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Maturation reaction norm evolution under varying conditions
of eco-evolutionary change

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Abstract

Probabilistic maturation reaction norms (PMRNs) are commonly used to infer evolution of maturation age and size in wild fish stocks, but how well estimates from phenotypic data actually reflect underlying genotypes is debated. We used an eco-genetic model of a commercially harvested freshwater fish to simulate populations undergoing various levels of fisheries-induced evolution and density-dependent feedback and evaluated effects on the estimation of PMRNs. We estimated PMRNs from phenotypic data sampled from simulated populations (age, length, and maturation status of individuals), as is done for wild stocks, and compared estimates with the known maturation genotypes of individuals in the simulated population. PMRN estimates were robust to changes in the strength of density-dependent growth and high levels of fisheries-induced evolution. However, our ability to detect slower rates of evolution was limited, especially when individuals matured within a narrow range of ages. Furthermore, low numbers of immature or mature individuals within a given age class limited our ability to robustly estimate midpoints for more than one or two ages, despite our large sample sizes ($n = 200$ randomly sampled individuals of each age per year), highlighting a challenge with this approach. Nonetheless, this study suggests that the widely applied method of estimating PMRNs from readily available phenotypic data to detect underlying evolution of maturation schedule is robust to some key factors that vary in wild populations.

37 Introduction

38 The timing of maturation plays an important role in determining the productivity and resilience of
39 fish stocks. Changes in maturation traits, generally towards earlier maturation at smaller sizes, have been
40 observed in numerous exploited fish populations (Trippel 1995; Law 2000; Sharpe and Hendry 2009) and
41 have preceded substantial declines in stock productivity (Trippel 1995; Olsen et al. 2004). Harvesting can
42 lead to maturation-related change demographically, through the truncation of the age and size
43 distribution of the population (Heino and Dieckmann 2008), through density-dependent phenotypic
44 plasticity (Trippel 1995), and from genetic change brought about by selective harvest (i.e., harvest- or
45 fisheries-induced evolution) (Hutchings and Fraser 2008; Heino et al. 2015; Dunlop et al. 2018). Rates of
46 change in the timing of maturation of exploited stocks have been rapid (Trippel 1995; Sharpe and Hendry
47 2009; Devine et al. 2012; Audzijonyte et al. 2013). For example, in stocks where harvest-induced
48 evolution was cited as a contributing factor, rates of change of up to 4% per year have been observed for
49 various measures of maturation timing (Audzijonyte et al. 2013). Changes that have a genetic basis affect
50 a stock's dynamics in fundamental ways (Dunlop et al. 2015), such as by altering rates of recovery from
51 overharvesting (Dunlop et al. 2009; Enberg et al. 2009; Uusi-Heikkilä et al. 2015) or changing the amount
52 of genetic variation present in a population (Pinsky and Palumbi 2014). Fisheries-induced evolution also
53 impacts economic returns from a fishery (Eikeset et al. 2013) and can affect critical biological reference
54 points used in management such as targets and limits for stock biomass and fishing mortality (Heino et al.
55 2013). However, owing to the challenges of demonstrating fisheries-induced evolution in wild
56 populations, disentangling evolutionary from plastic responses to harvest is the focus of intensive
57 research efforts and debate (Browman et al. 2008; Heino et al. 2008; Enberg and Jørgensen 2017).

58 Reaction norms are a fundamental concept in life history theory that describe the range of
59 phenotypes produced by a given genotype under different environmental conditions (Stearns and Koella

1986; Roff 1992; Reznick 1993), and they have been useful in the study of harvest-induced trait changes in fish populations (Heino and Dieckmann 2008). The concept of reaction norms has been applied to studying many different phenotypically plastic life history traits that vary with the environment and can undergo evolutionary change in response to selection. Using reaction norms as a way to understand trait changes in wild fish populations has been most commonly applied to the study of the age and size at maturation, through the estimation of probabilistic maturation reaction norms (PMRNs). Probabilistic maturation reaction norms (PMRNs) are a type of two-trait reaction norm that describe the probability to mature as a function of length and age (Stearns and Koella 1986; Heino et al. 2002; Barot et al. 2004). The probabilistic component of the reaction norm accounts for the intra-individual variation that occurs in the sizes and at maturation within a population (i.e., not all individuals mature at exactly the same age or size even if the environment is the same; Heino et al. 2002). PMRNs estimated from empirical data most commonly show an increase in the probability to mature as the immature fish of a cohort increase in age (corresponding to a decrease in the length at 50% maturation, or a negatively sloped reaction norm), but other PMRN slopes or non-linear shapes are possible (Dieckmann and Heino 2008). The PMRN is a way to empirically describe phenotypic plasticity in the maturation process, with different growth trajectories (and therefore sizes at age) being produced by different environmental conditions such as warmer temperatures or lower food availability. Variation in growth caused by the environment leads to plastic variation in the sizes and ages at maturation.

