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Fish life history dynamics: Shifts in prey size structure evoke shifts in predator maturation traits

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Abstract

A bioenergetics framework is developed to predict optimal life history responses to environmentally driven changes in the rate of energy production by a predator. This framework is used to predict the responses of age at maturation, size at maturation and asymptotic size to changes in the predator/prey size ratio. Predators feeding on relatively smaller prey (i.e., having larger predator/prey size ratios) have lower growth efficiency and are predicted as a consequence to mature earlier, at smaller sizes and reach smaller asymptotic sizes. This prediction was tested using a 78 year time series (1936-2013) of data from a natural population of Lake Trout (*Salvelinus namaycush*) in Lake Opeongo, Algonquin Park, Ontario, Canada. A large decrease in the predator/prey size ratio for this population occurred over the period 1950-65 when a preferred prey (Cisco: *Coregonus artedii*) was introduced to the lake. This decrease was followed by ~ 20 years of constancy in the size ratio and then 25 years of progressive increase. Lake trout life history responded plastically during both periods and consistently with our predictions. Extensive analysis of available data provided little empirical support for alternative explanations for the observed changes in Lake Trout size and maturity (e.g. changes in Cisco and/or Lake Trout density and harvest rates). The framework developed here derives plastic life history changes from fixed developmental thresholds that are based on the scaling of net production with body size, and can be used to predict the shape of maturation reaction norms for the major shifts in community structure that are compactly summarized by changes in size spectrum parameters.

Key-words: phenotypic plasticity, reaction norm, non-native species, predation, optimal resource allocation, size-structured populations, allometry, Lake Trout.

37 Introduction

38 Life history (LH) traits (e.g. age or size at maturation - A_{mat} , L_{mat}) directly influence
39 survivorship and reproduction and therefore much of their variation in nature is expected to be
40 adaptive (Roff 2002). Optimal resource allocation models have been developed on this premise,
41 providing predictions of how natural selection will ‘adjust’ key traits (e.g. A_{mat} , L_{mat} ,
42 reproductive investment, egg size, maximum or asymptotic adult size, and even entire growth
43 trajectories) to a given environment through maximization of an index of fitness such as lifetime
44 reproductive output (Quince et al. 2008a;b). In seasonal environments, characterized by the
45 existence of a growing and a non-growing season, the optimal growth pattern is generally
46 biphasic, with a juvenile phase where all surplus energy is allocated to somatic tissue, followed
47 by an adult phase of asymptotic growth that results from increasing annual investment in
48 reproduction (Kozlowski 1996; Czarnoleski and Kozlowski 1998; Kozlowski and Teriokhin
49 1999; Thygesen et al. 2005). This model has been successful in predicting the maturation and
50 growth pattern of many fish species (Lester et al. 2004; Shuter et al. 2005; Scott and Heikkonen
51 2012), including Lake Trout (Quince et al. 2008b). Typically, it has been used to predict the
52 optimal LH traits appropriate to a set of fixed environmental conditions, rather than the ways in
53 which LH traits might track temporal changes in these conditions.

54 Temporal changes in the LH traits of harvested fish populations have been noted by a
55 variety of authors since at least the 1970s’ (e.g. Bagenal 1973; Shuter and Koonce 1977). In the
56 older literature, the dynamics of LH traits are often seen as simple reflections of the temporal
57 changes in somatic growth rate often observed in members of an exploited population as its
58 abundance declines with increasing harvest (Shuter 1990; Weatherley and Gill 1987). In the
59 more recent literature (e.g., Ernande et al. 2004; Heino and Dieckmann 2008; Lester et al 2014),

rapid changes in LH traits are seen as examples of phenotypic plasticity with temporal dynamics driven by a programmed, evolutionarily optimal response (i.e. the reaction norm or RN, and more specifically the maturation reaction norm or MRN - Sultan and Stearns 2005) to the changes in the net production (P , g/day) available to an individual. P results from the difference between energy acquisition (i.e., food ingestion) and energy loss associated with metabolism, egestion, and excretion (Jobling 1994). The relevance of P to LH is evident as it determines how much energy can be allocated to different – and potentially conflicting – fitness enhancing components, such as somatic growth and reproduction. Since both these bioenergetics processes and fitness components are dependent on body mass (W , g) (Peters 1983; Jobling 1994; Brown et al. 2004; Kooijman 2010), the scaling of P with W is a key element to predict optimal LH strategies and understand norms of reaction to environmental variation: the decision of how to best allocate energy to somatic versus reproductive tissue will depend on the relative benefits and risks associated with achieving larger sizes. For instance, if survivorship is sufficiently high and production increases rapidly with mass, it is optimal to delay gonad production and grow further to attain greater fecundity. In addition, as the basic bioenergetics processes determining production – in particular food ingestion and active metabolic rate – are strongly dependent on prey availability and quality (Jeschke et al. 2002; Kooijman 2010; Giacomini et al. 2013c), the production function $P(W)$ also provides a fundamental link between LH and trophic ecology.

Many studies (e.g. Lorenzen and Enberg 2002; Post et al. 1999; Rijnsdorp and van Leeuwen 1992; Trippel 1995) have focused on plastic changes in growth and maturity associated with declines in population density and consequent increases in prey availability. However, overall prey availability is not the only environmental variable that could produce immediate changes in the energy budget of individual fish – prey quality, as well as quantity, also plays an

83 important role. Perhaps the simplest and most pervasive aspect of ‘quality’ is the size of prey
84 relative to the size of its predator, which is commonly measured by its inverted index: the
85 predator/prey size ratio (R) (Andersen and Beyer 2006; Brose et al. 2006). The average value of
86 R available to individual predators will depend on the distribution of individual body sizes within
87 the ecosystem (i.e., the community size spectrum, Sprules and Barth this issue), which in turn is
88 responsive to fishing pressure and other perturbations such as habitat modification, climate
89 change, and species invasions (Rice and Gislason 1996; Bianchi et al. 2000; Woodward and
90 Hildrew 2001; Jennings and Blanchard 2004; Emmrich et al. 2011; Yvon-Durocher et al. 2011;
91 Chu et al. this issue). The literature is replete with examples demonstrating how a change in R
92 may indirectly trigger plastic growth and LH responses in fish by changing the relative energetic
93 benefit associated with predating on large vs small organisms (Paloheimo and Dickie 1966;
94 Confer and Blades 1975; Kerr and Ryder 1977; Mittelbach 1981; Trudel et al. 2001; Sherwood et
95 al. 2002a,b; Pazzia et al. 2002; Iles and Rasmussen 2005; Kaufman et al. 2006; Giacomini et al.
96 2013c; Golet et al. 2015). A simple mechanism underlies these responses: the energetic costs of
97 acquiring a unit biomass of food usually becomes lower for relatively bigger prey. Therefore, as
98 a fish consumer grows and its individual metabolic demands increase, its growth efficiency will
99 generally decline if it must rely on prey of a constant size (Paloheimo and Dickie 1966; Kerr and
100 Ryder 1977; Werner and Gilliam 1984; Mittelbach and Persson 1998; Iles and Rasmussen 2005),
101 promoting an energetic bottleneck that prevents it from reaching larger sizes (Heath and Roff
102 1996; Sherwood et al. 2002a,b). This can happen despite higher overall biomass of smaller prey,
103 and has been observed in many fish populations in both freshwater and marine systems
104 (Paloheimo and Dickie 1966; Kerr and Ryder 1977; Hart and Connellan 1984; Forseth et al.
105 1994; Saint-Jacques et al. 2000; Trudel et al. 2001; Sherwood et al. 2002b; Pazzia et al. 2002;

Henderson et al. 2004; Kaufman et al. 2006; Golet et al. 2015). In some cases, it can lead to dwarfism (Ylikarjula et al. 1999), involving various combinations of slower growth, earlier maturation and smaller asymptotic sizes (Jansen 1996; Trudel et al. 2001; Pazzia et al. 2002; Dunlop et al. 2005; Juncos et al. 2011).

The literature on the aforementioned ‘fast’ LH changes associated with phenotypic plasticity in fish populations is paralleled by a broad and recent literature that focuses on the relatively ‘slow’ evolutionary LH changes (e.g. Andersen and Brander 2009; Conover et al. 2009; Engberg et al. 2012) that can be induced in fish populations by the intense mortality that exploitation can impose on sub-adults and adults. Evidence supporting the occurrence of these evolutionary effects in exploited populations has been presented for a variety of marine and freshwater species (e.g. Grift et al. 2003; Jorgensen et al. 2009; Sharpe and Hendry 2009). The joint empirical findings that slow evolutionary LH change can be driven by selection through imposed mortality and that fast plastic LH change can be driven by changes in individual production, provide the basis for a mechanistic theory of the interplay between fast and slow LH change. The mathematical framework for this theory has been developed in the context of models of optimal resource allocation in seasonal environments by Kozlowski et al. (1996) and Quince et al. (2008a). It leads to the following general predictions: (i) the optimal value for asymptotic mass (W_{∞}) or length (L_{∞}) occurs when the marginal gain in P , achieved by an increase in size (i.e., $\partial P / \partial W$), falls to a critical threshold value; (ii) the value of that threshold varies directly with adult mortality rate and inversely with growing season length.

This theory then goes on to define how the optimal W_{∞} (or L_{∞}) will change with any of the environmental variables that affect the individual production function $P(W)$, and how the pattern of such changes (i.e., the optimal RN for W_{∞}) will itself change under the selective

pressures imposed by changes in adult mortality rate. In this paper, we pursue two objectives: (i) to extend this theory by deriving the RNs for the LH traits that define the start of reproductive life – A_{mat} and L_{mat} ; (ii) to test this extended theory using a comprehensive 78 year data set that documents the plastic life history changes observed in a top predator population (Lake Trout – *Salvelinus namaycush*) after the individuals in that population experienced significant changes in their average predator/prey size ratio, because of the introduction of a new prey species (Cisco – *Coregonus artedii*).

Methods

Life History Theory

Since production $P(W)$ generally scales sublinearly with mass W (i.e., $\partial^2 P / \partial W^2 < 0$ for most of W , Ernest et al. 2003; Giacomini et al. 2013b,c), there will be a point at which the advantage of further growth will be outweighed by the risk of mortality, and all surplus energy should be used for reproduction. This condition defines the optimal asymptotic body mass (W_∞), and is given (Quince et al 2008a) by the equation:

$$\left. \frac{\partial P}{\partial W} \right|_{W_\infty} = \frac{(1-s)}{D} \tag{1}$$

where s is annual survivorship (proportion of surviving individuals) and D is growing season length (days). Eq. (1) defines an optimal threshold that is independent of the parameters of the production function and hence defines the optimal dynamics for W_∞ (i.e. the RN for W_∞) given a temporal change in any of the parameters that determine P . As part of our first objective in this paper, we want to determine if there exists a similar threshold on $\partial P / \partial W$, independent of the parameters of $P(W)$, that defines optimal size and age at maturation (the MRN linking A_{mat} and

L_{mat}) for changes in $P(W)$. This threshold is not analytically defined and neither are the optimal values for A_{mat} and L_{mat} : both require the numerical calculation of the entire optimal growth trajectory in order to be determined (Quince et al. 2008a). We calculated optimal growth trajectories using numerical methods described in Quince et al. (2008a) and in the online Supplementary Material. Different growth trajectories and life history traits were obtained by varying the production function $P(W)$ with biologically reasonable parameters, using Lake Trout and other salmonids as a reference (Table 1). We then compared the exact optimal MRN, calculated from optimal growth trajectories, to that estimated using a common threshold for $\partial P/\partial W$. To evaluate the relative sensitivity of the optimal maturation threshold (i.e., the value of $\partial P/\partial W$ at the optimal maturation size), we performed simulations varying each model parameter randomly and independently over a range equal to its reference value $\pm 10\%$. We discarded any simulation leading to W_{∞} outside the range 300g – 35kg, which are reasonable bounds for Lake Trout, until the total number of accepted simulations reached 100,000.

