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Body downsizing caused by non-consumptive social stress severely depresses population growth rate

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Chronic social stress diverts energy away from growth, reproduction and immunity, and is thus a potential driver of population dynamics. However, the effects of social stress on demographic density dependence remain largely overlooked in ecological theory. Here we combine behavioural experiments, physiology and population modelling to show in a top predator (pike *Esox lucius*) that social stress alone may be a primary driver of demographic density dependence. Doubling pike density in experimental ponds under controlled prey availability did not significantly change prey intake by pike (i.e. did not significantly change interference or exploitative competition), but induced a neuroendocrine stress response reflecting a size-dependent dominance hierarchy, depressed pike energetic status and lowered pike body growth rate by 23 per cent. Assuming fixed size-dependent survival and fecundity functions parameterized for the Windermere (UK) pike population, stress-induced smaller body size shifts age-specific survival rates and lowers age-specific fecundity, which in Leslie matrices projects into reduced population rate of increase (λ) by 37–56%. Our models also predict that social stress flattens elasticity profiles of λ to age-specific survival and fecundity, thus making population persistence more dependent on old individuals. Our results suggest that accounting for non-consumptive social stress from competitors and predators is necessary to accurately understand, predict and manage food-web dynamics.

Keywords: community interactions; corticosteroids; hypothalamo–pituitary–adrenal axis;
Leslie matrix; thyroid hormones; trait-mediated interactions

1. INTRODUCTION

Ecological theory classically considers that the dynamics of closed populations are driven by trophic links such as competition and predation, i.e. by consumptive and lethal effects (Amarasekare 2002; Sibly & Hone 2002; Begon *et al.* 2006; Krebs 2009). However, there is currently a growing appreciation that competitors and predators may also negatively affect their prey through non-consumptive and non-lethal effects, which remain cryptic because they are in the *same direction* as consumptive and lethal effects of predation. Non-consumptive and non-lethal effects of predators may be mediated by plastic physiological, morphological and/or behavioural changes in the prey. For instance, recent studies have shown that predator behavioural avoidance, which exposes prey to less favourable habitats, may alter prey dynamics and have cascading effects on whole community functioning (Peacor & Werner 2001; Schmitz *et al.* 2004; Pangle *et al.* 2007; Orrock *et al.* 2008; Peckarsky *et al.* 2008; Preisser & Bolnick 2008). However, predation risk may incur costs to prey not only through habitat shifts, but also in terms of chronic physiological stress. For instance, reduced fecundity owing to chronic social stress from predation risk might be involved in driving the famous 10-year population cycles in Canadian snowshoe hares (*Lepus americanus*) (Boonstra *et al.* 1998). However, the contribution of social stress to population dynamics remains poorly quantified and generally overlooked in ecological studies. Additionally, non-consumptive and non-lethal effects on vital rates are rarely addressed in the context of intraspecific interactions. Here we quantify the effects of intraspecific social stress on population finite rate of increase (λ) in pike (*Esox lucius*), an aquatic

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top predator widely distributed across the Northern Hemisphere (Craig 1996).

The physiological response to stress has been extensively studied. In vertebrates, increased density (crowding or confinement) often favours the development of dominance hierarchies in which rank is influenced by a host of factors among which body size is often prevalent (Cloutier & Newberry 2000; French & Smith 2005). Behaviourally, dominance hierarchies are often set through aggressive interactions (Alexander & Roth 1971; Cloutier & Newberry 2000), but social rank can also establish without any direct conflict through visual exposure to opponents and predators (Barcellos *et al.* 2007; Grosenick *et al.* 2007; Höjesjö *et al.* 2007). Behavioural responses to social stress from dominant conspecifics and predators are mediated by an elevated release of serotonin in the brain, reduced brain cell proliferation and activation of the hypothalamo–pituitary–adrenal (HPA) axis (Summers 2002; Hoshaw *et al.* 2006; Sørensen *et al.* 2007). In the short term, activation of the HPA axis results in a rise in plasma glucocorticoids. In the longer term, stress depresses thyroid activity (Kühn *et al.* 1998; Walpita *et al.* 2007), increases standard metabolic rate, stimulates oxygen uptake and transfer, mobilizes energy substrates, lowers liver glycogen and reallocates energy away from immunity, growth and reproduction (Wendelaar-Bonga 1997). Hence, although stress increases vigilance towards enemies and heightens capacity to fight and flight or to cope with environmental change, in the long term, stress can decrease individual survival and fecundity and potentially impact on population dynamics.

We test this hypothesis in pike, which shows both individual and population responses to density in the wild (Haugen *et al.* 2006, 2007; Edeline *et al.* 2007, 2008; Winfield *et al.* 2008). We first experimentally quantify the effects of social stress alone on pike somatic growth rate by cancelling consumptive, food-related intraspecific density dependence (i.e. we increase pike density without altering interference or exploitative competition), and then we transpose laboratory results to field projections in matrix population models parametrized for pike in Windermere (UK, §2). Windermere pike vital rates are strongly size dependent (Carlson *et al.* 2007; Edeline *et al.* 2007; Haugen *et al.* 2007), and we thus base our demographic approach on the assumption that social stress influences survival and fecundity indirectly through its effect on somatic growth rate. This assumption is valid in a vast majority of ecosystems in which body size determines individual fitness and affects food-web structure and dynamics (Woodward *et al.* 2005). Our results suggest that demographic density dependence may be driven by social stress alone in the absence of any significant consumptive effect.