PMRNs have been extensively used to infer whether an underlying evolutionary change has contributed to observed trends in age or size at maturation in wild fish stocks (Heino et al. 2015). Trends in maturation for some of the world's most prominent fisheries have been interpreted using estimates of PMRNs, including Atlantic cod off the east coast of Canada and European plaice in the North Sea (Jørgensen et al. 2007; Devine et al. 2012). PMRNs can account for much of the plastic variation in size-at-age that occurs in exploited stocks, and thus facilitate detection of underlying evolutionary changes in

maturation (Heino et al. 2002; Dieckmann and Heino 2007). Plastic variation in growth arises from a wide range of environmental and ecological factors that influence fish bioenergetics via consumption, metabolism, and waste production (Wootton 1998); some examples include variation in climate and community composition (Pörtner and Peck 2010), and intraspecific competition leading to density-dependent growth (Lorenzen and Enberg 2002). Changes in age and length at maturation that follow along the same reaction norm are interpreted as plastic responses to variation in growth rates (Fig. 1A), whereas changes in age and length at maturation that result in a shift in the reaction norm itself are interpreted as having a genetic basis (Fig. 1B) (Stearns and Koella 1986; Hutchings 2011).

Interpreting shifts of estimated PMRNs in wild populations remains a challenge. Recent advances have been made in our understanding and methodology by accounting for additional variables that influence the expression of age and size at maturation – for example modeling multi-dimensional PMRNs that include variables such as temperature, social environment, and condition, in addition to body length and age (e.g., Morita et al. 2009, Uusi-Heikkilä et al. 2011; Pauli and Heino 2013). Despite these new developments and continued use of PMRNs to study fisheries-induced evolution, only a few studies have assessed how PMRN estimates could be biased by natural conditions or violations of key assumptions (e.g., Barot et al. 2004, Pérez-Rodríguez et al. 2009, Olsen et al. 2014); none of which have explicitly tested the robustness of PMRN estimates to the density-dependent changes in growth for which the commonly-used estimation procedures were meant to account for (Barot et al. 2004).

Density-dependent growth is a key mechanism regulating fish stocks via competition for resources (Lorenzen and Enberg 2002) and therefore annual variation in population biomass associated with variable recruitment, mortality rates, and harvest are expected to impact size-at-age (Lorenzen 2008). By accounting for variation in growth, PMRNs are often used to determine whether phenotypic changes are plastic responses associated with growth variation (most commonly because harvest can

drastically change population density) or due to evolutionary change. However, assumptions associated with the estimation procedure, namely that mature and immature fish experience similar age- and size-specific growth and survival, could bias PMRN estimates. Previous work found a potential link between compensatory somatic growth and rates of change in estimated PMRNs (Devine et al. 2012), suggesting it is not always possible to disentangle evolution from growth-related plasticity. Furthermore, several studies have reported surprisingly rapid rates of change of PMRNs (Olsen et al. 2004; Devine et al. 2012), raising questions about whether the estimation procedure can robustly reflect actual evolution (Marshall and Browman 2007; Law 2007). For example, Feiner et al. (2015) detected substantial PMRN midpoint shifts within only one decade (evolutionary rate of nearly 2 haldanes) for yellow perch in the Great Lakes. Yet despite the many studies where PMRNs are estimated in wild populations and used to provide evidence of a genetic response to selection, an explicit examination of how different rates and magnitudes of evolutionary change might affect the robustness of PMRNs estimates has not been conducted to our knowledge.

In this study, we use a previously published eco-genetic model (Gobin et al. 2018) to evaluate our ability to estimate PMRNs from phenotypic data and detect harvest-induced evolution of maturation in the presence of ecological and evolutionary change in maturation traits. Eco-genetic models are life history models that include details pertinent to the evolutionary process such as inheritance and genetic trait expression, while also allowing for relevant ecological feedbacks between populations and the environment (Dunlop et al. 2009). Using our model, we simulate changes in growth and maturation within populations using various ecological and evolutionary scenarios. For each scenario, we then estimate the population's PMRN from individual phenotypes as would be done for a wild stock and compare them with the known mean maturation reaction norm genotype of individuals in the simulated population. Given the relevance of growth to the maturation process, we explicitly consider the effects of density-dependent growth on our ability to detect evolution of the PMRN. This allows critical examination

of one of the primary goals of estimating PMRNs—to account for the influence of changing growth through density-dependence on maturation age and size to reveal potential underlying evolutionary responses. In addition, we investigate the effects of different magnitudes of evolutionary change and environmental stochasticity by varying the coefficient of genetic variation and harvest rates in the model.

