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# Downsizing caused by non-consumptive social stress severely depresses population growth rate

**Running title: Physiology and demography of social stress**

Eric Edeline[1,2]*, Thrond O. Haugen[2,3], Finn-Arne Weltzien[4,5], David Claessen[6], Ian J. Winfield[7], Nils Chr. Stenseth[2], L. Asbjørn Vøllestad[2]

**Addresses:** [1] UPMC-Paris 6, UMR (CNRS) 7618, Laboratoire Biogéochimie et Ecologie des Milieux Continentaux, Ecole Normale Supérieure, 46 rue d'Ulm, F-75230 Paris CEDEX 05, France. [2] Centre for Ecological and Evolutionary Synthesis (CEES), Dept of Biology, University of Oslo, P.O. Box 1066 Blindern,N-0316 Oslo, Norway. [3] Norwegian Institute for Water Research (NIVA), Gaustadalléen 21, 0349 Oslo, Norway. [4] Department of Molecular Biosciences, University of Oslo, PB 1041 Blindern, NO-0316 Oslo, Norway. [5] Department of Basic Sciences and Aquatic Medicine, Norwegian School of Veterinary Science, PO Box 8146 Dep, NO-0033 Oslo, Norway. [6] Ecole Normale Supérieure, CERES-ERTI and UMR 7625, 24 Rue Lhomond, F-75230 Paris cedex 05, France. [7] Centre for Ecology & Hydrology, Lancaster Environment Centre, Library Avenue, Bailrigg, Lancaster, Lancashire LA1 4AP, UK.

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* To whom correspondence should be sent: Eric Edeline, UPMC-Paris 6, UMR (CNRS) 7618, Laboratoire Biogéochimie et Ecologie des Milieux Continentaux, Ecole Normale Supérieure, 46 rue d'Ulm, F-75230 Paris CEDEX 05, France. Telephone: +33 (0)1 44 32 38 84. Fax: +33 (0)1 44 32 38 85. E-mail: edeline@biologie.ens.fr.

ABSTRACT

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%. Assuming fixed size-dependent survival and fecundity functions parameterized for the Windermere (UK) pike population, stress-induced downsizing shifts age-specific survival rates and lowers age-specific fecundity, which in Leslie matrices reduces population rate of increase ($\lambda$) by 37 to 56%. Social stress also makes population persistence more dependent on old individuals by flattening elasticity profiles of $\lambda$ to age-specific survival and fecundity. Our results support the view that accounting for non-consumptive social stress from competitors and predators is necessary to accurately understand, predict and manage food-web dynamics.

**Key words**: Community interactions, Corticosteroids, Hypothalamo-pituitary-adrenal axis, Leslie matrix, Thyroid hormones, Trait-mediated interactions.

## 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 (Sibly & Hone 2002; Amarasekare 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 nonlethal effects of predators may be mediated by plastic physiological, morphological and/or behavioral 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; Preisser & Bolnick 2008; Peckarsky *et al.* 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 due to chronic social stress from predation risk might be involved in driving the famous 10-yr 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 ($\lambda$) in pike (*Esox lucius*), an aquatic 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 (Höjesjö *et al.* 2007; Grosenick *et al.* 2007; Barcellos *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). On the short term, activation of the HPA-axis results in a rise in plasma glucocorticoids. On the longer-term, stress depresses thyroid activity (Kühn *et al.* 1998; Walpita *et al.* 2007), increases standard metabolic rate, stimulates oxygen uptake and transfer, mobilises 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; Edeline *et al.* 2007; Haugen *et al.* 2007; Edeline *et al.* 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 parametrised for this species in Windermere (UK, see Materials and Methods). Windermere pike vital rates are strongly size-dependent (Haugen *et al.* 2007; Carlson *et al.* 2007; Edeline *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). We show that demographic density dependence may be driven by social stress alone in the absence of any significant consumptive effect.