To simulate changes in $P(W)$, we used the model developed by Giacomini et al. (2013c). This model explicitly accounts for the energetic costs and benefits of foraging for prey varying in density and size, thus establishing a formal link between LH and prey community structure. It assumes a simple energy budget in which assimilation and expenditure of energy are functions of body size (W , g) and swimming speed (U , m/day):

$$P = eI - (a_1 + a_2 U^c) W^{\beta} \quad (2)$$

where e is assimilation efficiency, which accounts for losses from egestion, excretion, and specific dynamic action (SDA, the metabolic cost of digesting food), I is ingestion rate (g/day), a_1 , a_2 , c and β are coefficients defining standard metabolic (given by $a_1 W^{\beta}$) and active metabolic

rate (given by $a_2 U^c W^\beta$), which are commonly assumed functions in bioenergetic models (Videler 1993; Jobling 1994; Kooijman 2010).

Ingestion rate (I , g/day) depends on the amount of foraging effort (i.e., swimming speed U , m/day) and on the abundance and quality (i.e., size) of prey, but with a satiation limit (maximum ingestion) given by the allometric function $I_{\max} = a_3 W^\beta$. Prey abundance N (number of individuals) varies with prey size w (g) according to a power law size spectrum $N = \kappa w^{1-\lambda}$, which gives the distribution of particles in the ecosystem along logarithmic uniform intervals of body size (Sprules and Barth this issue). The parameter κ gives a measure of overall abundance or productivity in the system, whereas λ (the size spectrum slope) measures the relative abundance of small versus large organisms. This is perhaps the simplest representation of community structure which has both strong theoretical (Andersen and Beyer 2006; Law et al. 2009; Capitan and Delius 2010) and strong empirical (Kerr and Dickie 2001; Reuman et al. 2008; Gomez-Canchong et al. 2013) support.

Prey size is in turn determined by the predator's own size, according to a predator/prey mass ratio $R = W/w$. Having a constant R implies the fish will feed on larger prey as it grows, thus implicitly including ontogenetic diet shifts which are widespread in fish. The costs and benefits of foraging on prey of different sizes (and abundance) will also depend on the average distance between successful prey capture events, which is assumed to scale with prey abundance as $a_4 N^{1/h}$ (i.e., it is proportional to the average distance between neighbor organisms, Chandrasekhar 1943; Clark and Evans 1954). In this case ingestion by non-satiated fish is given by $I = wU/(a_4 N^{1/h})$, where a_4 is a coefficient of proportionality and h is the dimensionality of the habitat searched by the predator (e.g., $h = 3$ for three-dimensional environments such as the pelagic zone, and $h = 2$ for two-dimensional environments such as the water surface or the lake

bottom). Non-integer h values are also possible, for instance if the space used by the forager is strongly compressed in one or more dimensions (e.g., fish constrained to a thin layer of water due to temperature limitations can have h between 2 and 3).

The optimal foraging strategy is defined by the choice of foraging effort leading to the highest possible production (i.e., $\partial P/\partial U = 0$ and $\partial^2 P/\partial U^2 < 0$), and can involve adjustments in both ingestion rate and active metabolism, or in active metabolism only. Giacomini et al (2013c) showed that, over most of the realistic parameter space for their model, active metabolism emerged as the most influential adjustable component. This implies that optimal feeding is achieved by adjusting activity to satiate consumption (i.e., $I = I_{\max} = a_3 W^\beta$). Production under this constant satiation hypothesis is given by:

$$P = (ea_3 - a_1)W^\beta - a_2 \left(\frac{a_3 a_4 R^{1-(\lambda-1)/h}}{\kappa^{1/h}} \right)^c W^{\beta+c(\beta+(\lambda-1)/h-1)} \quad (3)$$

The influence of prey on net production is represented mostly by κ , λ , and R . Higher κ will invariably enhance production, whereas higher exponent λ promotes more rapid declines in net production with increasing consumer mass (i.e., larger individuals become relatively more limited by food). This formulation is a mechanistic elaboration of the more generic bioenergetics structures used by Thygesen et al. (2005) and Christensen and Andersen (2011). There is an alternative version for $P(W)$ derived by Giacomini et al. (2013c), one in which optimal foraging does not lead to satiation. However, it has the same general structure as Eq. (1) – it is given by the difference between two power functions – and has similar functional dependencies between $P(W)$ and the same parameters analyzed here, so it is not going to be considered here for simplicity.

In this paper we will pay particular attention to the MRNs emerging from changes in the predator/prey mass ratio R . There are two reasons for this emphasis: (i) the MRNs to changes in R have received relatively little attention in the literature to date; (ii) we have access to an extensive empirical data set that is well suited to testing predictions about the shape of the MRN to changes in R . The predator/prey mass ratio is the product of both biomechanical constraints (e.g. gape limitation) and behavioral preference for prey of different sizes, depending on their availability and predator-specific characteristics. Larger R implies the predator forages on relatively smaller prey. The effect of R on P is dependent on h and λ , which determine its exponent in Eq. (3): its effect will most likely be negative ($\partial P / \partial R < 0$) as h is usually close to 3, especially in pelagic environments, while λ tends to be near 2 (Kerr and Dickie 2001; Andersen and Beyer 2006; Sprules and Barth this issue). This inference is consistent with the many studies of growth in fish that show the general benefits of feeding on large prey (e.g. Paloheimo and Dickie 1966; Kerr and Ryder 1977; Heath and Roff 1996; Pazzia et al. 2002; Sherwood et al. 2002b; Golet et al. 2015). With an overall decline in $P(W)$, we expect any threshold for $\partial P / \partial W$ (e.g., Eq. 1) to be reached at smaller body sizes, which implies that predicted optimal values for L_{∞} and L_{mat} (or W_{∞} and W_{mat}) should both decrease with increasing R .

Empirical Tests of Life History Models

Our tests are based not on controlled experiments, but on retrospective assessments of the observed behaviour of natural populations over several decades. Hence the risk of contamination of the results by uncontrolled factors associated with extraneous natural and human factors is high. We have tried to minimize these risks by carefully identifying likely issues, assessing their importance and, where necessary, allowing for their influence in the analysis. Because these efforts are essential to the credibility of our tests we have described them in some detail below.

All analyses were performed in MATLAB® (The MathWorks, version R2015a), unless stated otherwise.

Data Sampling and Estimation of Demographic Statistics

Background

Lake Opeongo is located in the centre of Algonquin Park, a large nature reserve established in south central Ontario in 1893. There has been little anthropogenic development around the lake over this period, therefore it has remained in an oligotrophic state, with high oxygen levels, low nutrient levels and normal pH values, (St Jacques et al 2005). Annual monitoring of the status of the resident Lake Trout population began in 1936 and has continued through 2015 (Martin and Fry 1973; Shuter et al. 1987; Matuszek et al. 1990; Shuter et al. 1998; Quince et al. 2008b). The core of the monitoring program is an annual angler survey that has been in operation continuously since 1936. Anglers are interviewed from the opening of the recreational fishery at ice-off (April or May) until its closure at the beginning of the Lake Trout spawning season (October). The creel program schedule ensures collection of a large, representative sample of angler catch and effort over the entire fishing season. These data are then used to estimate the total harvest and effort for the year. In addition to interviewing anglers and recording catch and effort, morphometrics, ageing structures and stomach contents are collected from each fish presented to the creel technicians (see Shuter et al. 1987 for a detailed description of the creel program on Lake Opeongo).

A primary goal of the creel program was to collect the data needed (i.e. total harvest by age group) to derive annual estimates of Lake Trout population abundance and mortality rate using catch at age methods. The protocols used to collect these data have remained relatively unchanged over the time-series, with the exception of a transition (from 1987 to 1990) from scale

to otolith ageing and the establishment of a standardized stratified sampling scheme for ageing fish. In the standardized approach, fork lengths (L , cm) of the sampled fish are categorized within 2 cm length bins. Within each bin, 15 fish are randomly selected for aging, the age distribution found in this aged sample is applied to any unaged fish within the length bin and the resulting age distributions are then pooled across bins to arrive at an estimate of the overall age distribution for the annual harvest. Prior to 1987, either all fish or a large random sample of fish were aged and the age distribution of the random sample was applied to the entire harvest. Typically the number of fish aged in each year was > 200 and this represented at least 25% of the total number of fish sampled.

Cisco (*Coregonus artedii*) were introduced to Lake Opeongo in 1948, had become the dominant prey of adult Lake Trout by 1960 and continue to be the dominant prey to the present. Sporadic summer gillnet sampling of the Cisco population began in 1972. These data provide information on changes in both the relative abundance of Cisco and the size structure of the Cisco population for the period from the early 1970's through 2010.

Age Assessment for Lake Trout

Scale samples were used to determine the age (A , years) of individual Lake Trout over the first 50 years of the time series. This was followed by a transition to otolith-based methods, which have been used consistently from 1991 to the present. Both methods give similar values up to $\sim$ age 12 (Shuter et al. 1987); for older fish, scale ages typically underestimate the 'true' otolith age. This shift in aging methodology was allowed for in all subsequent analyses by assuming that: (i) scale and otolith methods were comparable in their ability to assign fish to year- specific age groups up to and including age 12; (ii) both methods were comparable in their ability to assign fish to an age group consisting of fish aged 13 and over.

Assessing Individual Somatic Growth Patterns for Lake Trout

Fish aged 15 years and older were identified in the period 1994-2013 from otolith ages and 130 males and 130 females were selected randomly from this group. Back-calculation methods were applied to the otolith samples from these fish to estimate annual size at age throughout each individual's life time. These samples were limited to older fish so that the backcalculated lengths from each individual would cover ~7 years of pre-maturation growth and ~7 years of post-maturation growth (Matuszek et al. 1990). A calibration curve relating otolith size to fork length was generated for the fork length range [10, 80 cm] and this was used to estimate fork length at age from measurements to the individual annuli on each otolith.

Assessing Sexual Maturity for Lake Trout

Each fish sampled in the creel survey was categorized as immature ($M = 0$) or mature ($M = 1$) based on the observed stage of its gonad development at the time it was sampled. To avoid misclassifications due to early assessment of gonads, only maturity data collected on or after the 200th day of each year (late July) were included. This corresponds approximately to the time when the gonad-somatic index of those fish that are investing in reproductive tissue (and are therefore likely to attempt spawning in the coming October spawning period) diverges from those fish that are not investing in reproductive tissue in that year (Morbey and Shuter 2013).

A more complete picture of reproductive life could be estimated for the individuals whose lifetime somatic growth patterns were re-constructed through back-calculation. Each individual growth pattern was clearly bi-phasic, with an initial phase characterized by quasi-linear growth and a second phase characterized by declining annual growth as an asymptotic maximum size was approached. This shift from linear to asymptotic growth is the expected consequence of initiating investment in reproductive tissue (Quince et al. 2008a;b) at the age of

maturation. The shift from linear to asymptotic growth in each individual growth pattern was sufficiently distinct that A_{mat} and L_{mat} could be estimated directly by visual inspection. We identified A_{mat} as the fish age during the first year characterizing the second growth phase. The individual growth curves, A_{mat} , and L_{mat} determined using this method are presented in the Supplementary Material.

Assessing Lake Trout Stomach Content

The stomach of each sampled Lake Trout was removed, opened and each identifiable prey item was counted and categorized into one of the following groups: plankton, aquatic insects, terrestrial insects, crayfish, and fish. When the level of digestion permitted it, each fish was identified to species and its total length was measured.

Estimating Absolute Abundance and Mortality for the Lake Trout Population.