Methods

Model description

To investigate the effects of ecological and evolutionary change on the estimation of PMRNs, we used an individual-based eco-genetic model developed for Lake Huron lake whitefish (*Coregonus clupeaformis*). Lake whitefish are a commercially harvested fish in the Great Lakes, with Lake Huron contributing the highest catches among the Great Lakes (Brenden et al. 2013). In Lake Huron, lake whitefish have undergone changes over time in population abundance, growth, recruitment, and ages and sizes at maturity (Gobin et al. 2015, 2016, 2018). Lake whitefish ages and sizes at maturation are predicted to evolve in response to harvest, depending on factors such as the minimum size limit and harvest rate (Dunlop et al. 2018, Gobin et al. 2018). Our current study focuses on the ability of the PMRN estimation procedure to detect evolution and does not focus on the ecology of the wild Lake Huron stock.

The eco-genetic model we used in this study was previously used to examine the effects of ecological change on population dynamics and sustainability (Gobin et al. 2016), and how ecological change influences evolutionary responses to size-selective harvest (Gobin et al. 2018). Earlier versions of the model have been used to study life history trait evolution in various fish species including smallmouth bass (*Micropterus dolomieu*) (Dunlop et al. 2007), yellow perch (Dunlop et al. 2015), and Atlantic cod (Dunlop et al. 2009). A description of the eco-genetic modelling approach, including strengths and weaknesses and key considerations are found in Dunlop et al. (2009). The specific eco-genetic model used in this study is described in Gobin et al. (2018) as well as below.

The eco-genetic model follows evolution of the slope and intercept of a linear PMRN. The model runs on an annual timestep during which individuals in the population experience growth, maturation, reproduction, inheritance, phenotypic expression, and mortality. Individuals are born with two genetic traits that describe their tendency to mature, inheriting a slope (S_G) and intercept (I_G) of a linear maturation reaction norm. Each year, environmental variation is added to the expression of the inherited genetic traits following basic quantitative genetic principles (described below), which then determines whether an immature individual will mature that year:

$$L_{mat,t} = I_P + S_P * t, \quad (1)$$

where $L_{mat,t}$ is the body length at a given age (t) when the individual has a probability of 1 to mature, and I_P and S_P are the phenotypes for the intercept and slope of the maturation reaction norm, respectively. Equation 1 determines the lengths over which an individual can become mature as a function of their age and expressed genetic traits. An individual's age and length at the time of maturation represents the individual's "observed" phenotypes for age and length at maturation. Setting an individual's probability of maturing to 1 makes this process deterministic for an individual within a given year, which we did to keep predictions simple by reducing stochasticity in the maturation process and to avoid confounding interactions with the coefficient of genetic variation (Eikeset et al. 2016). However, variation among individuals within the population in the age and size at maturation remains due to genetic variation in maturation traits, environmental variation added to the expression of individual genotypes during inheritance on an annual basis, and density-dependent variation in growth and recruitment (all described below). This inherent among-individual variation and within-individual interannual variation creates a probabilistic "envelope" (or width) in the tendency to mature at the population level and permits the estimation of the population's PMRN.

An individual's genetic traits (I_G and S_G) are inherited following basic quantitative genetics (Dunlop et al. 2009): parameter values of offspring are randomly drawn from a normal distribution centered on the mean value of the parental traits with a variance determined by an assigned coefficient of genetic variation (CV_G). The CV_G is the standard deviation (the square root of the genetic variance) divided by the mean trait value in the initial population and is set between values of 0 and 0.12 (i.e., 0 and 12% of the mean, respectively) based on range of values reported in Houle (1992). Therefore, the CV_G parameter determines the genetic variance (σ_G^2) of PMRN parameters (i.e., slope and intercept values) upon which selection can act thus influencing how much the PMRN can evolve (Dunlop et al. 2015; Houle 1992).

Variation in the expression of genetic traits occurs through assuming a heritability H (the ratio of genetic variation to phenotypic variation) of 0.2 in the initial population (Houle 1992). Phenotypic variation comprises environmental and genetic variation (i.e., $\sigma_P^2 = \sigma_E^2 + \sigma_G^2$), which when substituted into the common heritability equation ($H = \sigma_G^2 / \sigma_P^2$) can be used to determine the level of environmental variation (σ_E^2) in the initial population based on this assumed value of H :

$$\sigma_E^2 = \sigma_G^2(1/H - 1). \quad (2)$$

An individual's phenotypic PMRN traits (I_P and S_P) are drawn from a normal distribution centered on the trait genotype (I_G or S_G) and with variation equal to σ_E^2 (Figure 2). Equation (2) ensures that the assumed heritability is achieved (0.2) in the initial population by scaling the environmental variation by the CV_G assigned for that simulation. Once the simulated population is initialized in the first year of the model, environmental variation is held constant at this value for the remaining years of the simulation and added to the expression of individual's genetic traits annually.