## MATERIALS AND METHODS

### Experimentally-induced chronic social stress

Pike of fork lengths (FL) ranging from 306 to 534 mm (mean 400 ± 55 SD) and body mass (BM) ranging from 200 to 1206 g (mean 511 ± 235) were caught with gill nets in the lake Årungen (~ 50 km south of Oslo, Norway) just after the spawning season that starts at ice break (from May 18 to June 7 2006, and from April 23 to May 7 2007). In order to reduce gill-netting injuries, the nets were lifted every 20–30 minutes. 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 ~ 10 km from Årungen. Pike were anesthetized with benzocain, measured for FL to the nearest mm, weighed for BM to the nearest g, and individually tagged with passive integrated transponders introduced into the body cavity. Tagged pike were then released in 4

circular (5 m diameter), indoor experimental ponds (water volume ≈ 13 $m^3$ each). Pike captured on the same day were homogeneously distributed among ponds, and pike body size at start of the experiments was not significantly different among ponds (body mass: $p = 0.9781$, body length: $p = 0.9999$) or pike density treatment (body mass: $p = 0.5012$, body length: $p = 0.7565$), but was higher in 2007 compared to 2006 (body mass: $p < 0.0001$, body length: $p < 0.0001$) 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. Feces 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 ± 1.0) at a flow rate of 1 $m^3\ h^{-1}$.

We experimentally induced chronic social stress in pike in 2006 (from June 9 to August 10) and in 2007 (from May 10 to September 12) 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 $m^{-2}$ vary widely from 0.0002 to 0.0117 in Windermere (age-2 pike and older (des Clers *et al.* 1994)), 0.0006 to 0.0013 in Årungen (pike longer than 45 cm 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 mm and immediately replaced (from a stock of extra pike kept in a 5th pond). Pike at the two densities were fed on average every 3.6 day (± 2.2 SD) with live prey (roach *Rutilus rutilus* and crucian carp *Carrassius carrasius*) caught in nearby lakes and delivered through pipes crossing the tarpaulins (prey availability was never 0). Prey total lengths ranged from 61 to 231 mm (mean 85.0 ± 18.5 SD) in roach and from 80 to 192 mm (mean 134.0 ± 19.5 SD) 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 to 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 carps 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 2006.

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 SD; high pike density: mean 1.51 ± 0.90 SD), taking care to maintain prey availability identical 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 to 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 (from model 1 in Table 1, $\sqrt{N_{prey\,eaten\,pike^{-1}}} = 0.0535 \times \sqrt{N_{prey\,available\,pike^{-1}}} - 0.1044$ ) indicates that we did not saturate pike with prey.

**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 us to observe pike daily from an office place. Additionally, on 38 occasions in 2006 we video-recorded pike attacks on a prey during a period of 30 sec 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 2$^{nd}$ to 7$^{th}$ day, allowing us to calculate a pike feeding rate *Fr* (in prey eaten pike$^{-1}$, 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 sec 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 mm, weighed to the nearest g, 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 at preventing coagulation of pike blood and we therefore used EGTA (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 (see introduction). Total plasma cortisol (in nmol $L^{-1}$) was assayed by radioimmunoassay following the manufacturer's recommendations (Siemens Healthcare Diagnostics, Los Angeles, CA). 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 analyzed.

**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 MANOVA approach (Multivariate Analysis of Variance) that controls for possible type-1 statistical errors due 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 analyzed these responses with

restricted maximum likelihood (REML) mixed-effects models with pond as the grouping factor (Table 1). To model feeding rate, 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)).

**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 $day^{-1}$ at low density and 0.23 mm $day^{-1}$ at high density, yielding a 23% 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 (Haugen *et al.* 2007; Carlson *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 program (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 total body length (TL, to the nearest cm), tagged and released. Resulting capture-mark-recapture (CMR) data have been extensively

described in two recent papers (Haugen *et al.* 2006; Haugen *et al.* 2007). Briefly, an individual pike tagged in spring of year *t* was considered to have survived through the summer of year *t* (survival = 1) if recaptured at any point in time after the summer of year *t* (Carlson *et al.* 2007). In 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 (Fig. 1A, GAM function, mgcv library of the R software (Wood 2006; R Development Core Team 2008); n = 5,065 including male, female and immature fish for a wide size range and a robust estimation; edf = 4.078; $Chi^2$ value = 528.1; $p < 0.0001$].