The general approach used in previous studies of the Opeongo Lake Trout population (e.g. Shuter et al. 1987) was applied to the annual estimates of angler harvest at age derived from the creel survey to generate annual abundance and mortality estimates. The VPA/ADAPT harvest-at-age analysis package (NOAA Fisheries Toolbox, 2010, VPA / ADAPT version 3.4.5) was used to estimate the annual abundance and mortality of Lake Trout 7 years of age and older for the period 1936 through 2005. The age range of 7 to 13 and older was chosen for the VPA/ADAPT analysis based on the age of full recruitment to the fishery and the precision of the aging protocol prior to 1990: (i) over the 70 year time series, Lake Trout aged 7 and over were expected to be fully recruited to the recreational fishery: focusing on this range of ages satisfied the VPA/ADAPT model assumption that age specific catchabilities are constant over time; (ii) from 1936 until 1989, aging was based primarily on scale samples and from 1990 on, aging was based primarily on otolith samples; scale ages progressively underestimate true age after $A \sim 12$

years, therefore we focused the VPA/ADAPT analysis on harvest estimates for each age from 7 through 12 plus an estimate of the harvest of all fish aged 13 and over; this ensured relatively consistent precision of age assignment throughout the time series. Following Shuter et al (1987), we assumed a constant natural mortality rate of 0.11 for all age groups in the analysis.

The abundance at age and fishing mortality values from 1936 to 2005 are reported in the Supplementary Material. The abundance at age estimates were evaluated using a bootstrapping approach with 10,000 samples as well as a retrospective analysis to ensure that the abundance assumed for the terminal data year (2010) plus 1 did not greatly influence the abundance at age time series. As in Shuter et al (1987), year-specific independent abundance estimates based on simple Peterson mark-recapture methods were used to verify the VPA/ADAPT abundance estimates and provide an abundance estimate for the terminal data year plus one. Peterson abundance estimates were based on annual fall mark/recapture sampling done on the primary Lake Trout spawning shoals over the period 1999 to 2011. Harvest data through 2010 were used in the VPA/ADAPT fits but we only report abundance/mortality estimates through 2005 because these values are robust to uncertainty in the estimate of terminal year abundance. This is a property that is common to all harvest at age methods for abundance estimation (Quinn and Deriso 1999).

Estimating Relative Abundance and Size Structure for the Cisco Population

A synoptic gill net sampling program was begun in 1971 and continued sporadically through 2010. The primary purpose of this program was to monitor temporal change in the species composition of the Lake Opeongo fish community. Nylon monofilament nets with standard mesh sizes (0.75, 1.0, 1.5 in) were used in all years but detailed information on the annual netting effort was only recorded in some years. These data were used to generate annual

catch-per-unit-effort indices for the Cisco population to track temporal changes in its relative abundance. The fork lengths and weights of all Cisco captured in this annual summer netting program were recorded in all years and these length distributions were used to track temporal changes in the size structure of the Cisco population and the condition factor ($\text{weight}/\text{length}^3$) of individual Cisco. Scales were collected from the 1970's through the 1990's and otoliths were collected in the 2000's. Size-at-age was back-calculated from scale measurements (Francis 1990) to assess changes in growth rate over the time series. Otolith ages from the 2000 and 2001 were used to construct an accurate lifetime growth pattern for that point in the time series.

Estimating R and Visualizing Associated Changes in Lake Trout Size Distributions

Overall changes in R for the time frame of the study were derived by aggregating the individual predator/prey mass ratios for Lake Trout sampled in the creel survey. Mass for both predator and prey were estimated assuming a cubic relation between mass and length. Only fish prey were included to ensure that the cubic assumption was not violated by including organisms of very different body shape. A total of 7366 prey length measurements were used in these calculations. The predator/prey mass ratio used as the 'predictor' variable in all subsequent analyses represents an aggregate of observed values within a given time interval – here we used the median of the individual mass ratio values because they were typically asymmetrically distributed within any time interval. We used different time intervals for different analyses, always attempting to define an interval that was most relevant to the objective of the analysis.

To visually assess the association between temporal changes in R , as defined above, and temporal changes in Lake Trout size structure, we compared both data sets over a sequence of seven 12-year intervals (except for the last interval, from 2008 to 2013). A 12-year interval was chosen because: (i) it matches the period with available data that precedes the Cisco introduction

in Opeongo (1936-1947); (ii) it is comparable to the generation time for Lake Trout. For each of these intervals, Lake Trout population size structure was characterized as the length frequency distribution of all fish with $L > 48\text{cm}$. This lower size limit was chosen to eliminate bias in the size distribution from size selectivity in the angler based sampling process: current and past (Shuter et al. 1987) VPA analyses confirm full recruitment to the fishery at ages 7 to 8 and, throughout the time series, fish with $L \geq 48\text{ cm}$ were aged ≥ 8 years.

In order to link R estimates more closely to what was actually experienced by each Lake Trout over its lifetime, we generated a ‘lifetime’ R value for each Lake Trout individual by calculating the median R value for the period from its year of birth (calculated from its age) to the year of its capture. For instance, if a lake trout was captured in year 1970 at age 10, the mass ratio attributed to this individual will be the median value of all mass ratios calculated from stomach contents sampled within the period 1960-1970. These lifetime R estimates were used as a predictor in the analyses described below relating LH RNs to changes in R ; the dependent variables were derived from the maturation status, size and age of individual Lake Trout. We also performed more restricted calculations of R including only stomach contents from Lake Trout belonging to the same cohort (i.e., those fish that experienced the same conditions in the same year at the same age) as the focal individual. In both sets of analyses, only calculations of R that involved at least 30 stomach items were considered.

Estimating RNs for A_{mat} , L_{mat} , and L_{∞}

Our objective was to estimate LH RNs to changes in R . There is much variation in the terminology associated with RNs, and the MRNs in particular. Here we use the terminology for RN estimators suggested by Sultan and Stearns (2005). The distinctions they draw between different estimators all derive from the very general definition that a RN is the pattern of

phenotypic expression for a given genotype resulting from changes in environmental conditions. Our focus will be on data that permit us to detect systematic changes in what Sultan and Stearns (2005) have called the ‘population average reaction norm’, which reflects the ‘average’ pattern of expression of the set of genotypes existing in the population at the time of sampling. These data are required to (i) reflect individual maturation decisions (ii) be consistently measurable throughout as much of the time series as possible, (iii) not reflect genotypic changes – which we regard as generally reasonable given the time scale of our study. Both the creel survey and individual back-calculated size-at-age data sets fit these criteria for A_{mat} , L_{mat} , and L_{∞} .

A primary distinction between the two data sets is that the creel gonad development data cannot distinguish between first-time and repeat spawners, and therefore estimates of A_{mat} and L_{mat} from these data will reflect not just age and size at first maturity, but also: (i) how the ratio of first-time to repeat spawners changes with age and size; (ii) how demographic factors (e.g. variation in mortality pre- and post-maturation) affect how this ratio changes with age and size (for details, see Pérez-Rodríguez et al. 2009). Estimates of A_{mat} and L_{mat} from the individual back-calculation data are free of such biases and should more clearly reflect changes in size and age at first maturity.

Estimating MRNs from Creel Gonad Development Data

Size and age MRNs to changes in R were estimated through logistic regressions (i.e., Generalized Linear Model with logit link and binomially distributed residuals), using maturation status M as a response variable and including lifetime R estimates for each Lake Trout as one of the predictors. We included sampling period (denoted here by S ; $S = 1$ for fish caught before 1975, and $S = 0$ otherwise) as a categorical covariate to account for differences in age determination methods and for potential environmental differences between the two periods,

each encompassing half of the time series and a similar range of variation in R , although with opposite temporal trends. It led to the following logit model:

$$\ln\left[\frac{\pi(X, R, S)}{1 - \pi(X, R, S)}\right] = \delta_0 + \delta_1 X + \delta_2 R + \delta_3 S + \delta_4 RS \quad (4)$$

where π is the probability that a Lake Trout individual will be mature ($M = 1$), which is a function of covariates X (representing either A or L), R , S , and their interaction RS ; and δ_j ($j = 0, 1, 2, 3, 4$) are model parameters. This model represents the best model for this set of predictors according to the Akaike Information Criterion (see Supplementary Material for model comparisons). A_{mat} (or L_{mat}) was estimated as the X value at 50% probability of maturity ($\pi = 0.5$), which can be determined for any combination of R and S by setting Eq. (4) = 0 and solving for X :

$$\hat{X}_{\text{mat}}(R, S) = -(\hat{\delta}_0 + \hat{\delta}_2 R + \hat{\delta}_3 S + \hat{\delta}_4 RS) / \hat{\delta}_1 \quad (5)$$

where $\hat{X}_{\text{mat}}$ and $\hat{\delta}_j$ denote the estimated age (or length) at maturation and the estimated parameter values. This model assumes a linear relationship between A_{mat} (or L_{mat}) and R , which is a reasonable approximation to theoretical predictions when R has a short-range of variation (Figure 1c,d). Females and males were analysed separately.

Estimating L_{∞} RNs from the Creel Size-at-Age Data

We estimated the L_{∞} RN to changes in R by modelling the creel size-at-age data as a function of R . We used a modified von Bertalanffy (vB) growth equation. The standard vB formulation relating length (L) to age (A) is:

$$L = L_{\infty} \left[1 - e^{-k(A - A_0)}\right] \quad (6)$$

where k is a growth rate parameter that measures the rate of approach to asymptotic length L_{∞} , and A_0 is a position parameter measuring the hypothetical age at length zero. The vB growth

pattern is the pattern expected of an adult fish that is optimizing its reproductive investment, in the specific case with $P(W) \propto W^{2/3}$ (Lester et al. 2004; Quince et al. 2008a). Although it does not conform strictly to the more complex unimodal production function of Eq. (3), which cannot be solved analytically, the power function above can serve as a useful approximation, further justified by the fact that optimal somatic growth should stop before P starts declining with W , so the realized production function will in general be a monotonic function of size (i.e., only the ascending part of Eq. 3 would be observed). It is also sufficient for our purpose of assessing the effect of R on P and the consequences of that influence on the optimal lifetime schedule for reproductive investment.

In the modified vB model, we assumed that L_{∞} decreased linearly with R , which is a reasonable approximation of theoretical predictions for short ranges of variation in R (Fig. 1). Sampling period S was also included as a covariate through its interaction with R , so that $L_{\infty} = L_{\infty}^* - \theta_1 R + \theta_2 RS$, where the intercept L_{∞}^* represents a hypothetical upper bound on L_{∞} , extrapolated to the limit $R = 0$, θ_1 measures the main effect of R , and θ_2 measures the effect of the interaction RS , which allowed us to test whether the influence of R was period specific. We did not include a term for the main effect of S on L_{∞} due to the lack of appropriate data for $A > 12$ years in the period before 1975 – a deficit which led to unreliable estimates of L_{∞} for this period (> 2 meters long) if a period-specific intercept estimation was adopted. The existence of reliable size estimates for older ages for the later period mitigates this problem and also helps anchor the size-at-age estimates for the earlier period, keeping them within realistic bounds. In addition, we expected that systematic variation associated with periods would affect growth curve shape, represented by the parameters k and A_0 . So the influence of S was also included in the exponential part of Eq. (6). The resulting model is:

$$L = (L_{\infty}^* - \theta_1 R + \theta_2 RS) \left[1 - e^{-k^* (A - A_0^* + \theta_3 S + \theta_4 AS)} \right] \quad (7)$$

where k^* and A_0^* correspond to the growth rate parameter k and the position parameter A_0 for the second sampling period starting in 1975 (i.e., when $S = 0$), θ_3 and θ_4 measure the influence of sampling period S and the age x period interaction (AS), respectively. The inclusion of the last interaction term results from the assumption that k is linearly related to S , in combination with the multiplicative relationship between k and A (Eq. 6). The parameter values of Eq. (7) were estimated through non-linear regressions, using least-squares estimation. As the asymptotic growth pattern characteristic of the vB equation is expected to occur as a consequence of investment in reproduction (Lester et al. 2004), only mature ($M = 1$) individuals were included in this analysis. We also excluded all fish younger than 7 years old, which is around the average age at maturation estimated from the previous analyses. Females and males were analysed separately.