Individuals grow according to the biphasic growth model by Lester et al. (2004). The length (L_t) of a juvenile increases linearly with age (t):

$$L_t = h_t * t, \quad (3)$$

where, h_t is the growth rate. Whereas the length of mature individuals increases in accordance with an asymptotic function described by a von Bertalanffy equation due to energetic investments in reproduction (given by parameter g):

$$L_{t+1} = \left(\frac{3}{3+g}\right) * L_t + \left(3 * \frac{h_t}{3+g}\right). \quad (4)$$

The annual growth rate (h_t) of individuals in the model is density-dependent, being scaled by the population biomass (B) according to Walters and Post (1993):

$$h_t = h_{max}/(1 + A * B), \quad (5)$$

where A represents the loss of food resources due to intraspecific competition and h_{max} describes the maximum growth rate when $B = 0$. During reproduction between pairs of mature individuals, the number of eggs produced by spawning females is determined by a length-fecundity relationship based on empirical data (Gobin et al. 2018; Table S1). The total number of newly born individuals into the population is determined by a Ricker-type stock-recruitment relationship, with the proportion of offspring produced by each parental pair being determined by the length-fecundity relationship

$$\frac{R}{S} = r_{max} * e^{(-\beta * S)}, \quad (6)$$

where R/S is the rate of recruitment of age 0 fish, r_{max} is the maximum recruitment per unit of spawner biomass, S is the spawning biomass, and β describes the strength of density dependence in recruitment (Hillborn and Walters 1992).

Model simulations were run for 200 years, with no harvest for the first 100 years to permit the population to stabilize, followed by 100 years of size-selective harvest with gill nets. Fisheries selectivity

(V_t) follows an empirically derived dome-shaped curve defined by a double-logistic function scaled to the maximum exploitation rate E_{max} (the proportion of fish harvested for the length most vulnerable to the fishing gear):

$$V_t = (1/(1 + \exp(-\delta_1 * (L_t/G - \varepsilon_1)))) * (1 - 1/(1 + \exp(-\delta_2 * (L_t/G - \varepsilon_2))))/V_{max} * E_{max}, \quad (7)$$

where L_t is the fish length, G is the mesh size, V_{max} is the maximum selectivity, and δ_1 , ε_1 , δ_2 , and ε_2 are empirically derived parameters (Zhao and Morbey 2017). In a previous study, we compared predictions for evolutionary rates of change from selectivity curves generated for gill nets with those generated for trap nets, the two types of gear used to commercially harvest lake whitefish in the Great Lakes. Both gears produced downward evolutionary shifts of the PMRN, although gill nets generally induced more evolution compared to trap nets (Gobin et al. 2018). For our current study, we only focused on gill nets because this is the gear most commonly used in Lake Huron (accounting for about 95% of lake-wide harvest). A minimum size limit for the fishery was set to 75% of the initial population PMRN intercept value (Fig. 2C), as in Dunlop et al. (2015). We wanted to choose a minimum size limit that permitted evolutionary change; size limits that are much smaller or larger than the mean size at maturation produce substantially slower rates of change in lake whitefish (Dunlop et al. 2018). The choice of the minimum size limit matters little in the context of our current study because of the size-selectivity curve; there are very few fish that are vulnerable to harvest (equation 4) but smaller than the minimum size limit. By choosing a minimum size limit that at least permits evolution, we could focus on how CV_G , our measure of evolvability, affects PMRN estimation.

237 Parametrization

As in Gobin et al. (2016, 2018), we parameterized the model for lake whitefish, primarily with data for the commercially harvested stock in the southern main basin of Lake Huron. We used data collected by the

Ontario Ministry of Natural Resources and Forestry (OMNRF) from their standardized offshore gillnetting survey (Speers 2013) and the statistical catch-at-age stock assessment model for lake whitefish in the QMA 4-5 management area (based on Ebener et al. 2005), where the largest fishery in the lake is located. Parameters that could not be estimated from available data were obtained from the published literature. A list of parameter values with their sources are provided in Table 1.

Model scenarios

We tested the effects of two growth-related scenarios: a density-independent scenario where growth rate is constant, and a density-dependent scenario where growth rate declines with increasing population density (Fig. 2A). In another set of scenarios, we varied the CV_G parameter such that the evolution of traits was not permitted ($CV_G = 0\%$), was moderate ($CV_G = 6\%$), or was high ($CV_G = 12\%$) (Figs. 2B). We also varied the exploitation parameter (E_{max}) such that populations experienced no ($E_{max} = 0$), moderate ($E_{max} = 0.5$), or high ($E_{max} = 1$) levels of harvest (Fig. 2C). We ran five replicate simulations for each model scenario.

Empirical estimation of PMRNs from simulated data

PMRNs are typically estimated from data collected from fishery monitoring programs. We simulated a basic fishery-independent sampling program in our modeled population by randomly sampling 200 individuals of each age per year for pre-harvest (years 50-55) and during harvest (years 170-175) time periods. The number of individuals sampled was based on sensitivity analyses that were previously performed for the PMRN estimation method (Barot et al. 2004). The pre-harvest years were chosen because population abundance would have been stabilized well before simulation year 50 of the model, while the during harvest years were chosen to allow sufficient time for FIE to occur after the initiation of harvest. We then estimated PMRNs from our sampled individuals to mimic the estimation of PMRNs in a wild stock using the phenotypic data that are typically collected during fishery surveys (age, length and maturation status). We compared these estimated PMRNs to the simulated population's actual known

PMRN, represented by the mean of individual PMRN genotypic traits. In this way, we can test the performance of the estimation method when we vary conditions for growth (density-independent or dependent) and evolvability (levels of CV_G).