2. MATERIAL AND METHODS

(a) *Experimentally induced chronic social stress*
Pike of fork lengths (FLs) ranging from 306 to 534 mm (mean 400 ± 55 s.d.) and body mass (BM) ranging from 200 to 1206 g (mean 511 ± 235) were caught with gill nets in lake Årungen (approx. 50 km south of Oslo, Norway) just after the spawning season that starts at ice break (from 18 May to 7 June 2006, and from 23 April to 7 May 2007). In order to reduce gill-netting injuries, the nets

were lifted every 20–30 min. All pike were visually inspected for injuries, and only unharmed pike were transported in large water-filled plastic bags to the Norwegian Institute for Water Research's biological station at Solbergstrand located approximately 10 km from Årungen. Pike were anaesthetized with benzocain, measured for FL to the nearest millimetre, weighed for BM to the nearest gram and individually tagged with passive integrated transponders introduced into the body cavity. Tagged pike were then released in four circular (5 m diameter), indoor experimental ponds (water volume approx. 13 m^3 each). Pike captured on the same day were homogeneously distributed among ponds, and pike body size at the start of the experiments was not significantly different among ponds (BM: $p = 0.9781$, body length: $p = 0.9999$) or pike density treatment (BM: $p = 0.5012$, FL: $p = 0.7565$), but was higher in 2007 compared with 2006 (BM: $p < 0.0001$, FL: $p < 0.0001$) when tested in one single ANOVA ($n = 120$ observations). To limit stress from human exposure, each pond was surrounded by tarpaulins. A habitat structure (concrete blocks) provided in 2006 was not used by the fish and complicated prey count, and we therefore did not provide any habitat structure in 2007. Faeces and waste were regularly removed from the ponds using fine mesh nets. Each pond was supplied with oxygenated well water (mean temperature = $11.7°C \pm 1.0$) at a flow rate of 1 $m^3 h^{-1}$.

We experimentally induced chronic social stress in pike in 2006 (from 9 June to 10 August) and in 2007 (from 10 May to 12 September) by increasing pike density from 10 pike pond^{-1} (duplicate low-density treatment, 0.5 pike m^{-2}) to 20 pike pond^{-1} (duplicate high-density treatment, 1 pike m^{-2}). In the wild, numbers of pike per square metre vary widely from 0.0002 to 0.0117 in Windermere (age 2 pike and older; des Clers *et al.* 1994), 0.0006–0.0013 in Årungen (pike longer than 45 cm total body length (TL) only; Sharma & Borgstrøm 2008), from 2.8×10^{-5} to 3.8×10^{-4} in small Wisconsin lakes (Margenau *et al.* 1998) and from 3×10^{-5} to 5.9×10^{-4} in Minnesota lakes (Pierce & Tomcko 2003). However, population densities in key habitats are probably much higher than when the lake is considered as a whole. Hence, the two densities induced by our experimental setup (0.5 and 1 pike m^{-2}) may be considered to lie in the upper range of naturally occurring densities. In our experiment, ponds used for the low-density treatment in 2006 were used for the high-density treatment in 2007, and *vice versa*. No pike died during the 2006 experiment, while two pike died in 2007 (both at low density). Dead pike were measured to the nearest millimetre and immediately replaced (from a stock of extra pike kept in a fifth pond). Pike at the two densities were fed on average every 3.6 day (± 2.2 s.d.) with live prey (roach *Rutilus rutilus* and crucian carp *Carrassius carrassius*) caught in nearby lakes and delivered through pipes crossing the tarpaulins. We defined prey availability as the ratio of prey number to pike number observed in each pond on a given day. If measured on a day of feeding, prey availability was measured *before* feeding. We took care to maintain prey availability nonzero. Prey TLs ranged from 61 to 231 mm (mean 85.0 ± 18.5 s.d.) in roach and from 80 to 192 mm (mean 134.0 ± 19.5 s.d.) in crucian carp. Prey size was not significantly different among ponds ($p = 0.3352$) or density treatments ($p = 0.9789$), but was higher in 2007 compared with 2006 ($p < 0.0001$) in one single ANOVA ($n = 980$ observations). Indeed, in 2006, prey included small-sized roach, while in

2007 we used only crucian carp that were on average larger than roach. Total number of prey eaten by pike was 1294 at high pike density and 676 at low pike density in 2006, and 703 at high pike density and 397 at low pike density in 2007.