Estimating RNs from Lifetime Growth Pattern Data

In the back-calculated size-at-age data, age and size MRNs to changes in R were estimated directly from observed A_{mat} and L_{mat} values and estimates of lifetime R for each Lake Trout individual included in the analysis. The estimation of the L_{∞} RN to changes in R proceeded in stages. The existence of reliable prematuration growth data for many individuals allowed us to estimate the allometry of net production P . This was useful in order to adjust the von Bertalanffy growth equation into a generalized form that is free from the assumption of a 2/3 exponent (Quince et al. 2008a; Boukal et al. 2014). A variable exponent β^* can encapsulate variation in the curvature of the production function, leading to a closer approximation of the more complex unimodal function of Eq. (3). The procedure to estimate β^* is described in the Supplementary Material, and the resultant estimates varied from 0.610 to 0.792, with an average 0.711 (2/3 is a

possible result but the estimated mean closely matches expectation given empirical bioenergetic estimates in Hanson et al. (1997) and Quince et al. (2008b).

The estimation of L_{∞} for each individual then followed a two-step procedure:

- (i) we used the estimated β^* to transform post-maturation length values into the size variable $V = L^{3.11(1-\beta^*)}$ (Quince et al, 2008a), and then fitted the von Bertalanffy Eq. (6) through least-squares nonlinear regression using V instead of L . The value 3.11 is the exponent of the weight-length relationship estimated for immature Lake Trout in the creel data.
- (ii) the asymptotic lengths were calculated by converting V_{∞} back into length units (cm), from the relationship $L_{\infty} = V_{\infty}^{1/[3.11(1-\beta^*)]}$.

The influence of R on the life history traits A_{mat} , L_{mat} , and L_{∞} was first tested by analyses of covariance including sex as a covariate. Due to the absence of a significant effect of sex, male and female data were grouped and analyzed through simple linear regressions.

Results

Theoretical Results

Examples of optimal growth curves are presented in Figure 1a. Growth is discontinuous within each year, as L remains unchanged during the period that includes gonad production and the non-growing season. Fig. 1 also illustrates the expected effect of changing R on the shape of growth curves (Fig. 1a), and on optimal life history traits (Fig. 1b-d). The values of other parameters are presented in Table 1. Optimal L_{∞} occurs at the intersection of the production derivative $\partial P/\partial W$ with a threshold (hereafter defined as P'_{∞}) defined by survivorship and growing season length, as stated in Eq. (1) and shown in Fig. 1b. Increasing R leads to sharper declines in the derivative, resulting in smaller L_{∞} (Fig. 1c).

The same reasoning can apply to L_{mat} . Although the exact optimal L_{mat} does not follow strictly a constant threshold (Fig. 1b) as L_{∞} does (i.e. this variation stems mostly from the fact that growth is discontinuous within a year), using a common threshold (i.e., P'_{mat}) leads to approximate solutions that are very close to the optimal (Fig. 1c-d). Age at maturation also responded negatively to R , so the MRN relating L_{mat} and A_{mat} as a consequence of variation in R has a positive slope (Fig. 1d).

In addition, extensive simulations showed that the value of $\partial P/\partial W$ at the optimal L_{mat} is strongly related to P'_{∞} (Fig. 2a), depending strongly on the same parameters (s and D) that determine P'_{∞} (Eq. 1) and, to a lesser extent, on the parameters of the production function (Fig. 2b). Despite the apparently short range used to vary model parameters in these simulations (i.e., reference values $\pm 10\%$), it was sufficient to generate a broad and representative distribution of optimal L_{mat} (Fig. 2c), with a mean of 50.2cm and a range from $\sim 20\text{cm}$ to $\sim 120\text{cm}$, which covers the range found in many salmonids (e.g. Lake Trout, see McDermid et al. 2010, Table 2).

Empirical Results

Temporal Changes in Lake Trout and Prey

There was a marked increase in overall Lake Trout size in the first half of the time series, peaking in the 1960's and followed by a later period of decline towards levels similar to those observed early in the time series (Fig. 3a). These changes in body size distribution are closely related to changes in the median predator/prey mass ratio R regarding fish prey in general, and Cisco in particular (Fig. 3a,b). A more detailed picture of the U-shaped pattern of variation in R over the time series is evident in our lifetime estimates of R for individual fish (Fig. 3c).

The initial decline in R is associated with the emergence of Cisco as a prey item during the 1950's and the concomitant decline of Yellow Perch (Fig. 3d). The initial persistence of

Whitefish and the inclusion of some big prey species such as Burbot also explain the observed change in R during the first half of the time series, as noted by Martin (1970). After the 1960's, Cisco became the dominant prey item and no further change in prey composition was observed. Lake Trout abundance increased and fishing mortality decreased during the first decades (Fig. 3e) of the time series. Lake Trout abundance peaked in the early 1970's, fluctuated around relatively high levels through the 1980's and 90's and then gradually rose to a new peak in 2005 (Fig. 3e). The decline in fishing mortality during the first period of study (1936-1974) follows the observed increase in Lake Trout size, represented by annual values of the 90% percentile (Spearman correlation coefficient $\rho = -0.56$, $p < 0.001$, $n = 39$). However, the correlation is no longer significant during the second period from 1975 to 2005 ($\rho = 0.21$, $p = 0.25$, $n = 31$), when fishing mortality had no apparent trend but Lake Trout size declined substantially. In contrast, Lake Trout abundance is positively correlated to the 90% size percentile in the first period ($\rho = 0.86$, $p < 0.001$, $n = 39$), but is negatively correlated in the second period ($\rho = -0.56$, $p = 0.001$, $n = 31$).

When we compared the size distributions of Cisco in the gill net monitoring program with the size distributions of Cisco in Lake Trout stomachs (Fig. 3f), we found that temporal variation in the median and 90th percentile values were essentially identical in both groups, suggesting that Lake Trout were feeding non-selectively on the range of Cisco sizes available in each year. These findings, and the observed sizes of Cisco in Lake Trout stomachs from the 1950's and 1960's (Fig. 3f), suggest that adult Cisco were larger in the initial phase of their expansion into Lake Opeongo than in later years. This gradual contraction in the upper tail of the Cisco size distribution parallels the progressive increase in R observed after ~1975.

Cisco abundance was effectively 0 in the very early 1950's (Fig. 3d,g), when the Cisco invasion of the lake began. Relative abundance estimates for Cisco for the 1970's (mean {1973-76, 79, 80} = 1.8, SE = 0.42), late 1990's (mean {95, 00, 01} = 8.2, SE = 2.2) and 2000's (mean {2007-10} = 4.1, SE = 1.5) show no consistent trend but do suggest that abundance continued to increase into the 1990's. This increase in abundance was accompanied by a ~ 15% decrease in condition factor (1976 vs 2000, Fig. 3g) and a parallel decrease in size at ages 1, 2, 3, and 4 of ~15 % (Fig. 3h). The typical Cisco growth pattern in place in 2000-01 (based on a non-linear least squares fit of a von Bertalanffy growth equation to 103 otolith aged fish) exhibited a value of L_{∞} equal to 14.5 cm, with essentially no somatic growth observed for fish aged 3 to 8 years.

Influences of R on LH Traits

Analysis of the creel data showed that predator/prey mass ratio had a positive effect on the probability of being mature for females and males (Table 2), which means that both A_{mat} and L_{mat} decreased with R (Figure 4a,b). As a consequence, the estimates of the MRNs arising from changes in R were positive (Figure 4c). L_{∞} estimates decreased significantly with increases in R (Table 3, Figure 4d). Males matured earlier, at smaller sizes, but reached larger L_{∞} values than females. The influence of R depended on the period, as evidenced by significant interactions between R and S (Table 2 and 3), being stronger during the second period (Figure 4). The estimated growth curves of the first period were also characterized by a faster rate of approach to L_{∞} (i.e., higher growth coefficient k), as indicated by the positive estimates of parameter θ_3 (Table 3).

The analysis of back-calculated length-at-age data shows similar patterns regarding the effects of predator/prey mass ratio: L_{mat} and L_{∞} both decreased with increases in R (Figure 5). This result is robust to residual heterocedasticity, observed particularly for L_{∞} , as R and length

(both asymptotic and maturation) were still highly significantly correlated according to a Spearman correlation test ($\rho = -0.39$ and -0.60 for L_{mat} and L_{∞} , respectively; $p < 0.001$ for both). A_{mat} was not significantly affected by R ($F_{1,239} = 0.586$, $p = 0.445$), however this could be due to the relatively small variation observed in this variable (A_{mat} SD = 1.15 year). Nonetheless, A_{mat} and L_{mat} were positively related (Figure 5d), configuring the same type of MRN as observed in the creel data analyses, although quantitatively weaker.

When individual R were calculated using stomach contents of fish belonging to the same cohort only, sample size and representativeness were greatly reduced, which compromised the feasibility of some analyses. However, the logistic regressions on maturity could still be carried out on the creel data and showed the same overall pattern of decrease in A_{mat} and L_{mat} with R as in the original analyses (Supplementary Material).

Discussion

Theory

Although the model does not incorporate explicitly the proximate mechanisms generating variation in the LH traits, it is reasonable to hypothesize that the organism would be physiologically able to detect the rate of change in biomass accumulation (i.e., changes in production P), adjusting its development (e.g., through hormonal production) when the rate slows down enough to reach a given threshold. The trait itself (e.g. L_{mat} or L_{∞}) would be plastic, but the threshold (P'_{mat} or P'_{∞}) would be genetically determined and fixed by natural selection (e.g., through genetic accommodation, West-Eberhard 2003) based on the regime of survivorship rates experienced in the past, which, unlike internal states associated with P , are not subject to direct perception by the organism. This type of plastic response would be advantageous given

the high variability of factors determining P within and between populations. This finding suggest that, given fixed values for survivorship s and growing season length D , the associated pair of fixed values for P'_{mat} and P'_{∞} can be used to predict the RNs associated with a broad range of changes in the production function $P(W)$. Thus, L_{mat} would respond plastically in a similar fashion as L_{∞} (e.g. decreasing with predator/prey mass ratio R) but would be dependent on a higher threshold value for $\partial P/\partial W$: in the numerical examples shown in Fig. 2a, $P'_{\text{mat}} = \sim 1.5P'_{\infty}$.

The simulations also indicate that the production parameters with the strongest influence on the optimal threshold (i.e., the value of $\partial P/\partial W$ at the optimal L_{mat}) are those appearing in the exponents of body mass W in Eq. (3): λ , c , h , and β . The implication of this is that any plastic maturation response, based on a fixed developmental threshold P'_{mat} , and which occurs as a consequence of environmental variation in these exponent parameters, would be less optimal when compared to plastic responses to other parameters of the production function (i.e., a_1 – a_4 , e , κ , and R , which make up the linear coefficients of Eq. 3). On the one hand, this result makes sense as the shape of $P(W)$ itself is expected to be more sensitive to variation of its exponents than of its linear coefficients. On the other hand, the exponent parameters are also those expected to have the shortest range of variation in nature: λ can fluctuate as a function of environmental factors and fishing (Petchey and Belgrano 2010; Chu et al. this issue), but it tends to be a relatively conserved property of ecosystems and does not deviate much from 2 (Sprules and Barth this issue); c is determined by hydrodynamic forces interacting with body morphology (Ware 1978; Pettersson and Hedenstrom 2000), so its variation within a population is expected to be small; h measures the geometry of foraging space, which should not change unless the population is forced to drastically change habitat use (e.g., from purely pelagic to purely

benthic); β reflects basic allometric constraints on metabolism (Sibly et al. 2012), and typically lies between 0.67 and 1.0 (Glazier 2010). In contrast, other parameters such as the bioenergetics coefficients a_1 , a_2 , and a_3 , the size spectrum intercept κ , and the predator/prey mass ratio R , can span several orders of magnitude (Giacomini et al. 2013c), depending strongly on environmental factors such as temperature, productivity and community structure.