As part of the scientific program, pike are also sampled in winter (October-February) using gill-nets (64 mm mesh size) which target pike longer than 54 cm (Frost & Kipling 1967). All pike captured this way are killed, sexed, measured for TL (to the nearest cm), 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 (Fig. 1C; n = 3,695 females; edf = 5.915; F value = 2,339; $p < 0.0001$). We equated age-specific survival and fecundity as $s_a = s(BL_a)$ and $F_a = F(BL_a)$, respectively (black solid lines in Figs. 1B and 1D), where $BL_a$ is 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 Figs. 1B and 1D) by reducing $BL_a$ by 23 % 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 $s_a = s[(0.77 \pm 1.96\ SE)*BL_a]$ and $F_a = F[(0.77 \pm 1.96\ SE)*BL_a]$, where SE 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):

$$M = \begin{bmatrix} 0 & 0 & \sigma s_{2,D} F_{3,D} & \cdots & \cdots & \sigma s_{a-1,D} F_{a,D} \\ 6.56\times10^{-4} & 0 & 0 & \cdots & \cdots & 0 \\ 0 & s_{1,D} & 0 & \cdots & \cdots & \vdots \\ 0 & 0 & s_{2,D} & \cdots & \cdots & \vdots \\ \vdots & \vdots & \vdots & \ddots & \vdots & \vdots \\ 0 & 0 & 0 & \cdots & s_{a-2,D} & 0 \end{bmatrix}$$

where $\sigma$ 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; Masse *et al.* 1993; Minns *et al.* 1996; Farell 2001). We estimated the demographic cost of social stress by computing population finite rate of increase ($\lambda$; $N_{t+1} = \lambda N_t$) 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. We calculated 95% confidence limits for the effect of social stress on $\lambda$ by using upper and lower 95% confidence limits of $s_a$ and $F_a$ in $M$. Finally, we computed elasticities of $\lambda$ to age-specific survival and fecundity as:

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

where parameter $x$ is age-specific survival or fecundity, and $m_{ij}$ is matrix $M$ entry in row $i$ and column $j$ (Caswell 2001). Computations were performed using SCILAB software (Digiteo,

http://www.scilab.org/). 95% confidence limits for the effect of social stress on elasticities were obtained from matrices $M$ including upper and lower 95% confidence limits of $s_a$ and $F_a$.

## RESULTS

### 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 (SD) 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 due 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 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 feeding rate was 0.43 ± 0.57 prey $pike^{-1}$ $day^{-1}$ at high density, and 0.45 ± 0.55 prey $pike^{-1}$ $day^{-1}$ at low density. Pike feeding rate 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 $day^{-1}$ and 1.41 ± 0.68 g $day^{-1}$ at high density, and 0.35 ± 0.25 mm $day^{-1}$ and 1.85 ± 0.76 g $day^{-1}$ at low density (see also predicted somatic growth in Materials and Methods). 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 hepatosomatic index (model 5, Table 1), further supporting the view that social stress depressed pike energetic status.

**Demographic cost of social stress in pike**

Decreased somatic growth from social stress shifts age-specific survival rightward (Fig. 1B), and age-specific fecundity downward (Fig. 1D). These stress-induced changes in pike vital rates result in a 37 to 56 % decrease in $\lambda$ for maximum ages in the population decreasing from 10 to 3 (Fig. 2). Average age of mature female pike in Windermere is 5 years old, an age at which somatic growth depression from social stress reduces $\lambda$ by 49 %. Somatic growth reduction from social stress shifts elasticities of $\lambda$ to age-specific survival and fecundities towards older age classes (Fig.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 (Fig. 3).