We varied within-pond prey availability across the experiments (from 0.2 to 4.7 prey pike^{-1}; low pike density: mean 1.59 ± 0.97 s.d.; high pike density: mean 1.51 ± 0.90 s.d.), taking care to maintain prey availability identically among ponds and pike density treatments in order to avoid differences in competition intensity. Square root-transformed prey availability was not significantly different among ponds ($p = 0.6208$) or density treatments ($p = 0.3219$) but was higher in 2006 compared with 2007 ($p < 0.001$, reflects smaller prey size in 2006) when tested in one single ANOVA ($n = 244$ observations). Our aim in varying prey density was to cover a wide range of the pike functional response, but the linear relationship between prey availability and pike feeding rate (Fr) (from model 1 in table 1, $\sqrt{N_{\text{prey eaten pike}^{-1}}} = 0.0535 \times \sqrt{N_{\text{prey available pike}^{-1}}} - 0.1044$) indicates that we did not saturate pike with prey.

(b) *Measurement of response to social stress*

We checked the effects of social stress by measuring pike behaviour, energetic condition, hormonal status and somatic growth. Internet cameras were set above each pond, allowing remote observations. Additionally, on 38 occasions in 2006 we video-recorded pike attacks on a prey during a period of 30 s to 5 min following prey addition. Pike are notoriously cannibalistic and males are territorial during the breeding season (Craig 1996), but we did not observe any direct agonistic behaviour or cannibalism (pike also had no physical injury). Finally, prey number in each pond was counted every second to seventh day, allowing us to calculate a pike Fr (in prey eaten per pike, $n = 244$ observations). At the end of each experiment, pike were sampled from their pond using hand nets and immediately killed with an overdose of metomidate (270 mg l^{-1}, cortisol-release inhibitor; Iversen *et al.* 2003) mixed with benzocain (40 mg l^{-1}, pain killer). Time used to sample all pike from one pond varied from 1 to 4 min (2 min 12 s on average), i.e. less than the putative delay necessary for onset of cortisol release in teleosts (5 min; Gamperl *et al.* 1994). Killed pike were measured for FL to the nearest millimetre, weighed to the nearest gram and dissected for determination of gender. Liver mass was weighed to the nearest 10^{-1} g. Hepatosomatic index (HSI) provided an indication of energetic status (glycogen storage, long-term stress effects), and was calculated as the ratio between measured and standard liver mass. We estimated standard liver mass using the linear regression of log-transformed liver mass on log-transformed BM.

Additionally, blood was sampled by caudal puncture, centrifuged and plasma was stored at a temperature of −80°C before hormonal measurements. Heparin was not efficient in preventing coagulation of pike blood, and we therefore used ethylene glycol tetraacetic acid powder as an anticoagulant. This did not interfere with hormonal measurements. Hormone analyses were performed at the Hormone Laboratory, Aker University Hospital, Oslo, Norway. Plasma cortisol is usually used as a stress indicator but because cortisol levels rapidly respond to handling (Gamperl *et al.* 1994) we also measured plasma concentrations of thyroxine (T$_4$), and triiodothyronine (T$_3$) as well as the T$_3$/T$_4$ ratio (Edeline *et al.* 2004) as endocrine markers of long-term physiological stress (§1). Total plasma cortisol (in nmol l^{-1}) was

assayed by radioimmunoassay following the manufacturer's recommendations (Siemens Healthcare Diagnostics, Los Angeles, CA, USA). Free circulating T$_3$ (in nmol l^{-1}) and total circulating T$_4$ (in pmol l^{-1}) were assayed by competitive fluoroimmunoassays, again following the manufacturer's instructions (Delfia, Perkin Elmer Life Sciences, Turku, Finland). All assays were tested and validated for pike plasma before samples were analysed.

(c) *Modelling response to social stress*

We explored the effect of social stress on pike using statistical models for which detailed structures are provided in table 1. However, before proceeding to the analysis of individual responses, we performed a preliminary multivariate analysis of variance (MANOVA) approach that controls for possible type-1 statistical errors owing to multiple tests on non-independent response variables. In one MANOVA, we grouped responses and predictors from models 1 and 2 in table 1 (behavioural responses measured at the pond level), while in another MANOVA we grouped responses and predictors for models 3–9 in table 1 (responses measured at the individual level). Both MANOVAs indicated a highly significant effect of pike density ($p = 0.0036$ on pond-level behavioural responses and $p = 0.0002$ on individual responses). We detected a significant pond effect on pike behaviour, somatic growth, HSI, plasma cortisol, plasma T$_4$ and plasma T$_3$/T$_4$, and we thus analysed these responses with restricted maximum-likelihood (REML) mixed-effects models with pond as the grouping factor (table 1). To model Fr, somatic growth, HSI, plasma cortisol, plasma T$_4$ and plasma T$_3$/T$_4$, we used linear REML mixed models in the nlme library of R (Pinheiro & Bates 2000; R Development Core Team 2008). To model pike behaviour (probability for observing a video-recorded attack by a pike on a prey during prey addition), we used a binomial (logit link) generalized mixed model fitted by REML and Laplace approximation in the lme4 library of R (Bates 2005). Finally, there was no significant pond effect on plasma T$_3$, which was more efficiently modelled with a standard linear model than with a mixed model (comparison of Akaike's information criterions (AICs) based on ML parameter estimation; Pinheiro & Bates 2000).