In our model, A_{mat} can respond to environmental factors in a variety of ways, depending on the combination of production parameters. For the parameter values used here, which are representative of salmonid life histories and commonly observed aquatic communities (i.e., a Sheldon-like size spectrum with $\lambda = 2$, Sprules and Barth this issue), A_{mat} is expected to decrease with increasing R , similar to the response of L_{mat} . So the MRN has a positive slope, which is in line with empirical observations of life history responses to prey size structure, especially in cases involving dwarfism (Roff 1992; Ridgway and Chapleau 1994; Trudel et al. 2001; Pazzia et al. 2002). However, MRNs with negative slope can also occur in the model, depending mainly on the relative magnitudes of the exponents of W in Eq. (3). If the size spectrum is shallow enough (i.e., low λ), the second size-dependent term in Eq. (3), representing metabolic costs, will have a smaller exponent and the production function will be s-shaped. This implies relatively lower production at small sizes so that a disproportionally longer time will be needed to attain a given value of L_{mat} with increases in R , resulting in a negative relationship between L_{mat} and A_{mat} . This dependence between allometric exponents in the production function and the resulting shape of the MRN is consistent with the analysis of Christensen and Andersen (2011), who demonstrated that the existence of optimal MRNs with negative slopes requires relatively large values of the exponent relating body size to metabolic costs. Their analysis differs from ours in that survivorship is size-dependent, whereas here we assume a constant survivorship, which is a

limitation of our model. However, in their model the environment is assumed to be non-seasonal. As a consequence, the predicted optimal life history strategy is determinate growth ($L_{\text{mat}} = L_{\infty}$), which limits its applicability to fish.

Tests of Theory

The observed changes in Lake Trout maturation throughout the time series were essentially concurrent with the observed changes in R . This temporal coincidence is consistent with the hypothesis that the change in maturation is a plastic response to the changes in the individual size-dependent production function that are an inevitable consequence of changes in R . In addition, the observed changes in A_{mat} , L_{mat} , and L_{∞} are qualitatively similar to those predicted from our model of the optimal RNs relating these LH traits to changes in R . The similarity of the observed response at both the population (Figure 4) and individual (Figure 5) level provides strong circumstantial evidence that Lake Trout do exhibit RNs to changes in R that are similar to those predicted by our model.

Some alternative explanations for our results can be discounted. To begin, it is unlikely that the observed variation in R (Figure 3) over the range [30, 110] results simply from the relaxation of gape limitation arising from the observed changes in Lake Trout adult size. Firstly, if changes in prey size were simply a consequence of changes in predator size due to gape limitation constraints (i.e., bigger predators can eat bigger prey), the expected result would be a nearly constant predator/prey size ratio. In contrast, our results show a negative relationship between R and Lake Trout size across time. Second, gape limitation of prey size for Lake Trout establishes a maximum prey length of $\sim 50\%$ of Lake Trout length (e.g. Martin 1970), which corresponds to $R \sim 8$. Observed values for R are always $\gg 8$ (minimum observed ~ 30 , Fig 3), hence throughout the time series Lake Trout were always capable of consuming larger prey when

available, and this capability is confirmed by the fact that changes in the size distribution of Cisco in Lake Trout stomachs closely tracked changes observed in the size distribution of the Cisco population at large (Figure 3f). This latter observation also rules out the possibility that variation in the size of Cisco in Lake Trout stomachs simply reflects a passive response to changes in Lake Trout size according to the gape limitation hypothesis. Similarly, there is little support for the possibility that the changes in Lake Trout sizes were simply a passive response to changes in fishing mortality: (i) correlation between fishing mortality and the 90th percentile of the Lake Trout size distribution was not significant for the period of decreasing size, after 1975; (ii) the parallel changes in L_{mat} that accompanied the observed size distribution changes could not have been the passive product of changes in fishing mortality.

The two pronounced and opposite trends in the values for R , characterizing each half of the period of study, can be attributed to the influence of two distinct factors. The initial decline in R during the first period (1936-1974) was associated with major changes in prey composition, mainly a decrease in importance of Yellow Perch (a relatively small prey), as early noted by Martin (1970), and the emergence of Cisco, a relatively large prey item that became abundant in Lake Opeongo and remained as the main prey item for Lake Trout thereafter. The following increase in R during the later period (1975-2013) could not be attributed to prey species composition, but is most likely to have been caused by density-dependent effects. Adult Lake Trout population abundance increased four-fold during the period following the Cisco introduction (Fig. 3e). An expected effect of increased Lake Trout population is increasing predation pressure on Cisco, which in turn is expected to reduce directly their average size through selective removal of larger individuals from the population. However, Cisco abundance continued to increase through the 1990's and this increase was accompanied by a ~15% decrease

in Cisco condition factor and an overall decrease in juvenile and adult sizes (i.e. ages 1 thru 4) of ~ 15%. This set of results strongly suggests that increased competition in a growing Cisco population, and not simply the direct removal by Lake Trout predation, was responsible for the parallel disappearance, by the late 1990's, of large Cisco from both the Cisco population monitoring data and the Lake Trout diet data (Fig. 2f).

Overall, the available data provides considerable support for our hypothesis regarding a bottom up effect of R on individual lake trout bioenergetics leading to a shift in Lake Trout size structure: relatively larger prey led to enhanced growth and delayed maturation, which in turn is expected to promote changes in the typical individual growth curve, with higher values for both L_{mat} and L_{∞} . Similar bottom-up processes might be occurring with Cisco as well; i.e., changes in Cisco prey availability and size, resulting from intensified intraspecific competition, could have affected Cisco life history and consequently Cisco size distribution. This hypothesis finds additional support in a study by St. Jacques et al. (2005), who reported that the size composition of the zooplankton prey of Cisco shifted, from large-bodied species to small-bodied species after the Cisco introduction to Lake Opeongo. However, more explicit models of size-structured population dynamics (e.g., Hartvig et al. 2011; de Roos and Persson 2013), as well as continued monitoring in Opeongo, will be necessary to disentangle the relative contribution of these processes and to verify whether the system has reached a relatively stable state or whether the observed fluctuations will repeat in the long term as a limit cycle. Additional data on other species can also be useful to understand potential effects of food web interactions on the observed patterns. These interactions may influence lake trout diet and growth through a variety of mechanisms, such as lower food availability caused by competition, though we unfortunately do not have enough data on the abundance of fish species other than Lake Trout and Cisco to test

for more complex hypotheses in a useful manner. However, the fact that Cisco abundance has increased whereas average size (in both catches and stomachs) has decreased, associated with the dominance of Cisco in Lake Trout diet, supports the idea that prey size is more important than prey abundance in explaining the observed temporal changes in Lake Trout growth and life history.

Another temporal component observed in our study was the interaction between R and study period (S): although the effect of R on LH traits was consistently negative, it was more pronounced during the second period. While we recognize that there might be ecological factors affecting this interaction, we think one plausible explanation is based on differences in methodology. During most of the second period (1990-2013), age determination in the creel data was made by otolith reading, whereas in the first period it was entirely made by scale reading. This last method is known to be less accurate for older ages (Shuter et al. 1987), which justified the exclusion of ages >12 years from our growth analyses on the creel data. Together, these factors are expected to introduce a higher amount of noise in the data, making the effect of R less detectable. This is further supported by the results from the individual back-calculated size-at-age data, which are free of some of the biases in the creel data that contribute to the unexplained variance in LH traits: the predicted change in L_{mat} and L_{∞} from back-calculated data (5-20cm) was similar to that predicted from the second period of the creel data, but over a range of $R \sim 4$ times shorter (65-85 compared to 35-105).

Implications

The qualitative match between observed and predicted LH RNs to changes in R encourages further exploration of the mechanism that underlies the RNs identified in our optimization analysis. The RNs are generated by the pair of threshold values for $\partial P / \partial W$ that link

changes in $P(W)$ to changes in the pair of critical sizes ($L_{\text{mat}}, L_{\infty}$) that shape reproductive life. In principle, this structure can be used to derive RNs for any of the parameters in the production function. Like R , many of these parameters are likely to exhibit significant short term variation in natural environments. Such variation would generate changes in ($L_{\text{mat}}, L_{\infty}$) determined by the threshold $\partial P/\partial W$ values ($P'_{\text{mat}}, P'_{\infty}$) illustrated as horizontal dashed lines in Fig. 1b. Although several kinds of RNs have been observed empirically in fish populations (Stearns and Koella 1986; Dunlop et al. 2005; Heino and Dieckmann 2008), our model is one of the first attempts to derive them mechanistically from the types of size-dependent species interactions that are expected to be experienced by fish in nature (e.g., Thygesen et al. 2005; Christensen and Andersen 2011; Giacomini et al. 2013c). In addition, since our model explicitly includes the parameters of the community size spectrum, it provides a vehicle for predicting how changes in the size spectrum itself would affect L_{mat} and L_{∞} . For example, intense harvest of top predators can produce trophic cascades that lead to significant changes in both κ and λ (Petchey and Belgrano 2010; Andersen and Pedersen 2010) – the optimal responses of L_{mat} and L_{∞} to such changes could be directly evaluated using our model.

The theory developed above can be used as a framework to represent LH plasticity in size and trait-based models of population and ecosystem dynamics (e.g., Law et al. 2009; Hartvig et al. 2011; de Roos and Persson 2013; Giacomini et al. 2013a; Rossberg 2013; Andersen et al. this issue). In these models, fish populations are characterized by a species-specific or community-specific predator/prey size ratio, or more generally some variant of the rule ‘big-eat-small’, which permits ontogenetic diet changes and has been shown to be a key determinant of ecosystem structure, stability and vulnerability to anthropogenic impacts (Emmerson and Raffaelli 2004; Brose et al. 2006; Planck and Law 2011; Audzijonyte et al. 2014). However, LH

traits determining reproductive strategies, particularly L_{mat} and L_{∞} , are either ignored or assumed invariant and independent of R and the other parameters that determine individual production. Changes in the distribution of these LH traits are generally assumed to involve evolutionary or compositional changes (e.g., Loeuille and Loreau 2005; Giacomini et al. 2013a; Hartvig and Andersen 2013), without the short-term feedbacks that could occur if phenotypic plasticity was explicitly accounted for. Including these feedbacks is important for predicting population and ecosystem robustness to perturbations (Chase 1999; Miner et al. 2005; Mougi and Kishida 2009; Reed et al. 2010) and this could be accomplished by including P'_{mat} and P'_{∞} as developmental thresholds in such models, thus allowing L_{mat} and L_{∞} to change accordingly with fluctuations in production.

Our findings establish a variety of directions for future research: (i) testing elements of the theory through controlled laboratory experiments designed to assess how changes in $P(W)$ affect the hormonal systems that determine reproductive investment; (ii) exploring the theory to identify the sets of environmental conditions that determine positive vs negative MRNs; (iii) using this natural extension of the bioenergetic representation of growth to include LH plasticity in compatible community models in order to evaluate its potential influence on such phenomena as: (a) the pattern of population fluctuations, (b) the extent of species coexistence, and (c) community responses to those perturbations that change the distribution of food supply, for example selective fisheries that promote cascades down the food web (Andersen and Pedersen 2010).