We used the indirect method of Barot et al. (2004) to estimate PMRNS, a commonly used procedure which estimates the probability of maturation when the age and size at maturation of individuals is not directly observed. A direct estimation of PMRNs from phenotypic data requires individual-level observations of the age and size at which a fish matures. However, fishery data often provides only a description of maturation status - whether individuals are immature or mature in the year the sample was taken. The sub-sample of fish defined as being mature would therefore include fish that had matured in a previous (unknown) year, not allowing for the direct estimate of the probability of maturing at a given age and size. Note that males and females are identical in our model so are not differentiated in our analysis. Barot et al.'s (2004) indirect approach is the most commonly applied method for estimating PMRNs in the wild (Heino et al. 2015) and has been used for numerous high-profile stocks (Devine et al. 2012) such as northern cod (Olsen et al. 2004) and North Sea plaice (Grift et al. 2007).

The indirect method estimates the maturation probability m for a given age a and size s using available information on the proportion mature at age ($o(a,s)$) (i.e., the maturity ogive) and age-specific growth rate $\Delta s(a)$,

$$m(a, s) = \frac{o(a,s) - o(a-1, s - \Delta s(a))}{1 - o(a-1, s - \Delta s(a))}. \quad (8)$$

First, the proportion of mature individuals is estimated by fitting the following model for each age and cohort separately:

$$\text{logit}(o) = \ln\left(\frac{o}{1-o}\right) = c_0 + s * c_1, \quad (9)$$

where $\text{logit}(o)$ is the logit link function, and c_0 and c_1 are estimated as parameters. Then, annual age-specific growth rates are estimated for each cohort as the difference in mean sizes of consecutive ages. Maturity ogives from equation (9) and age-specific growth rates are then used to estimate probabilities of being mature over the observed range of ages using equation (8) for each cohort. PMRN parameters are derived by fitting logistic regression models to maturation probabilities and sizes for each age and cohort:

$$\text{logit}(m) = d_0 + s * d_1, \quad (10)$$

where d_0 and d_1 are parameters. PMRN midpoints (i.e., the size at which an individual of a given age has a 0.5 probability of maturing) (s_{50}) are calculated by setting $m=0.5$ in equation (10):

$$s_{50} = \frac{-d_0}{d_1}. \quad (11)$$

We estimated PMRN midpoints only for ages and cohorts of our simulated population where the proportion of mature fish fell in the range of 0.2 to 0.8, as having few fish that are either mature or immature can produce unrealistic reaction norm estimates (Barot et al. 2004). Restricting estimates of PMRNs to only those ages with sufficient samples of mature fish reflects what is done in wild populations to obtain robust estimates of PMRNs. In many cases for wild populations, this results in only 1 or a few ages for which PMRN midpoints can be estimated. In the scenarios when CV_G is set to 0 in our simulated populations (removing the possibility of any evolution), the lack of variation among individuals prevents the estimation of a PMRN and we thus just present the population's observed age and size at maturation.

We compare estimated midpoints that are based on individuals' phenotypic maturation traits with midpoints predicted from the true known PMRN of the population, which is calculated as the mean of the PMRN slope and intercept genotypes of all individuals.

Results

Similarity between estimated and known PMRN midpoints indicate the accuracy of the estimation procedure and can be seen by comparing white and grey bars within each scenario in Fig. 3. Assessment of the ability of the estimation procedure to detect fisheries-induced evolution can be seen by comparing left-hand panels with right-hand panels in Fig.3. Density-dependent growth did not affect our ability to accurately estimate PMRN midpoints and detect harvest-induced evolution, which is indicated by the similarity in the results between panels A and B in Fig. 3. PMRN midpoints could be estimated for only one or two ages where there were a sufficient number of both immature and mature individuals, given the population's growth trajectory and maturation genotypes. In most cases, estimated midpoints reasonably reflected midpoints predicted by the simulated population's known PMRNs (population PMRNs shown in Fig. S1). The largest differences between estimated and known midpoints (15-17% of the true population PMRN) was when the amount of fisheries-induced evolution was highest (i.e., $CV_G = 12\%$ and $E_{max} = 1$) and growth was density dependent (Table 2). However, for these scenarios with more evolution, there was a clear separation (a decline in PMRN midpoints) between the estimated midpoints before and after fishing, meaning that the estimation procedure was able to detect some proportion of the genetic change. Under more moderate harvest levels ($E_{max} = 0.5$) when there was less evolution of the PMRN, estimated midpoints were similar before and after fishing and thus were unable to detect the more subtle evolutionary response that had occurred, regardless of the presence or absence of density-dependent growth.