(d) *Demographic cost of social stress*

We computed effect size of social stress on pike somatic growth using predictions from model 3 in table 1 for averaged covariates (i.e. adjusted mean density effect). Predicted length increase is 0.30 mm d^{-1} at low density and 0.23 mm d^{-1} at high density, yielding a 23 per cent difference in somatic growth rate. We used this effect size of social stress on length increase to project the demographic costs of social stress in Windermere pike, for which survival and fecundity are length dependent (Carlson *et al.* 2007; Haugen *et al.* 2007). Windermere is a glacial valley lake of the English Lake District in which pike have been sampled each year since 1944 as part of a long-term scientific monitoring programme (Le Cren 2001; Winfield *et al.* 2008). A spring (March–April) component of this sampling was designed to capture a large size range of pike, which were all measured for TL (to the nearest centimetre), tagged and released. Resulting capture–mark–recapture data have been extensively described in two recent papers (Haugen *et al.* 2006, 2007). Briefly, an individual pike tagged in spring of year t was considered to have survived through

Table 1. The effect of social stress (density) on pike behaviour and physiology (BM: body mass, D: density, Exp: experiment, FL: fork length, Fr: feeding rate, HSI: hepatosomatic index; selection of model structure based on AIC).

model number	response	random grouping factor	random effect	variance–covariance matrix for random effects	heteroskedastic structure	fixed effects	estimate	s.e. of the estimate	p-value[a]
1	$\sqrt{Fr}$ (n = 244 observations)[c]	pond (n = 4 levels)	$\sqrt{\text{prey availability}}$	symmetric	product of an Exp-specific power function of time and different variance levels for each D	Exp (2007 versus 2006)	−0.278	0.042	<0.0001
						time interval (days)	0.053	0.005	<0.0001
						$\sqrt{\text{prey availability}}$[b]	0.809	0.035	<0.0001
						pike D (L versus H)	0.022	0.039	0.5613
2	probability of an attack on a prey (n = 38 observations)	pond (n = 4 levels)	intercept	symmetric	none	pike D (L versus H)	−2.649	0.888	0.0029
3	log(growth in FL) (n = 109 observations)	pond (n = 4 levels)	FL at start	symmetric	product of different variance levels for each Exp and D	Exp (2007 versus 2006)	−0.344	0.126	<0.0001
						FL at start (mm)	−0.008	0.001	<0.0001
						gender (male versus female)	−0.208	0.095	0.0340
4	growth in BM (n = 119 observations)	pond (n = 4 levels)	intercept	symmetric	product of different variance levels for each Exp and D	pike D (L versus H)	0.265	0.098	0.0079
						Exp (2007 versus 2006)	−0.772	0.110	<0.0001
5	HSI (n = 120 observations)	pond (n = 4 levels)	intercept	symmetric	none	pike D (L versus H)	0.418	0.115	0.0004
						Exp (2007 versus 2006)	10.335	2.317	<0.0001
6	log(plasma cortisol) (n = 98 observations)	pond (n = 4 levels)	pike D	diagonal	product of different variance levels for each Exp and D	pike D (L versus H)	5.605	2.344	0.0184
						Exp (2007 versus 2006)	−0.896	0.336	0.0038
7	log(plasma T4) (n = 98 observations)	pond (n = 4 levels)	intercept	diagonal	product of different variance levels for each Exp and sex	log(FL at end (mm))	−0.754	0.368	0.0422
						pike D (L versus H)	−0.250	0.345	0.4699
						log(FL at end (mm))	1.743	0.365	<0.0001
						pike D (L versus H)	0.380	0.094	0.0001
8	plasma T3 (n = 98 observations)	pond (n = 4 levels)	no random effect	diagonal	none	Exp (2007 versus 2006)	−0.682	0.145	0.0059
						log(FL at end (mm))	2.996	0.600	<0.0001
						pike D (L versus H)	0.325	0.125	0.0112
9	log(plasma T3/T4 ratio) (n = 98 observations)	pond (n = 4 levels)	intercept	diagonal	product of different variance levels for each D and sex	Exp (2007 versus 2006)	−0.160	0.112	0.0194
						log(FL at end (mm))	−0.946	0.436	0.0117
						pike D (L versus H)	−0.288	0.097	0.0037