**DISCUSSION**

In this study, we couple behaviour, physiology and demographic 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 severely lower population rate of increase under size-dependent survival and fecundity. Importantly, our results show that social stress can alone drive demographic density-dependence without any significant change in the consumptive effects of interference or exploitative competition.

**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 (Höjesjö *et al.* 2007; Barcellos *et al.* 2007; Grosenick *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 is 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 can not be excluded that pike plasma cortisol levels were affected by this process. In particular, sampling might have increased cortisol levels more at low than 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 4 to 8 fish per 8 $m^3$ enclosure) reduced somatic growth by 50 % 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, 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.

**Demographic cost of stress**

Our results suggest 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 may have overestimated stress by doubling pike density (from 0.5 to 1 pike $m^{-2}$) because the doubling of densities rarely occurs under natural conditions, and because we did not provide any suitable habitat structure in which individuals could hide from each other. However, we may also have underestimated social stress by employing densities that were in the upper range of naturally occurring densities (see Materials and Methods). Indeed, if we reasonably assume that the relationship between density and social stress is asymptotic, a large density increase at high densities (as in our experiment) is likely to induce less stress than a smaller density increase at lower densities (as in the wild). Therefore, we suggest that stress increase in our experiment was within a roughly natural magnitude. 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 $\lambda$ than estimated here because (1) Windermere data taken as control in fact already include naturally occurring social stress, (2) we did not evaluate the direct effects of social stress on survival (e.g., immunity depression) and fecundity, and (3) we did not assume any social stress effect for survival to age-1. Therefore, we suggest that somatic growth decrease from social stress can have a *very* strong effect on population dynamics. Reduced individual growth from social stress also flattens the elasticity profiles of $\lambda$ 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.

**Conclusion and implications**

Stress allows vertebrates to cope with perturbations and recover homeostasis at the expense of suppressing non immediately-essential activities. In the long term, stress diverts energy away from immunity, somatic growth and reproduction. Our results suggest that somatic growth reduction from chronic social stress can, alone, severely decrease population rate of increase when survival and fecundity are size-dependent. Somatic growth reduction from social stress also flattens the elasticity of $\lambda$ to age-specific survival and fecundity, i.e., makes population persistence more fragile as it depends more on older, scarcer 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 is 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...) 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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**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: hepathosomatic index; selection of model structure based on AIC).

| Model number | Response | Random grouping factor | Random effect | Variance-covariance matrix for random effects | Heteroscedastic structure | Fixed effects | Estimate | SE of the estimate | P value* |
|---|---|---|---|---|---|---|---|---|---|
| 1 | $\sqrt{Fr}$ (n=244 observations)‡ | 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 vs. 2006) | -0.278 | 0.042 | <0.0001 |
| | | | | | | Time interval (days) | 0.053 | 0.005 | <0.0001 |
| | | | | | | $\sqrt{\text{Prey availability}}$ † | 0.809 | 0.035 | <0.0001 |
| | | | | | | Pike D (L vs. 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 vs. 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 vs.2006) | -0.344 | 0.126 | <0.0001 |
| | | | | | | FL at start (mm) | -0.008 | 0.001 | <0.0001 |
| | | | | | | Gender (males vs. females) | -0.208 | 0.095 | 0.0340 |
| | | | | | | Pike D (L vs. H) | 0.265 | 0.098 | 0.0079 |
| 4 | Growth in BM (n=119 observations) | Pond (n=4 levels) | Intercept | Symmetric | Product of different variance levels for each Exp and D | Exp (2007 vs. 2006) | -0.772 | 0.110 | <0.0001 |
| | | | | | | Pike D (L vs. H) | 0.418 | 0.115 | 0.0004 |
| 5 | HSI (n=120 observations) | Pond (n=4 levels) | Intercept | Symmetric | None | Exp (2007 vs. 2006) | 10.335 | 2.317 | <0.0001 |
| | | | | | | Pike D (L vs. H) | 5.605 | 2.344 | 0.0184 |
| 6 | Log(Plasma Cortisol) (n=98 observations) | Pond (n=4 levels) | Pike D | Diagonal | Product of different variance levels for each Exp and D | Exp (2007 vs. 2006) | -0.896 | 0.336 | 0.0038 |
| | | | | | | Log(FL at end (mm)) | -0.754 | 0.368 | 0.0422 |
| | | | | | | Pike D (L vs. H) | -0.250 | 0.345 | 0.4699 |
| 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)) | 1.743 | 0.365 | <0.0001 |
| | | | | | | Pike D (L vs. H) | 0.380 | 0.094 | 0.0001 |
| 8 | Plasma T3 (n=98 observations) | No random effect | | | None | Exp (2007 vs. 2006) | -0.682 | 0.145 | 0.0059 |
| | | | | | | Log(FL at end (mm)) | 2.996 | 0.600 | <0.0001 |
| | | | | | | Pike D (L vs. 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 vs. 2006) | -0.160 | 0.112 | 0.0194 |
| | | | | | | Log(FL at end (mm)) | -0.946 | 0.436 | 0.0117 |
| | | | | | | Pike D (L vs. H) | -0.288 | 0.097 | 0.0037 |