Density-dependent growth, harvest, and evolution of PMRNs altered growth and maturation phenotypes, as expected. Density-dependence reduced growth (Fig.S2), delaying maturation to slightly older ages and smaller sizes (Figs. S3 and S4). PMRNs evolved on a downward shifting trajectory (along the y-axis) in response to harvest (Fig. S1). Growth trajectories then intersect the PMRN at a smaller size and younger

age (Figs. S1 and S2), leading to maturation at younger ages and sizes (Figs. S3 and S4). Density-dependent growth and PMRN evolution together induced the most change in maturation phenotypes (Figs. S3 and S4). This was not owing to differences in PMRN evolution associated with density-dependent growth, as we observed only subtle differences in known PMRNs in density-dependent and -independent scenarios (i.e., comparing panels A and B in Fig. 3 and S1).

Discussion

The estimation of PMRNs from phenotypic data was generally robust to changes that were purely ecological in nature (i.e., due to density-dependent growth causing a purely plastic response) or that had a strong evolutionary component. At the outset of our study, we expected that large shifts in age and size at maturation caused by density-dependent growth and evolutionary responses to harvest (as was seen in our simulations, Figure S2-S4) might bias estimates of PMRNs, owing to the estimation procedure's simplifying assumptions that mature and immature fish experience similar age- and size-specific growth and survival. By simulating a population that could undergo various levels of eco-evolutionary change, we were able to perform a model-based evaluation of the ability of PMRNs estimated from phenotypic data to detect fisheries-induced evolution of genetically based maturation traits. When a population's PMRN underwent substantial evolution in response to high harvest rates, estimated PMRN midpoints captured these shifts. Density-dependent growth did not have a substantial impact on PMRN estimates, or the ability to detect evolutionary change. Several studies have found rapid changes in PMRNs estimated in wild populations (Olsen et al. 2004; Feiner et al. 2015), raising the question as to whether such rapid evolution in fish populations is possible. Our simulations suggest that, not only are such rapid evolutionary changes of genetic-based PMRNs possible for a fish species with a modest generation time (in this case, lake whitefish), but they are also detectable even when density-dependent growth is adding considerable environmental variation to the expression of maturation traits.

Our results provide support for the hypothesis that an important component of the PMRN shifts observed in several wild fish stocks could indeed represent evolution of those stocks' genetic propensity to mature. Numerous studies have reported downward shifts in the PMRNs of fish stocks and have ascribed those shifts to fisheries-induced evolution (reviewed in Heino et al. 2015). However, PMRNs are estimated from phenotypic data (i.e., based on observed traits), leading many to question their ability to detect underlying genetic changes in the maturation tendency of a population (Marshall and Browman 2007; Marshall and McAdam 2007; Kuparinen et al. 2011). Interpreting trends in wild stocks based on phenotypes is challenging, especially given the inherent plasticity in traits like growth. One of the key reasons the PMRN estimation procedure was developed was to account for changes in maturation age and size that were caused by variation in growth (Barot et al. 2004), but to our knowledge, the robustness of the PMRN estimates to varying levels of density-dependent growth and evolution has not been previously examined. Previous studies evaluating the PMRN estimation method have involved either simulated datasets in which maturation was not evolving (e.g., Barot et al. 2004), or observations from wild populations where it is impossible to have certain knowledge of how the population's true PMRNs might be changing over time (e.g. Pérez-Rodríguez et al. 2009, Olsen et al. 2014). Here, we expand upon these previous studies by showing that the estimates derived from our simulated populations were robust to several key conditions found in nature, such as density-dependent growth and recruitment, stochasticity in life history traits, various levels of harvest, and different amounts of evolutionary change. Of course, there are many other factors that could influence estimation of PMRNs in wild populations, but we believe our study is an important step in understanding how key aspects of variability influence our ability to detect evolution.

In some cases, the way in which density dependence altered growth and evolutionary trajectories slightly reduced the accuracy of PMRN midpoint estimates (Fig. S4). This could warrant further investigation in the broader context of large-scale ecosystem change, given that environmental factors

affecting the phenotypic expression of traits and that influence the timing of maturation are non-stationary, and thus change over time and space (Gobin et al. 2018). Here however, reductions in the accuracy of PMRN midpoint estimates when growth was density dependent were sufficiently small that they did not affect our ability to detect more substantial levels of harvest-induced evolution of maturation. It is also the more substantial rates of evolution that are likely the most important to detect from a management perspective. When estimated midpoints did not match up with predictions based on the population's known PMRN, they generally underestimated the length at 50% maturation probability and thus slightly overestimated the amount of fisheries-induced evolution.