[a]Sequential decomposition of the contributions of fixed-effects terms.
[b]i.e. Number of prey pike^{-1} at previous observation.
[c]This model also incorporated within-pond temporal autocorrelation (autoregressive function of time of order 1 and moving average of time of order 2).
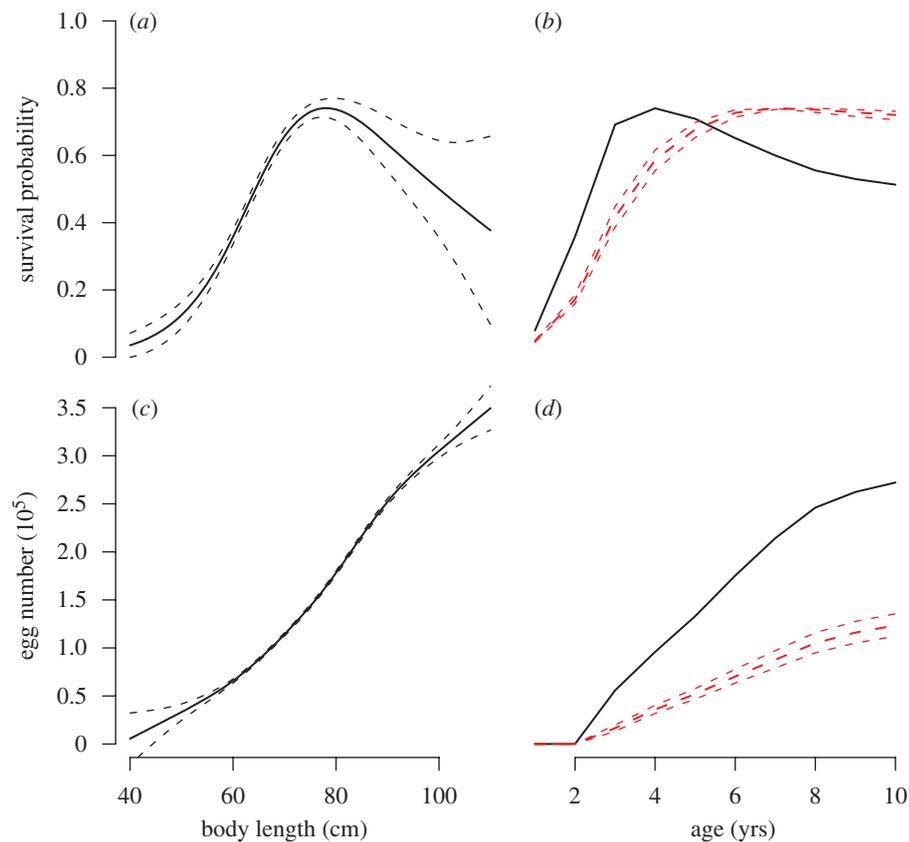
Figure 1. Projected effect of body downsizing caused by social stress on age-dependent vital rates of Windermere pike. (a) Windermere pike survival probability as a function of body length with 95% confidence limits. (b) Corresponding survival probability as a function of pike age without (black solid line, control) and with (red dashed line) the added effect of social stress on pike somatic growth rate (thin red dashed lines represent 95% confidence limits for the effect of social stress on pike somatic growth). (c) Windermere female pike fecundity as a function of body length with 95% confidence limits. (d) Corresponding fecundity as a function of pike age without (black solid line, control) and with (red dashed line) the added effect of social stress on somatic growth rate (first reproduction occurs at age 3; thin red dashed lines represent 95% confidence limits for the effect of social stress on pike somatic growth).

the summer of year t (survival = 1) if recaptured at any point in time after the summer of year t (Carlson *et al.* 2007). By contrast, a fish that was never recaptured after the summer of year t was attributed a survival of 0 for this summer. We built a size-dependent nonlinear survival function $s(BL)$ with a natural cubic spline in a binomial (logit link) additive model (figure 1a, GAM function, mgcv library of the R software (Wood 2006; R Development Core Team 2008); $n = 5065$ including male, female and immature fish for a wide size range and a robust estimation; edf = 4.078; $\chi^2 = 528.1$; $p < 0.0001$).

As part of the scientific programme, pike are also sampled in winter (October–February) using gill nets (64 mm mesh size) that target pike longer than 54 cm (Frost & Kipling 1967). All pike captured this way are killed, sexed, measured for TL (to the nearest centimetre) and back-calculated for length-at-age using opercular bones (Frost & Kipling 1959). Additionally, female fecundity (total number of eggs) has been estimated since 1963 (Kipling & Frost 1969). We used the fecundity data to obtain a size-dependent fecundity function $F(BL)$ with a natural cubic spline in a GAM (figure 1c; $n = 3695$ females; edf = 5.915; $F = 2339$; $p < 0.0001$). We equated age-specific survival and fecundity as $s_a = s(BL_a)$ and $F_a = F(BL_a)$, respectively (black solid lines in figure 1b,d), where BL_a is the mean female body length at age a in the back-calculated length-at-age

dataset. We quantified the effect of added social stress on Windermere pike survival and fecundity (red dashed lines in figure 1b,d) by reducing BL_a by 23 per cent so that $s_a = s(0.77 * BL_a)$ and $F_a = F(0.77 * BL_a)$. We calculated 95% confidence limits for this effect as $s_a = s[(0.77 \pm 1.96$ s.e.$) * BL_a]$ and $F_a = F[(0.77 \pm 1.96$ s.e.$) * BL_a]$, where s.e. is the standard error of the predicted effect size of stress on pike somatic growth.