*: sequential decomposition of the contributions of fixed-effects terms.
†: i.e. number of prey pike$^{-1}$ at previous observation.
‡: this model also incorporated within-pond temporal autocorrelation (autoregressive function of time of order 1 and moving average of time of order 2).

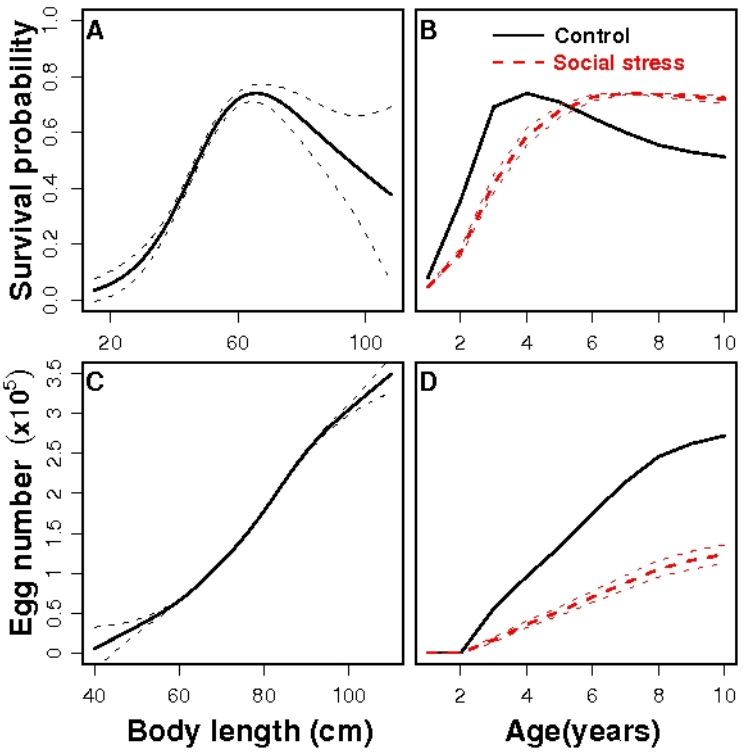


**Fig. 1. Projected effect of social stress-induced downsizing on size- and age-dependent vital rates of Windermere pike**. **A**. Windermere pike survival probability as a function of body-length with 95 % confidence limits. **B**. Corresponding survival probability as a function of pike age without (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).