Density-dependent growth resulted in slower growth trajectories of individuals (Fig. S2); while at the same time, evolving population PMRNs shifted downwards in response to harvest (Fig. S1). Changes in growth curves and PMRNs would alter where growth trajectories and PMRNs intersect, affecting the proportion of mature and immature individuals at a given age and size. With the indirect estimation method, having too few individuals that are mature or immature at a given age prevents robust parameter estimation for the logistic regressions used to describe maturity ogives shown in equation (9) (Barot et al., 2004). This is why midpoint estimates were limited to only a few ages where the proportion of mature and immature fish fell between 0.2 and 0.8 in the current study, as would be the case for data obtained for a wild population. Nonetheless, proportions that fall closer to these limits could produce midpoint estimates with larger errors than when the ratio of mature to immature individuals is approximately equal. Such errors could become important when the evolution of maturation is low to moderate in magnitude because slight inaccuracies in estimated midpoints relative to a population's known PMRN could limit the ability to detect slower rates of evolution.

Obtaining sufficient sample sizes to estimate PMRNs for individual ages is often challenging in wild populations, as well as in our simulated populations. This is even the case with our optimistic

sampling program that captures fish across all age classes. Barot et al. (2004) previously demonstrated that the indirect estimation method is sensitive to sample sizes below 100 individuals for a given age and cohort. This is due to the effect that smaller sample sizes can have on the proportions of mature and immature individuals sampled at ages when one or the other is relatively rare, leading to non-robust logistic regression parameter estimates as described previously. One of the key ecological reasons that obtaining an adequate sample size can be challenging is that maturation is only observed over the range of ages and sizes determined by a population's life history and the current environmental conditions. Midpoints can only be estimated for ages at which some individuals mature, which may span only a few years for some species or populations and many years for others. Similarly, if population biomass is very high and growth rates resultingly slow, there simply wouldn't be enough individuals maturing at early ages to allow an estimate of maturation probability at younger ages. In our model, there is inter-individual variability in maturation due to density-dependent growth, phenotypic expression (i.e., the addition of environmental variation), genetic variation, and individual-level stochasticity (i.e., probability-based events like mortality). However, it is possible that simplifying our model by implementing a deterministic genetic-based maturation threshold rather than having a probabilistic width at the individual level might have reduced the inter-individual variation in maturation timing, resulting in a narrower range of observed maturation ages. Nonetheless, our simulations remain relevant, given that most empirical studies are only able to estimate midpoints for one or a few ages due to the challenges discussed above (e.g., Wang et al. 2008, Hidalgo et al. 2014, Feiner et al. 2015). This means that important information relating to the slope or shape of PMRNs when interpreting shifts in PMRN midpoints over time or space is often limited in empirical studies. Interpretation of trends with few PMRN midpoints is further complicated by the fact that the ages for which we are able to estimate midpoints also shifts with changes in the age and size at maturation (as seen in our simulations where evolvability and harvest were high as well as empirically in wild populations), due to effects on the ratio of mature to

immature individuals. These sample size issues continue to be a challenge in the estimation procedure, both for wild and simulated populations.

The traditional PMRN estimation procedure by Barot et al. (2004), as well as our model, do not include other variables besides growth that may affect the maturation process. Other factors shown to influence maturation in fish include temperature (Dhillon and Fox 2004; Kuparinen et al. 2011; Tobin and Wright 2011), body condition (Uusi-Heikkilä et al. 2011), male and female social cues (Diaz Pauli and Heino 2013), growth history (Morita and Fukuwaka 2006; Diaz Pauli and Heino 2013; Siegel et al. 2018), and population density (Hunter et al. 2015, 2019). These additional factors might cause changes in maturation phenotypes and estimated PMRNs even if no underlying fisheries-induced evolution occurred (Kraak 2007). By potentially increasing variation in maturation phenotypes, additional environmental variation could have implications for the ability of the PMRN estimation procedure to detect underlying change of a population's PMRN. Additional environmental variables may affect traits in directional ways either through phenotypic plasticity or through selection. Fortunately, new estimation techniques for PMRNs have emerged (Hunter et al. 2015, 2019), including 3-dimensional PMRNs, which include a third variable (e.g., temperature) in addition to size and age (Grift et al. 2007). However, these newer methods require data that are not collected as routinely as individual age and size data, and researchers continue to rely on estimates of two-dimensional PMRNs based solely on length and age (e.g., Kokkonen et al. 2015; Gislason et al. 2019). Other uncertainties relating to the prevailing methods of estimating PMRNs are under investigation, including the role of individual growth histories in influencing maturation (Morita and Fukuwaka 2006; Pauli and Heino 2013; Siegel et al. 2018) and the effects of non-random sampling on estimates (Sahashi and Morita 2015).