Resultant age- and density-specific survival and fecundity estimates $s_{a,D}$ and $F_{a,D}$ provided entries in Leslie matrices of the following form (Caswell 2001):

$$M = \begin{bmatrix} 0 & 0 & \sigma s_{2,D} F_{3,D} & \cdots & \cdots & \sigma s_{a-1,D} F_{a,D} \\ 6.56 \times 10^{-4} & 0 & 0 & \cdots & \cdots & 0 \\ 0 & s_{1,D} & 0 & \cdots & \cdots & \vdots \\ 0 & 0 & s_{2,D} & \cdots & \cdots & \vdots \\ \vdots & \vdots & \vdots & \ddots & \vdots & \vdots \\ 0 & 0 & 0 & \cdots & s_{a-2,D} & 0 \end{bmatrix}$$

where σ is the proportion of females in a clutch (0.5) and maturity occurs at age 3 (i.e. $F = 0$ for $a < 3$). Survival from the egg to age 1 was taken equal to 6.56×10^{-4}, which is the average of literature values (Wright 1990; Massé *et al.* 1993; Minns *et al.* 1996; Farell 2001). We

estimated the demographic cost of social stress by computing population finite rate of increase (λ; $N_{t+1} = \lambda N_t$) as the dominant eigenvalue of M for maximum a ranging from 3 to 10 under both control conditions (i.e. Windermere conditions) and added social stress conditions. Finally, we computed elasticities of λ to age-specific survival and fecundity as

$$\frac{x \partial \lambda}{\lambda \partial x} = \frac{x}{\lambda} \sum_{i,j} \frac{\partial \lambda}{\partial m_{ij}} \frac{\partial m_{ij}}{\partial x},$$

where parameter x is age-specific survival or fecundity, and m_{ij} is matrix M entry in row i and column j for $a = 10$ (Caswell 2001). Computations were performed using SCILAB software (Digiteo, http://www.scilab.org/). For the effect of social stress on elasticities, 95% confidence limits were obtained from matrices M, including upper and lower 95% confidence limits of s_a and F_a.

3. RESULTS

(a) *Pike response to social stress*

Endocrine measures generally supported the occurrence of social stress at an increased pike density. Plasma hormone levels were as follows: 34.5 ± 43.8 (s.d.) nmol l^{-1} cortisol, 16.2 ± 8.7 pmol l^{-1} T_4 and 3.5 ± 0.7 nmol l^{-1} T_3 at high density, and 24.9 ± 37.7 nmol l^{-1} cortisol, 22.8 ± 11.8 pmol l^{-1} T_4 and 3.9 ± 0.6 nmol l^{-1} T_3 at low density. Possibly owing to a very large individual variability, the effect of increased pike density on pike circulating cortisol levels was not statistically significant (model 6, table 1). However, increased pike density significantly decreased pike thyroid activity (both plasma T_4 and T_3, models 7 and 8 in table 1) and significantly increased the pike T_3/T_4 ratio (model 9, table 1), indicating a physiological stress. Interestingly, pike physiological stress increased with a smaller body size (models 6–9 in table 1), suggesting a size-dependent dominance hierarchy.

Pike mean Fr was 0.43 ± 0.57 prey pike^{-1} d^{-1} at high density, and 0.45 ± 0.55 prey pike^{-1} d^{-1} at low density. Pike Fr was stimulated by an increase in the number of prey available, but was not significantly affected by pike density, indicating that no significant difference in interference or exploitative competition occurred among density treatments (model 1, table 1). Note that a Time $*$ Density interaction was not significant in model 1 ($p = 0.1511$) and increased model AIC from -62.5 to -46.8, further indicating that pike consumed prey at the same rate in the two density treatments. The probability of observing an attack by a pike on a prey during prey addition into the ponds increased with pike density (model 2, table 1).

Finally, pike mean somatic growth was 0.32 ± 0.28 mm d^{-1} and 1.41 ± 0.68 g d^{-1} at high density and 0.35 ± 0.25 mm d^{-1} and 1.85 ± 0.76 g d^{-1} at low density (see also predicted somatic growth in §2). Increased pike density significantly depressed individual pike somatic growth in both length (model 3, table 1) and mass (model 4, table 1), indicating a depressed energetic status despite a similar food intake. Increased pike density significantly decreased pike HSI (model 5, table 1), further supporting the view that social stress depressed pike energetic status.
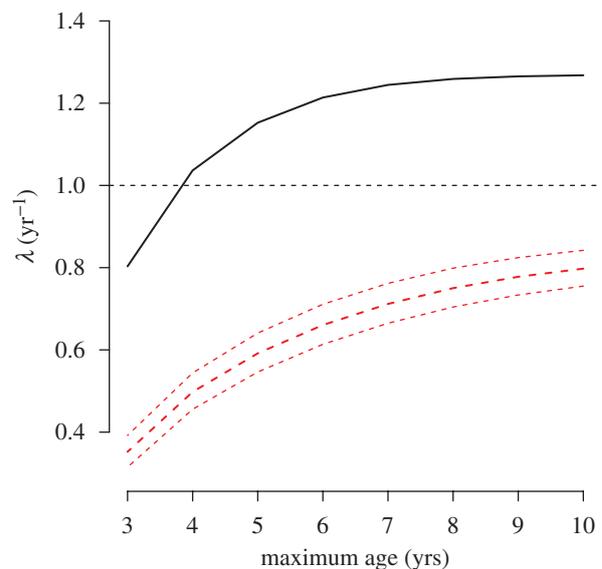
Figure 2. The demographic cost of social stress in pike. Projected effect of experimentally measured somatic growth reduction from social stress on Windermere pike population rate of increase (λ), estimated for different maximum ages in the population (starting from female first reproduction at age 3; thin red dashed lines represent 95% confidence limits for the effect of social stress on pike somatic growth). Horizontal, black dashed line at $\lambda = 1$ represents the limit at which population size is temporally stable ($N_{t+1} = N_t$). Black solid line, control.