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References

- Andersen, K.H., and Beyer, J.E. 2006. Asymptotic size determines species abundance in the marine size spectrum. *Am. Nat.*, 168: 54–61.
- Andersen, K. H., and Brander, K. 2009. Expected rate of fisheries-induced evolution is slow. *Proceedings of the National Academy of Sciences*, 106(28): 11657-11660.
- Andersen, K.H., and Pedersen, M. 2010. Damped trophic cascades driven by fishing in model marine ecosystems. *Proc. R. Soc. B*, 277: 795–802.
- Andersen, K.H., Jacobsen, N.S., and Farnsworth, K.D. This Issue. The theoretical foundations for size spectrum models of fish communities. *Can. J. Fish. Aquat. Sci.*
- Audzijonyte, A., Kuparinen, A., and Fulton, E. A. 2014. Ecosystem effects of contemporary life-history changes are comparable to those of fishing. *Marine Ecology Progress Series*, 495: 219-231.
- Bagenal, T.B. 1973. Fish fecundity and its relations with stock and recruitment. *Rapports et Proces-Verbaux des Reunions, Conseil International pour l'Exploration de la Mer*, 164: 186-198.
- Bianchi, G., Gislason, H., Graham, K., Hill, L., Jin, X., Koranteng, K., Manickchand-Heileman, S., Paya', I., Sainsbury, K., Sanchez, F., and Zwanenburg, K. 2000. Impact of fishing on size

- 810 composition and diversity of demersal fish communities. *ICES Journal of Marine Science*,
811 57: 558-571.
- 812 Boisclair, D., and Tang, M. 1993. Empirical analysis of the influence of swimming pattern on the
813 net energetic cost of swimming in fishes. *Journal of Fish Biology*, 42:169-183.
- 814 Boukal, D.S., Dieckmann, U., Enberg, K., Heino, M., and Jorgensen, C. 2014. Life-history
815 implications of the allometric scaling of growth. *Journal of Theoretical Biology*, 359: 199-
816 207.
- 817 Brose, U., Williams, R.J., and Martinez, N.D. 2006. Allometric scaling enhances stability in
818 complex food webs. *Ecology Letters*, 9: 1228–1236.
- 819 Brown, J. H., Gillooly, J. F., Allen, A. P., Savage, V. M., and West, G. B. 2004. Toward a
820 metabolic theory of ecology. *Ecology*, 85: 1771–1789.
- 821 Capitan, J.A., and Delius, G.W. 2010. Scale-invariant model of marine population dynamics.
822 *Phys Rev E*, 81(6): 061901.
- 823 Chandrasekhar, S., 1943. Stochastic problems in physics and astronomy. *Rev. Mod.*
824 *Phys.* 15,0001–0089.
- 825 Chase, J. M. 1999. To grow or to reproduce? The role of life-history plasticity in food web
826 dynamics. *The American Naturalist*, 154(5): 571-586.
- 827 Christensen, A., and Andersen, K. H. 2011. General classification of maturation reaction-norm
828 shape from size-based processes. *Bulletin of mathematical biology*, 73(5): 1004-1027.
- 829 Chu, C., Lester, N.P., Giacomini, H.C., Shuter, B.J., Jackson, D.A. This Issue. Catch-per-unit-
830 effort and size spectra of lake fish assemblages reflect underlying patterns in ecological
831 conditions and anthropogenic activities across regional and local scales. *Canadian Journal of*
832 *Fisheries and Aquatic Sciences*.

- 833 Clark, P., Evans, F. 1954. Distance to nearest neighbor as a measure of spatial relationships in
834 populations. *Ecology* 35: 445–453.
- 835 Confer, J. L., and Blades, P. I. 1975. Omnivorous zooplankton and planktivorous fish. *Limnol.*
836 *Oceanogr.* 20(4): 571-579.
- 837 Conover, D.O., Munch, S.B., and Arnott, S.A. 2009. Reversal of evolutionary downsizing caused
838 by selective harvest of large fish. *Proc. R. Soc. B* 276:2015-2020.
- 839 Czarnoleski, M., and Kozłowski, J. 1998. Do Bertalanffy's growth curves result from optimal
840 resource allocation? *Ecology Letters*, 1: 5-7.
- 841 de Roos, A. M., and L. Persson. 2013. Population and community ecology of ontogenetic
842 development. Princeton University Press, Princeton, NJ.
- 843 Dunlop, E.S., Orendorff, J.A., Shuter, B.J., Rodd, F.H., and Ridgway, M.S. 2005. Diet and
844 divergence of introduced smallmouth bass (*Micropterus dolomieu*) populations. *Can. J. Fish.*
845 *Aquat. Sci.*, 62: 1720–1732.
- 846 Emmerson, M.C., and Raffaelli, D. 2004. Predator-prey body size, interaction strength and the
847 stability of a real food web. *Journal of Animal Ecology*, 73: 399-409.
- 848 Emmrich, M., Brucet, S., Ritterbusch, D., and Mehner, T. 2011. Size spectra of lake fish
849 assemblages: responses along gradients of general environmental factors and intensity of
850 lake-use. *Freshwater Biology*, 56: 2316-2333.
- 851 Engberg, K. C. Jorgensen, E.S. Dunlop, O. Varpe, D.S. Boukal, L. Baulier, S. Eliassen and M.
852 Heino. 2012. Fishing-induced evolution of growth: concepts, mechanism and the empirical
853 evidence. *Marine Ecology* 33:1-25.

- 854 Ernande, B., Dieckmann, U., and Heino, M. 2004. Adaptive changes in harvested populations:
855 plasticity and evolution of age and size at maturation. *Proceedings of the Royal Society of*
856 *London B: Biological Sciences*, 271: 415-423.
- 857 Ernest, S.K.M., Enquist, B.J., Brown, J.H., Charnov, E.L., Gillooly, J.F., Savage, V., White,
858 E.P., Smith, F.A., Hadly, E.A., Haskell, J.P., Lyons, S.K., Maurer, B.A., Niklas, K.J., and
859 Tiffney, B. 2003. Thermodynamic and metabolic effects on the scaling of production and
860 population energy use. *Ecology Letters*, 6: 990–995.
- 861 Forseth, T., Ugedal, O., and Jonsson, B. 1994. The energy budget, niche shift, reproduction and
862 growth in a population. *Journal of Animal Ecology* 63: 116–126.
- 863 Francis, R.I.C.C. 1990. Back-calculations of fish length: a critical review. *J.Fish Biol.*, 36: 883–
864 902.
- 865 Giacomini, H.C., and Shuter, B.J. 2013. Adaptive responses of energy storage and fish life
866 histories to climatic gradients. *J. Theor. Biol.*, 339:100-111.
- 867 Giacomini, H.C., DeAngelis, D.L., Trexler, J.C., and Petrere Jr., M. 2013a. Trait contributions to
868 fish community assembly emerge from trophic interactions in an individual-based model.
869 *Ecol. Model.*, 251: 32-43.
- 870 Giacomini, H.C., Shuter, B.J., deKerckhoe, D.T., and Abrams, P.A. 2013b. Does consumption
871 rate scale superlinearly? *Nature* 493(7434): E1-E2, doi:10.1038/nature11829.
- 872 Giacomini, H.C., Shuter, B.J., and Lester, N.P. 2013c. Predator bioenergetics and the prey size
873 spectrum: do foraging costs determine fish production? *J. Theor. Biol.*, 332:249-260.
- 874 Glazier, D. S. 2010. A unifying explanation for diverse metabolic scaling in animals and plants.
875 *Biological Reviews*, 85(1): 111-138.

- 876 Golet, W.J., Record, N.R., Lehuta, S., Lutcavage, M., Galuardi, B., Cooper, A.B., and Pershing,
877 A.J. 2015. The paradox of the pelagics: why bluefin tuna can go hungry in a sea of plenty.
878 Marine Ecology Progress Series 527: 181-192.
- 879 Gómez-Canchong, P., Blanco, J.M., and Quiñones, R.A. 2013. On the use of biomass size
880 spectra linear adjustments to design ecosystem indicators. Sci. Mar., 77: 257–268.
- 881 Grift, R.E., A.D. Rijnsdorp, S. Barot, M. Heino and U. Dieckmann. 2003. Fisheries-induced
882 trends in reaction norms for maturation in North Sea plaice. Marine Ecology Progress
883 Series. 257: 247-257.
- 884 Hanson, P.C., Johnson, T.B., Schindler, D.E., and Kitchell, J.K., 1997. Fish Bioenergetics 3.0.
885 University of Wisconsin-Madison Center for Limnology and University of Wisconsin Sea
886 Grant Institute.
- 887 Hart, P. J. B., & Connellan, B. 1984. Cost of prey capture, growth rate and ration size in pike,
888 *Esox lucius* L., as functions of prey weight. Journal of Fish Biology, 25(3), 279-292.
- 889 Hartvig, M., and Andersen, K. H. 2013. Coexistence of structured populations with size-based
890 prey selection. Theoretical population biology, 89: 24-33.
- 891 Hartvig, M., Andersen, K.H., and Beyer, J.E. 2011. Food web framework for size-structured
892 populations. Journal of Theoretical Biology, 272: 113–122.
- 893 Heath, D.D., and Roff, D.A. 1996. The role of trophic bottlenecks in stunting: A field test of an
894 allocation model of growth and reproduction in Yellow Perch, *Perca flavescens*.
895 Environmental Biology of Fishes, 45: 53–63.
- 896 Heino, M., and Dieckmann, U. 2008. Detecting fisheries-induced life-history evolution: An
897 overview of the reaction-norm approach Bulletin of Marine Science, 83: 69-93.

- 898 Henderson, B.A., Morgan, G.E., and Vaillancourt, A. 2004. Growth, ingestion rates and
899 metabolic activity of walleye in lakes with and without lake herring. *Journal of Fish*
900 *Biology* 65 : 1270–1282.
- 901 Iles, A.C., and Rasmussen, J.B. 2005. Indirect effects of metal contamination on energetics of
902 yellow perch (*Perca flavescens*) resulting from food web simplification. *Freshwater*
903 *Biology* 50: 976–992.
- 904 Jansen, W. A. 1996. Plasticity in maturity and fecundity of yellow perch, *Perca flavescens*
905 (Mitchill): comparisons of stunted and normal-growing populations. *Annales Zoologici*
906 *Fennici* (pp. 403-415). Finnish Zoological and Botanical Publishing Board.
- 907 Jennings, S. and Blanchard, J. L. 2004. Fish abundance with no fishing: predictions based on
908 macroecological theory. *J. Anim. Ecol.* 73: 632–642.
- 909 Jeschke, J.M., Kopp, M., and Tollrian, R. 2002. Predator functional responses: Discriminating
910 between handling and digesting prey. *Ecol. Monogr.*, 72: 95–112.
- 911 Jobling, M. 1994. *Fish Bioenergetics*. Springer, Fish & Fisheries Series.
- 912 Jørgensen, C., Ernande, B., and Fiksen, Ø. 2009. Size-selective fishing gear and life history
913 evolution in the Northeast Arctic cod. *Evolutionary Applications*, 2: 356–370.
- 914 Juncos, R., Milano, D., Macchi, P.J., Alonso, M.F., Vigliano, P.H., 2011. Response of Rainbow
915 Trout to Different Food Web Structures in Northern Patagonia: Implications for Growth,
916 Bioenergetics, and Invasiveness. *Transactions of the American Fisheries Society* 140, 415–
917 428.
- 918 Kaufman, S.D., Gunn, J.M., Morgan, G.E., and Couture, P. 2006. Muscle enzymes reveal
919 walleye (*Sander vitreus*) are less active when larger prey (cisco, *Coregonus artedii*) are
920 present. *Canadian Journal of Fisheries and Aquatic Sciences* 63: 970–979.

- 921 Kerr, S., and Ryder, R. 1977. Niche theory and percid community structure. *Journal of the*
922 *Fisheries Research Board of Canada*, 34: 1952–1958.
- 923 Kerr, S.R., and Dickie, L.M. 2001. *The biomass spectrum: a predator-prey theory of aquatic*
924 *production*. Columbia University Press.
- 925 Kooijman, S.A.L.M. 2010. *Dynamic Energy Budget Theory for Metabolic Organization*, 3rd
926 *edn*. Cambridge, UK: Cambridge University Press.
- 927 Kozłowski, J. 1996. Optimal allocation of resources explains interspecific life history patterns in
928 *animals with indeterminate growth*. *Proc. R. Soc. B – Biol. Sci.*, 263: 559–566.
- 929 Kozłowski, J., and Teriokhin, A. 1999. Allocation of energy between growth and reproduction:
930 *the Pontryagin Maximum Principle solution for the case of age- and season-dependent*
931 *mortality*. *Evol. Ecol. Res.*, 1: 423–441.
- 932 Law, R., Plank, M.J., James, A., and Blanchard, J.L. 2009. Size-spectra dynamics from
933 *stochastic predation and growth of individuals*. *Ecology*, 90: 802–811.
- 934 Lester, N.P., Shuter, B. J., Venturelli, P., and Nadeau, D. 2014. Life history plasticity and
935 *sustainable exploitation: a theory of growth compensation applied to walleye management*.
936 *Ecol. Applic.*, 24:38-54.
- 937 Lester, N.P., Shuter, B.J., and Abrams, P.A. 2004. Interpreting the von Bertalanffy model of
938 *somatic growth in fishes: the cost of reproduction*. *Proc. R. Soc. Lond. B*, 271: 1625–1631.
- 939 Loeuille, N., and Loreau, M. 2005. Evolutionary emergence of size-structured food webs.
940 *Proceedings of the National Academy of Sciences of the United States of America*, 102(16),
941 5761-5766.

