol)	✓ Elevated cortisol increases the metabolic rate [14].	✓ Elevated cortisol reduces food intake [15,16].	? Stress may lead to mortality [17].	✓ Elevated cortisol decreases fertilization rate [18] , and, leads to deformities in fish larvae [19] .
Foraging behaviour (Decrease of foraging behavior, such as: food consumption, foraging efficiency, and discrimination error of prey items.)	✓ More energy spent to come to the same food intake [20,21].	✓ Lower food intake due to less successful foraging or less time spent foraging [22].	× No direct consequences documented.	× No direct consequences documented.
Movement behaviour (Increase of movement related behaviour, such as: adjusted swimming depth, directional changes, schooling adjustments, swimming speed.)	✓ Schooling reduces the energetic costs of movement [23] and faster swimming costs more energy [24].	× No direct consequences documented.	× No direct consequences documented.	× No direct consequences documented.
Auditory system (Changes in the auditory system, such as the hearing threshold.)	× No direct consequences documented.	? Lower prey detection.	? Lower predator detection.	? Cod uses sounds for mate finding [25].

723 *Table S2 Model variables and default parameter values based on van Leeuwen et al. (2013).*

Symbol	Unit	Description	Value	Source	Derivation
Model variables					
a	[d]	Age			
x	[g]	Structural mass			
y_r	[g]	Reserves			
y_g	[g]	Gonads			
s	-	Survival probability			
R_0	-	Reproductive output			
Derived model variables					
l	[cm]	Length	$m = \gamma\, l^{\delta}$		
w	[g]	Total body mass	$w = x + y_r + y_g$		
m	[g]	Length-based mass	$m = x(1 + q_j)$		Structural mass and liver weight
Parameters					
Υ	[d]	Length of growing season	250	[3]	
τ_r	[d]	Day of spawning decision	200	[3]	
a_b	[d]	Age of first feeding	22	[3]	Atlantic cod, Norway
l_b	[cm]	Length at birth	0.39	[3]	Atlantic cod, Norway
l_s	[cm]	Characteristic length mortality	3.68	[3]	
l_m	[cm]	Maturation length	62	[12]	Length 50% mature (North Sea cod)
l_v	[cm]	Size of start fishing vulnerability	10	[9]	North Sea cod
l_{vh}	[cm]	Size of 50% fishing vulnerability	34	[9]	North Sea cod
l_{vd}	[cm]	Size of start decrease fisheries vulnerability	58	[9]	North Sea cod

l_{vdh}	[cm]	Size of %50 decrease fisheries vulnerability	78	[9]	North Sea cod
ρ	-		0.55	[9]	North Sea cod
q_j	-	Juvenile condition target	0.7	[3]	
q_a	-	Adult condition target	1.2	[13]	Atlantic cod, NW Atlantic
γ	$[(\mathrm{g\ cm^{-1}})^{\delta}]$	Length-weight scaling constant	0.01	[3]	Atlantic cod, NE Atlantic
δ	-	Length-weight scaling exponent	3.0	[3]	Atlantic cod, NE Atlantic
ε	$[\mathrm{d\ cm^{-\theta} g^{-1}}]$	Digestion time scaling constant	270.651	[5,8]	Derived from maximum growth rate, metabolic rate and $I(l)$
θ	-	Digestion time scaling exponent	-2.389	[5,8]	Derived from maximum growth rate, metabolic rate and $I(l)$
α	$[\mathrm{g^{1-\beta} d^{-1} g^{-1}}]$	Metabolic rate scaling constant	0.022	[5]	Derived from oxygen consumption of Atlantic cod at 8 C
β	-	Metabolic rate scaling exponent	0.9124	[5]	Derived from oxygen consumption of Atlantic cod at 8 C
σ_0	-	Conversion efficiency	0.6	[3]	Across fish species
σ_r	-	Gonad-offspring conversion efficiency	0.5	[3]	Female offspring only
μ_e	$[\mathrm{d^{-1}}]$	Egg mortality	0.03	[3]	
μ_s	$[\mathrm{d^{-1}}]$	Size-dependent mortality constant	0.03	[3]	
μ_0	$[\mathrm{d^{-1}}]$	Size-independent background mortality	0.003	[3]	

μ_v	[d^{-1}]	Fishing mortality	0.00124	[11]	North Sea cod fisheries, FMSY
F_{high}			1.0		
F_{int}			0.7 + 0.25 $X(l, 0.39, 30)$		Length dependent
F_{low}			0.55 + 0.35 $X(l, 0.39, 30)$		Length dependent
ψ_D		Acoustic mortality multiplier	0 - 10		
ψ_B		Acoustic reproductive failure multiplier	0 - 1		
ψ_T		Acoustic energy expenditure multiplier	0 - 1		
ψ_I		Acoustic feeding failure multiplier	0 - 1		

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