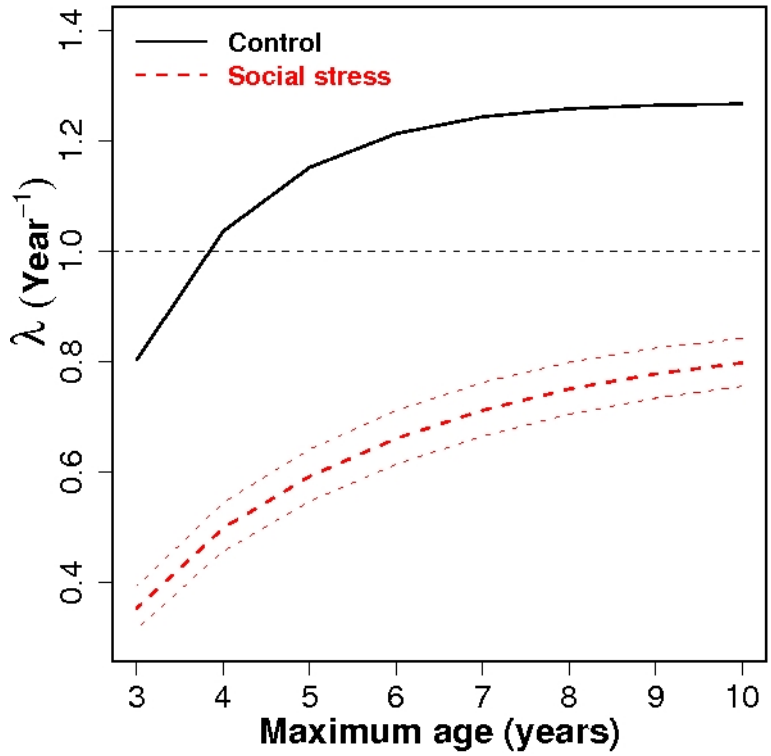


**Fig. 2. The demographic cost of social stress in pike.** Projected effect of experimentally-measured somatic growth reduction from social stress on Windermere pike population rate of increase (λ), estimated for different maximum ages in the population (starting from female first reproduction at age 3; thin red dashed lines represent 95% confidence limits for the effect of social stress on pike somatic growth). Horizontal, black dashed line at λ = 1 represents the limit at which population size is temporally stable ($N_{t+1} = N_t$).

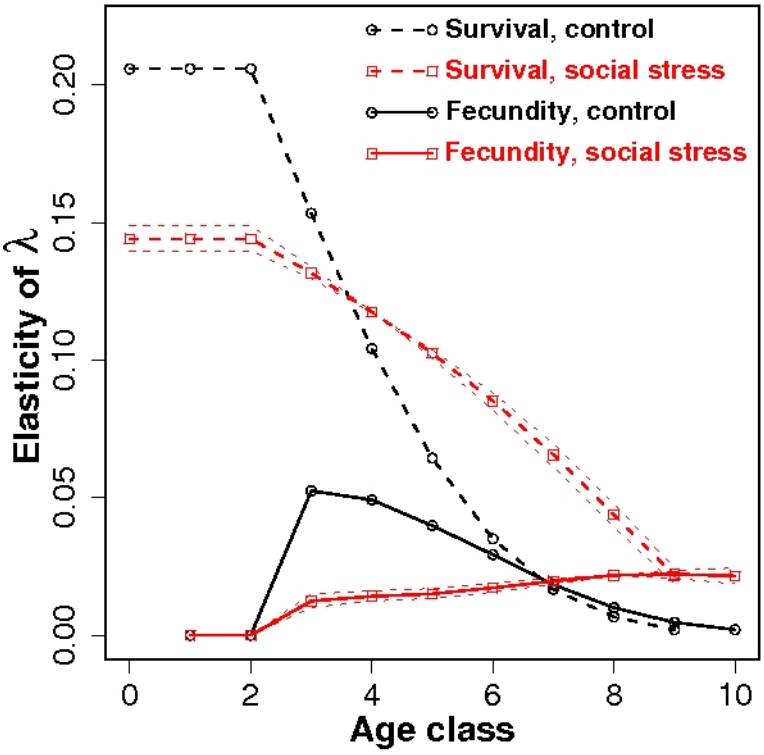


**Fig.3. Effect of social stress on population response to perturbation.** The elasticity of population asymptotic growth rate (λ) with respect to changes in age-specific survival and fecundity without and with the added effect of social stress on somatic growth rate (thin red dashed lines represent 95% confidence limits for the effect of social stress on pike somatic growth).