(b) *Demographic cost of social stress in pike*

Decreased somatic growth from social stress shifts age-specific survival rightward (figure 1*b*), and age-specific fecundity downward (figure 1*d*). In Leslie matrices, these stress-induced changes in pike vital rates result in a $37–56\%$ decrease in λ for maximum ages in the population decreasing from 10 to 3 (figure 2). The average age of mature female pike in Windermere is 5 years, an age at which somatic growth depression from social stress reduces λ by 49 per cent. Somatic growth reduction from social stress shifts elasticities of λ to age-specific survival and fecundities towards older age classes (figure 3), i.e. under social stress, fitness is less dependent on early vital rates and more dependent on late vital rates. However, social stress does not change survival to maturity as being the most critical fitness component (figure 3).

4. DISCUSSION

In this study, we couple behaviour, physiology and mathematical modelling to reconstruct fully the whole suite of biological mechanisms underlying the effects of social stress on population dynamics. We demonstrate that cues from conspecifics can induce a neuroendocrine stress response reflecting a size-dependent dominance hierarchy, depress individual energetic status and somatic growth, and that the resultant downsizing projects into severely lowered population rate of increase under size-dependent survival and fecundity. Hence, we predict that social stress can alone drive demographic density dependence without any significant change in the consumptive effects of interference or exploitative competition.
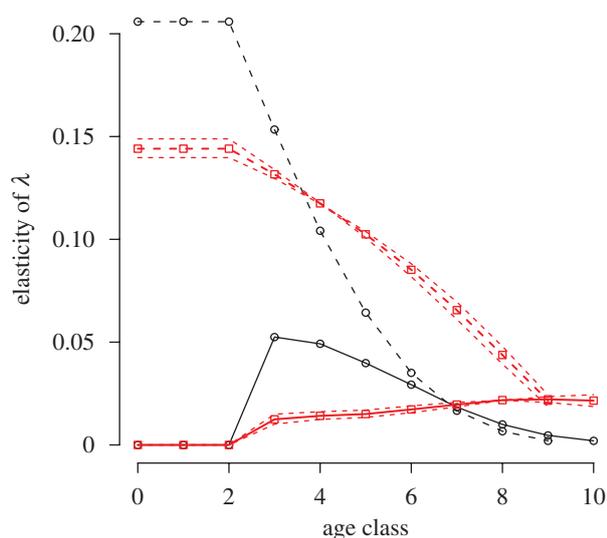
Figure 3. Effect of social stress on population response to perturbation. The elasticity of population rate of increase (λ) with respect to changes in age-specific survival and fecundity without and with the added effect of social stress on somatic growth rate (thin red dashed lines represent 95% confidence limits for the effect of social stress on pike somatic growth). Black dashed line, survival, control; red dashed line, survival, social stress; black solid line, fecundity, control; red solid line, fecundity, social stress.

(a) Social stress

Social stress is underlain by a cascade of neuroendocrinological, physiological and behavioural mechanisms. Activation of the HPA axis in the presence of potential competitors and predators is mediated by visual, auditive, chemical and other types of cues. In our experiment, we did not observe any direct agonistic interaction or physical injury on pike, but pike physiological stress was negatively linked to individual body size, suggesting that individuals were able to assess risk from a distance. This result is in line with data in various taxa of vertebrates showing that social rank in dominance hierarchies increases with body size (Cloutier & Newberry 2000; French & Smith 2005), and that visual cues alone are enough to induce social stress and set dominance hierarchies (Barcellos *et al.* 2007; Grosenick *et al.* 2007; Höjesjö *et al.* 2007). Our results are also consistent with results in the wild, showing that individual pike spatially avoid their larger conspecifics (Nilsson 2006). Indeed, pike are strongly cannibalistic (Craig 1996), and larger conspecifics do not only represent potential competitors for pike but also potential predators.