- 942 Lorenzen, K., and Enberg, K. 2002. Density-dependent growth as a key mechanism in the
943 regulation of fish populations: evidence from among-population comparisons. Proceedings
944 of the Royal Society of London B 269:49–54.
- 945 Martin, N.V. 1970. Long-term effects of diet on the biology of the Lake Trout and the fishery in
946 Lake Opeongo, Ontario. J. Fish. Res. Bd. Canada, 27:125-146.
- 947 Martin, N.V., and Fry, F.E.J. 1973. Lake Opeongo: the ecology of the fish community and of
948 man's effects on it. Great Lakes Fish. Comm. Tech. Rep., 24:1-34.
- 949 MATLAB and Statistics Toolbox Release 2015a, The MathWorks, Inc., Natick, Massachusetts,
950 United States.
- 951 Matuszek, J.E., Shuter, B.J., and Casselman, J.M. 1990. Changes in Lake Trout growth and
952 abundance after the introduction of the Cisco into Lake Opeongo, Ontario. Transactions of
953 the American Fisheries Society, 119: 718-729.
- 954 McDermid, J. L., Shuter, B. J., and Lester, N. P. 2010. Life history differences parallel
955 environmental differences among North American lake trout (*Salvelinus namaycush*)
956 populations. Canadian Journal of Fisheries and Aquatic Sciences, 67(2): 314-325.
- 957 Miner, B. G., Sultan, S. E., Morgan, S. G., Padilla, D. K., and Relyea, R. A. 2005. Ecological
958 consequences of phenotypic plasticity. Trends in Ecology & Evolution, 20(12): 685-692.
- 959 Mittelbach, G. G. 1981. Foraging efficiency and body size: a study of optimal diet and habitat
960 use by bluegills. Ecology, 62(5): 1370-1386.
- 961 Mittelbach, G.G., and Persson, L., 1998. The ontogeny of piscivory and its ecological
962 consequences. Canadian Journal of Fisheries and Aquatic Sciences, 55: 1454–1465.
- 963 Morbey, Y.E., and Shuter, B.J. 2013 Intermittent breeding in the absence of a large cost of
964 reproduction: evidence for a non-migratory, iteroparous salmonid. Ecosphere, 4: article 150.

- 965 Mougi, A., and Kishida, O. 2009. Reciprocal phenotypic plasticity can lead to stable predator–
966 prey interaction. *Journal of animal ecology*, 78(6): 1172–1181.
- 967 NOAA Fisheries Toolbox, 2010, VPA / ADAPT version 3.4.5.
- 968 Paloheimo, J.E., and Dickie, L. 1966. Food and growth of fishes .3. Relations among food body
969 size and growth efficiency. *Journal of the Fisheries Research Board of Canada*, 23: 1209.
- 970 Pazzia, I., Trudel, M., Ridgway, M., and Rasmussen, J.B. 2002. Influence of food web structure
971 on the growth and bioenergetics of lake trout (*Salvelinus namaycush*). *Canadian Journal of*
972 *Fisheries and Aquatic Sciences*, 59: 1593–1605.
- 973 Pérez-Rodríguez, A, Morgan, J., Koen-Alonso, M., and Saborido-Rey, F. 2013. Disentangling
974 genetic change from phenotypic response in reproductive parameters of Flemish Cap cod
975 *Gadus morhua*. *Fisheries Research*, 138: 62–70.
- 976 Pérez-Rodríguez, A., Morgan, M. J., and Saborido-Rey, F. 2009. Comparison of demographic
977 and direct methods to calculate probabilistic maturation reaction norms for Flemish Cap cod
978 (*Gadus morhua*). *Evolutionary Applications*, 2(3): 291–298.
- 979 Petchey, O.L., and Belgrano, A. 2010. Body-size distributions and size-spectra: universal
980 indicators of ecological status? *Biol. Lett.* 6: 434–437.
- 981 Peters, R.H. 1983. *Ecological implications of body size*. Cambridge University Press, New
982 York.
- 983 Pettersson, L.B., and Hedenstrom, A. 2000. Energetics, cost reduction and functional
984 consequences of fish morphology. *Proceedings of the Royal Society of London Series B-*
985 *Biological Sciences* 267: 759–764.
- 986 Plank, M.J., and Law, R. 2011. Ecological drivers of stability and instability in marine
987 ecosystems. *Theoretical Ecology*, 5: 465–480.

- 988 Post, J. R., E. A. Parkinson, and Johnston, N.T. 1999. Density dependent processes in structured
989 fish populations: interactions strengths in whole-lake experiments. *Ecological Monographs*
990 62:155–175.
- 991 Quince, C., Abrams, P.A., Shuter, B.J., and Lester, N.P., 2008a. Biphasic growth in fish I:
992 Theoretical foundations. *Journal of Theoretical Biology*, 254: 197–206.
- 993 Quince, C., Abrams, P.A., Shuter, B.J., and Lester, N.P. 2008b. Biphasic growth in fish II:
994 Empirical assessment. *Journal of Theoretical Biology*, 254: 207-214.
- 995 Quinn, T.J., and Deriso, R.B. 1999. *Quantitative Fish Dynamics*. Oxford University Press, New
996 York.
- 997 Reed, T. E., Waples, R. S., Schindler, D. E., Hard, J. J., and Kinnison, M. T. 2010. Phenotypic
998 plasticity and population viability: the importance of environmental predictability.
999 *Proceedings of the Royal Society of London B: Biological Sciences*, 277: 3391-3400.
- 1000 Reuman, D.C., Mulder, C., Raffaelli, D., and Cohen, J. 2008. Three allometric relations of
1001 population density to body mass: theoretical integration and empirical tests in 149 food
1002 webs. *Ecol. Lett.*, 11 : 1216–1228.
- 1003 Rice, J., and Gislason, H. 1996. Patterns of change in the size spectra of numbers and diversity of
1004 the North Sea fish assemblage, as reflected in surveys and models. *ICES J. Mar. Sci.*, 53 :
1005 1214–1225.
- 1006 Ridgway, L.L., and Chapleau, F. 1994. Study of a stunted population of Yellow Perch (*Perca*
1007 *flavescens*) in a monospecific lake in Gatineau Park, Quebec. *Can. J. Zool.*, 72 : 1576-1582.
- 1008 Rijnsdorp, A. D., and van Leeuwen, P.I. 1992. Density dependent and independent changes in
1009 somatic growth of female North Sea plaice (*Pleuronectes platessa*) between 1930 and 1985
1010 as revealed by back-calculation of otoliths. *Marine Ecology Progress Series* 88:19–32.

- 1011 Roff, D.A. 1992. Evolution of life histories: Theory and analysis. Chapman and Hall, New York.
- 1012 Roff, D.A. 2002. Life history evolution. Sinauer, MA.
- 1013 Rossberg, A.G. 2013. Food Webs and Biodiversity: Foundations, Models, Data. Wiley. ISBN 9-
1014 780470973- 55-4.
- 1015 Saint-Jacques, N., Harvey, H.H., Jackson, D.A., 2000. Selective foraging in the white sucker
1016 (*Catostomus commersoni*). Canadian Journal of Zoology-Revue Canadienne De Zoologie
1017 78, 1320–1331.
- 1018 Scott, R. D., and Heikkonen, J. 2012. Estimating age at first maturity in fish from change-points
1019 in growth rate. Marine Ecology Progress Series, 450: 147-157.
- 1020 Sharpe, D.M.T., and Hendry, A.P. 2009. Life history change in commercially exploited fish
1021 stocks: an analysis of trends across studies. Evolutionary Applications, 2: 260–275.
- 1022 Sherwood, G.D., Kovecses, J., Hontela, A., and Rasmussen, J.B., 2002a. Simplified food webs
1023 lead to energetic bottlenecks in polluted lakes. Canadian Journal of Fisheries and Aquatic
1024 Sciences, 59: 1–5.
- 1025 Sherwood, G.D., Pazzia, I., Moeser, A., Hontela, A., and Rasmussen, J.B., 2002b. Shifting gears:
1026 enzymatic evidence for the energetic advantage of switching diet in wild-living fish.
1027 Canadian Journal of Fisheries and Aquatic Sciences, 59: 229–241.
- 1028 Shuter, B.J. 1990. Population level indicators of stress. Pages 145-166 in M.R. Adams, (ed)
1029 Biological Indicators of Stress. American Fisheries Society Symposium No. 8.
- 1030 Shuter, B.J. and Koonce, J.F. 1977. A dynamic model of the western Lake Erie walleye
1031 (*Stizostedion vitreum vitreum*) population. Journal of the Fisheries Research Board of
1032 Canada, 34:1972-1982.

- 1033 Shuter, B.J., Jones, M., Korver, R., and Lester, N. 1998. A General, Life History Based Model
1034 for Regional Management of Fish Stocks: The Lake Trout Fisheries of Ontario. Can. J. Fish.
1035 Aquat. Sci., 55: 2161-2177.
- 1036 Shuter, B.J., Lester, N., La Rose, J., Purchase, C., Vascotto, K., Morgan, G., Collins, N., and
1037 Abrams, P.A. 2005. Optimal life histories and food web position: linkages among somatic
1038 growth, reproductive investment, and mortality. Can. J. Fish. Aquat. Sci., 62: 738–746.
- 1039 Shuter, B.J., Matuszek, J.E., and Regier, H.A. 1987. Optimal use of creel survey data in
1040 assessing population behaviour: Lake Opeongo Lake Trout (*Salvelinus namaycush*) and
1041 smallmouth bass (*Micropterus dolomieu*) 1936-83. Canadian Journal of Fisheries and
1042 Aquatic Sciences, 44 (Supp. II):229-238.
- 1043 Sibly, R.M., Brown, J.H., and Kodric-Brown, A. 2012. Metabolic ecology: a scaling approach.
1044 Wiley-Blackwell.
- 1045 Sprules, W.G., and Barth, L.E. This Issue. Surfing the biomass size spectrum: some remarks on
1046 history, theory, and application. Can. J. Fish. Aquat. Sci.
- 1047 St. Jacques, J-M., Douglas, M. S. V., Price, N., Drakulic, N., and Gubala, C. P. 2005. The effect
1048 of fish introductions on the diatom and cladoceran communities of Lake Opeongo, Ontario,
1049 Canada. Hydrobiologia, 549: 99-113.
- 1050 Stearns, S. C., and Koella, J. C. 1986. The evolution of phenotypic plasticity in life-history traits:
1051 predictions of reaction norms for age and size at maturity. Evolution, 893-913.
- 1052 Stewart, D., Weininger, D., Rottiers, D., and Edsall, T., 1983. An energetics model for Lake
1053 Trout, *Salvelinus namaycush* - application. Canadian Journal of Fisheries and Aquatic
1054 Sciences, 40: 681–698.

- 1055 Sultan, S. E., and Stearns, S. C. 2005. Environmentally contingent variation: phenotypic
1056 plasticity and norms of reaction. Pages 303-332 in B. Hallgrímsson and B.K. Hall (eds.)
1057 *Variation: A central concept in biology*, Academic Press.
- 1058 Tang, M., Boisclair, D., Menard, C., and Downing, J.A. 2000. Influence of body weight,
1059 swimming characteristics, and water temperature on the cost of swimming in brook trout
1060 (*Salvelinus fontinalis*). Canadian Journal of Fisheries and Aquatic Sciences, 57: 1482–1488.
- 1061 Thygesen, U., Farnsworth, K., Andersen, K.H., and Beyer, J. 2005. How optimal life history
1062 changes with the community size-spectrum. Proceedings of the Royal Society of London B,
1063 272: 1323–1331.
- 1064 Trippel, E. A. 1995. Age at maturity as a stress indicator in fisheries. Bioscience 45:759–771.
- 1065 Trudel, M., and Welch, D.W. 2005. Modeling the oxygen consumption rates in Pacific salmon
1066 and steelhead: Model development. Trans. Am. Fish. Soc., 134: 1542–1561.
- 1067 Trudel, M., Tremblay, A., Schetagne, R., and Rasmussen, J.B. 2001. Why are dwarf fish so
1068 small? An energetic analysis of polymorphism in Whitefish Lake Whitefish (*Coregonus*
1069 *clupeaformis*). Canadian Journal of Fisheries and Aquatic Sciences, 58: 394–405.
- 1070 Videler, J.J. 1993. Fish Swimming. Chapman & Hall.
- 1071 Ware, D. 1978. Bioenergetics of pelagic fish - theoretical change in swimming speed and ration
1072 with body size. Journal of the Fisheries Research Board of Canada 35: 220–228.
- 1073 Weatherley, A.H., and Gill, H.S. 1987. The biology of fish growth. Academic Press, New York.
- 1074 Werner, E., and Gilliam, J. 1984. The ontogenetic niche and species interactions in size
1075 structured populations. Annual Review of Ecology and Systematics, 15: 393–425.
- 1076 West-Eberhard, M.J. 2003. Developmental Plasticity and Evolution. Oxford Univ. Press, New
1077 York.