The effects of social stress on individual physiology are remarkably consistent among vertebrates. Stress-associated rise in plasma glucocorticoids depresses thyroid activity (Kühn *et al.* 1998; Walpita *et al.* 2007), increases standard metabolic rate and reallocates energy away from immunity, growth and reproduction (Wendelaar-Bonga 1997). In our experiment, depressed pike thyroid status (lower T_4 and T_3, elevated T_3/T_4 ratio), liver mass and somatic growth rate (revealing activated gluconeogenesis and resting metabolism) are typical of an activation of the HPA axis. However, we did not observe any significant effect of pike density on pike plasma cortisol level, probably because individual variability far exceeded mean differences. Although we followed standard

sampling procedures (Gamperl *et al.* 1994), it cannot be excluded that pike plasma cortisol levels were affected by this process. In particular, sampling might have increased cortisol levels more at low than at high density because chronically stressed animals show a less rapid onset of cortisol response to acute stressors (Summers 2002).

At the behavioural level, an activation of the HPA axis is often associated with increased locomotor activity. In brook trout (*Salvelinus fontinalis*) held in lake enclosures, doubling the density (from four to eight fish per 8 m^3 enclosure) reduced somatic growth by 50 per cent without any change in food consumption (Marchand & Boisclair 1998). This effect was attributed to increased locomotor activity (swimming and aggression). In our experiment, pike were highly static, except during attacks on prey or when we were cleaning ponds of faeces. Weak swimming activity by pike was to be expected because pike are ambush hunters (Craig 1996). Also, the absence of pronounced swimming by pike might reflect conflicting metabolic demands between digestion and movement since individuals were fed at a high rate. However, we detected that attack rate during prey addition increased at an increased pike density, a result that might reflect an increased locomotor activity.

(b) Demographic cost of stress

Our results predict that social stress can be a primary driver of pike population dynamics. Was the strength of pike response to stress of a reasonable magnitude in our experiment? We believe that we might have overestimated baseline stress compared with natural conditions, but not stress increase from increased density (i.e. not the quantity of interest here). Indeed, baseline stress was probably higher than in the wild because we employed densities that were in the upper range of naturally occurring densities (§2), we maintained pike for months at these densities (in the wild, subordinates may disperse to lower quality patches), and we did not provide any suitable habitat structure in which individuals could hide from each other. However, a high baseline stress more likely dampened than amplified stress increase from increased pike density. Indeed, if we reasonably assume that the relationship between density and social stress is asymptotic, a large density increase at high densities (and at high baseline stress, as in our experiment) is likely to induce less stress increase than a smaller density increase at lower densities (and at lower baseline stress, as in the wild).

How reliable is our estimate of the demographic cost of social stress in pike? A full account of the effects of social stress on pike vital rates would have probably yielded a stronger decrease in λ than estimated here because (i) Windermere data taken as control in fact already include naturally occurring social stress, (ii) we did not evaluate the direct effects of social stress on survival (e.g. immunity depression) and fecundity, and (iii) we did not assume any social stress effect for survival to age 1. Therefore, we predict that somatic growth decrease from non-consumptive social stress can have a *very* strong effect on population dynamics. This prediction should now be tested by directly measuring population dynamics response to stress. Our models also suggest that

downsizing from social stress flattens the elasticity profiles of λ to age-specific survival and fecundity, therefore making population persistence more dependent on old individuals. The impact of social stress on population dynamics and its ramifications for ecosystem-level processes are likely to vary according to species' position in the food web. In key species like top predators, the effects of social stress are likely to propagate down in the food web and influence whole ecosystem functioning. At lower trophic levels, social stress might have less influence on the food web, but a multiplicity of competitors and predators may increase stress intensity. Hence, further studies are needed to characterize social stress and its demographic cost at different trophic levels, and to ultimately quantify the contribution of stress to whole community dynamics.

(c) *Conclusion and implications*

Stress allows vertebrates to cope with perturbations and recover homeostasis at the expense of suppressing non-immediate essential activities. In the long term, stress diverts energy away from immunity, somatic growth and reproduction. Our results predict that somatic growth reduction from chronic social stress can, alone, severely decrease population rate of increase when survival and fecundity are size dependent. We also predict that downsizing from social stress flattens the elasticity profile of λ to age-specific survival and fecundity, i.e. possibly makes population persistence more contingent on environmental fluctuation as it depends more on old individuals. Ecosystems are replete with stressful social interactions that can depress body size, which is itself strongly linked to individual fitness in a vast majority of ecosystems. Therefore, we suggest that our results are of a general ecological value, and that a better integration of the effects of social stress into theory would enrich our understanding of population and community dynamics. For instance, the existence and strength of particular trophic links are often assessed using observational data on changes in population and species abundance through time. Such studies may overestimate the strength, rate or scale of trophic links if they do not account for the non-consumptive effects of social stress. Invasive or introduced competitors and predators, or other sources of disturbance (e.g. human activities, habitat loss, etc.) can also be more harmful to local populations than initially expected if they increase social stress. We therefore suggest that management and conservation plans should routinely integrate a quantitative evaluation of the effects of social stress.

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