1078 Woodward, G. and Hildrew, A. G. 2001 Invasion of a stream food web by a new top predator. J.
1079 Anim. Ecol. 70: 273–288.

1080 Ylikarjula, J., Heino, M., and Dieckmann, U. 1999. Ecology and adaptation of stunted growth in
1081 fish. Evolutionary Ecology, 13: 433–453.

1082 Yvon-Durocher, G., Montoya, J. M., Trimmer, M., and Woodward, G. 2011. Warming alters the
1083 size spectrum and shifts the distribution of biomass in freshwater ecosystems. Global
1084 Change Biology, 17: 1681–1694.

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Table 1. Model parameters. The values presented are the reference values used to generate the numerical simulations illustrated in Figure 2. For each parameter, the range of values covered in the simulations = reference value $\pm 10\%$.
References: (1) Stewart et al. (1983), (2) Boisclair and Tang (1993), (3) Tang et al. (2000), (4) Brose et al. (2006), (5) Giacomini et al. (2013c), (6) Hanson et al. (1997), (7) Quince et al. (2008b), (8) this study, (9) Trudel and Welch (2005), (10) Giacomini and Shuter (2013), (11) Andersen and Beyer (2006).

Symbol	Description	Values	References
a_1	Coefficient of standard metabolism ($\text{g}^{1-\beta}/\text{day}$)	0.007	1
a_2	Coefficient of active metabolism ($\text{g}^{1-\beta}/[\text{m}^c \text{day}^{1-c}]$)	4×10^{-7}	2,3
a_3	Coefficient of maximum ingestion ($\text{g}^{1-\beta}/\text{day}$)	$a_1 \times 7$	1,4
a_4	Coefficient of distance between individual prey captures	0.5	5
β	Allometric exponent of metabolism and maximum ingestion	0.7	6,7,8
c	Exponent of activity cost	1.6	9
D	Growing season length (days/year)	180	10
e	Assimilation efficiency	0.6	6
h	Habitat dimensionality	2.9*	5
κ	Elevation of prey size spectrum ($\text{g}^{\lambda-1}/\text{m}^h$)	10^{-7}	5, calibrated
λ	Slope of prey size spectrum	2	11
R	Predator/prey mass ratio	$100^\dagger$	5,11
s	Annual adult survivorship	0.8	7

*a value slightly smaller than 3 was used to represent the limitation on depth use imposed on some Lake Trout populations by temperature constraints. The upper boundary for h used for simulations in Fig. 2 was slightly lower than a 10% increase in the reference value (3.19) due to the upper limit of this parameter (3). † this reference value ($\pm 10\%$) was used for simulations shown in Fig. 2, whereas a range from 10 to 1000 was used for Fig. 1.

Table 2. Parameter estimates and associated p-values from the logistic regressions on Lake Trout maturity. Symbols for parameters and effects (predictors) are as in Eq. (4). Sample size (n = number of lake trout individuals) is also presented for each analysis.

Predictor (X)	Sex	Parameter	Effect	Estimate	p-value
Age	Female ($n = 6688$)	δ_0	Intercept	-9.2756	< 0.001
		δ_1	A	1.0039	< 0.001
		δ_2	R	0.0400	< 0.001
		δ_3	S	1.1450	< 0.001
		δ_4	$R*S$	-0.0272	< 0.001
	Male ($n = 6820$)	δ_0	Intercept	-9.8385	< 0.001
		δ_1	A	1.2404	< 0.001
		δ_2	R	0.0292	< 0.001
		δ_3	S	0.8873	< 0.001
		δ_4	$R*S$	-0.0168	< 0.001
Length	Female ($n = 6922$)	δ_0	Intercept	-12.8788	< 0.001
		δ_1	L	0.2480	< 0.001
		δ_2	R	0.0350	< 0.001
		δ_3	S	1.1786	< 0.001
		δ_4	$R*S$	-0.0251	< 0.001
	Male ($n = 7028$)	δ_0	Intercept	-12.9780	< 0.001
		δ_1	L	0.2882	< 0.001
		δ_2	R	0.0162	< 0.001
		δ_3	S	0.7779	< 0.001
		δ_4	$R*S$	-0.0103	0.0172

Table 3. Parameter estimates and confidence intervals from nonlinear regression on length-at-age data. Parameter symbols are as in Eq. (7). The main effect of predator/prey mass ratio (R) is given by θ_1 . Sample size (n = number of lake trout individuals) is also presented for each analysis.

Sex	Parameter	Estimate	95% CI (lower)	95% CI (upper)
Female ($n = 5078$)	L_{∞}^*	75.0343	73.0174	77.0513
	k^*	0.2106	0.1825	0.2388
	A_0^*	1.7218	1.1247	2.3188
	θ_1	0.1091	0.0927	0.1255
	θ_2	0.0672	0.0485	0.0860
	θ_3	0.5522	-0.0682	1.1727
	θ_4	-0.1382	-0.2181	-0.0582
Male ($n = 5645$)	L_{∞}^*	76.8935	74.6405	79.1464
	k^*	0.2029	0.1775	0.2283
	A_0^*	2.0440	1.5482	2.5398
	θ_1	0.1213	0.1047	0.1379
	θ_2	0.0933	0.0746	0.1121
	θ_3	0.9581	0.4474	1.4689
	θ_4	-0.1935	-0.2591	-0.1280

Figure legends

Figure 1. Growth trajectories and life history patterns predicted by optimal resource allocation for changes in the production function $P(W)$ caused by changes in R . Values of other parameters are presented in Table 1. (a) Optimal growth trajectories for different predator/prey mass ratios, $R = 10$ (upper curve) and $R = 500$ (lower curve). The dotted horizontal lines mark the optimal asymptotic and maturation lengths (L_{∞} and L_{mat}), and the black dots mark the onset of reproductive life. (b) The derivative of net production with respect to body mass ($\partial P/\partial W$) as a function of body length (L). The gray curves represent $\partial P/\partial W$ for values of R ranging from 10 (right-most curve) to 1000 (left-most curve); the white dots mark the optimal L_{∞} , which are predicted to occur when the $\partial P/\partial W$ curve intersects a threshold (P'_{∞} , lower horizontal dotted line) defined by annual survivorship s and the duration of growing season D (Eq. 1). The black dots mark the optimal L_{mat} and the respective $\partial P/\partial W$ values, and the upper horizontal dotted line represents their average (P'_{mat}). (c) Optimal L_{∞} and L_{mat} (white and black dots, respectively), presented as a function of R . The continuous lines represent body lengths resulting from the intersection of production derivatives ($\partial P/\partial W$) with the fixed thresholds presented in (b). (d) Maturation reaction norm (MRN) resulting from variation in R – arrow indicates reaction to an increase in R . Black dots are optimal values, and the continuous line is the relationship predicted from using the same fixed threshold (P'_{mat}) presented in (b).

Figure 2. Results from the 100,000 stochastic simulations over the parameter space for the production function $P(W)$. (a) Contour plot of $\partial P/\partial W$ at the optimal L_{∞} (x-axis) and optimal L_{mat} (y-axis) derived from each of the simulations generated over the parameter space for $P(W)$. Colours represent the relative frequency of occurrence of results from individual simulations (1 = highest frequency of occurrence). (b) Coefficients of partial correlation between each model parameter and $\partial P/\partial W$ at the optimal L_{mat} (c) Histogram of the distribution of optimal L_{mat} values

Figure 3. Time series of predator/prey mass ratios (R), Lake Trout size distribution, abundance and stomach contents, and Cisco size, abundance and condition in Lake Opeongo. (a) 25, 50, 75, and 90% percentiles of Lake Trout fork length L (gray areas) at 12 year intervals from 1936 to 2013. The red line is the median R including all available fish prey, and the blue line is the median R including only Cisco as prey. (b) Phase space of the upper end of the Lake Trout size distribution (represented by the 90% percentile) versus R for the same periods presented in

(a). (c) Lifetime estimates of R for individual Lake Trout with available age estimates versus time of capture. Each circle represents fish captured in a given year with a given R , and circle sizes represent relative frequency of occurrence (number of Lake Trout with overlaid values). (d) Running averages of the relative frequency of occurrence of Yellow Perch (red), Whitefish (black), Cisco (blue), and other species or unidentified fish remains (white) in Lake Trout stomachs. (e) Running averages of VPA estimates of Lake Trout abundance (≥ 7 years old, black dots) and fishing mortality (white dots). A time window of 3 years was used to calculate running averages in both (d) and (e). (f) 50% (open dots) and 90% (closed dots) percentiles of body length of Cisco caught in gill nets (red) and found in Lake Trout stomachs (blue). The red dots represent annual percentiles; the blue dots represent percentiles within groups of 15 years (dots centered at 1957), 5 years (dots centered at 1967), and 3 years (all subsequent dots) – time frames were varied to maintain acceptable sample sizes. Lines represent linear regression estimates. (g) Annual values of Cisco CPUE (number of individuals per unit gill net effort, black dots and continuous line) and condition factor (g/mm^3 , white dots and dashed line); years without dots have no available data. (h) Length-at-age of Cisco. Age 1, 2, 3, and 4 are represented by white, blue, red, and green dots, respectively. Black dots represent maximum size estimates using Pauly's method (i.e., taking the average of the 10 largest fish). Lines represent linear regression estimates.

Figure 4. Life history responses of Lake Trout to variation in predator/prey mass ratio R . Green and black lines represent males and females, respectively. Dashed and continuous lines represent data before and after 1975, respectively. (a) Age and (b) Length at maturation predicted by logistic regressions. (c) Maturation reaction norms as predicted by logistic regressions and resulting from the same range of variation in R shown in (a) and (b). The arrows represent the temporal change observed in each period, from $R = 100$ to $R = 30$ in the first period (1936 to 1974) and from $R = 30$ to 100 in the later period (1975 to 2013). (d) Asymptotic lengths predicted by nonlinear regressions for R from 30 to 110.

Figure 5. Results from the analysis of back-calculated size-at-age data. (a) Gray lines are back calculated growth curves, and black dots mark estimated length and year at maturation. (b) Length at maturation L_{mat} and (c) asymptotic length L_{∞} as a function of predator/prey mass ratio R . (d) Maturation reaction norm. The straight lines are linear regression estimates: (b) $L_{\text{mat}} = -0.266R + 68.950$, $r^2 = 0.182$, $F_{1,239} = 42.09$, $p < 0.001$; (c) $L_{\infty} = -$

1161 $0.8337R+130.9213, r^2 = 0.161, F_{1,233} = 40.659, p < 0.001$; (d) $L_{\text{mat}} = 0.550A_{\text{mat}} + 45.482, r^2 = 0.024, F_{1,239} = 5.156, p$
1162 $= 0.024$.

Draft

Figure 1
71x118mm (300 x 300 DPI)

Figure2
235x316mm (120 x 120 DPI)

Figure3
403x352mm (120 x 120 DPI)

Figure 4
70x118mm (300 x 300 DPI)

Figure 5
70x120mm (300 x 300 DPI)