Overall, our study demonstrates that PMRN estimates can be used to detect rapid evolution of maturation in response to harvest in the presence of key ecological feedbacks. Given the difficulty of

detecting fisheries-induced evolution using direct genetic approaches (Casey et al 2016; Jakobsdóttir et al. 2011), PMRNs remain the best available option for analyzing phenotypic changes in maturation in natural populations (Heino et al. 2015) and have become a fundamental tool for modeling the evolution of maturation (e.g., Dunlop et al. 2007, 2009, 2015; Enberg et al. 2009; Mollet et al. 2016; Dercole and Della Rossa 2017). The current study did not consider variables that can influence maturation traits beyond growth (e.g., temperature, body condition), which is an important next step in understanding the potential limitations of indirect PMRN estimation for evaluating ecological and evolutionary change in maturation traits for wild populations. Given their substantial flexibility (Dunlop et al. 2009), eco-genetic models and simulation-based approaches similar to those applied here could be particularly useful for further investigations in these areas.

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676 Table 1. Parameter values and sources used in the individual-based eco-genetic model for lake
 677 whitefish in the southern main basin of Lake Huron.

Description	Symbol	Source	Value
Mean PMRN* intercept [†] (cm)	I	1	42.7
Mean PMRN slope [†] (cm/yr)	S	1	-1.77
Heritability [†]	H	2	0.2
Coefficient of genetic variation	CV_G	2	0, 0.06, 0.12
PMRN width (defining 0.25 and 0.75 probabilities)	ω		0
Mean maximum growth potential (cm/yr)	h_{max}	3	14.4
Intraspecific competition food loss rate (kg ⁻¹)	A	3	6e-5
Reproductive investment [‡]	g	4,5	0.346
Mean maximum recruitment potential (# of recruits)	r_{max}	3	9
Ricker stock-recruitment parameter (kg ⁻¹)	β	3	2e-4
Constant in length-weight relationship (kg/cm ^d)	c	6	3.318e-6
Exponent in length-weight relationship	d	6	3.29
Exponent of length-fecundity relationship	j	7	3.83
Slope of length-fecundity relationship (eggs/mm ^j) ^{-10^3}	i	7	1.1e-6

Background natural mortality [§] (year ⁻¹)	M_B	8	0.30
von Bertalanffy asymptotic length (cm)	L_∞	8	60.0
Brody's growth coefficient (in von Bertalanffy growth model) (year ⁻¹)	K	8	0.25
Water temperature (°C)	T	8	6
Wounding probability logistic model coefficient	p	9	8.60
Wounding probability logistic model coefficient (mm ⁻¹)	n	9	0.01
Lamprey mortality probability		10	0.75
Double logistic gillnet selectivity parameter (mm ⁻¹)	δ_1	11	5.84
Double logistic gillnet selectivity parameter (mm)	ε_1	11	4.19
Double logistic gillnet selectivity parameter (mm ⁻¹)	δ_2	11	2.19
Double logistic gillnet selectivity parameter (mm)	ε_2	11	4.74
Gillnet selectivity mesh size (mm)	G	11	114
Fishery minimum size limit (mm)		12	320
Maximum exploitation rate	E_{max}		0, 0.5, 1

[§]Probabilistic maturation reaction norm

[†]Values are defined for the initial population and are then free to change through time

[‡] Calculated by multiplying the gonadosomatic index (gonad mass divided by body mass) by a conversion factor of 1.73 to account for differences in the energetic content of gonad and somatic tissues (Lester et al. 2004).

[§] Background natural mortality was estimated from the relationship between von Bertalanffy growth model parameters and mean annual temperature (Ebener et al. 2005; Pauly 1980)

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Table 2. Percent differences between mean midpoints estimated from observed phenotypic maturation traits and those predicted from the true known population PMRN. Differences between midpoints (± 1 SD) are shown before and after fishing at three harvest levels (none – $E_{\max} = 0$; moderate – $E_{\max} = 0.5$, high – $E_{\max} = 1$), when growth was density-independent and density-dependent, for two levels of evolvability (moderate – $CV_G = 0.06$; high – $CV_G = 0.12$). Negative values reflect that estimated midpoints underestimated the true known midpoints by the percentage shown, whereas positive values reflect overestimation of the true known midpoints by the percentage shown.

	evolvability	harvest	age	density-independent growth (%)	density-dependent growth (%)
Before fishing	moderate	none	3	1.3 (± 1.5)	0.3 (± 1.2)
		moderate	3	1.0 (± 3.8)	0.2 (± 3.5)
		high	3	0.3 (± 0.8)	-0.4 (± 0.9)
	high	none	2	2.7 (± 4.8)	-0.6 (± 4.7)
			3	-6.8 (± 9.1)	-9.9 (± 8.8)
		moderate	2	0.4 (± 2.3)	-3.0 (± 2.4)
			3	-7.1 (± 7)	-10.5 (± 6.8)
		high	2	-1.3 (± 3.3)	-4.2 (± 3.5)
			3	-8.3 (± 3.5)	-11.1 (± 4)
After fishing	moderate	none	3	3.6 (± 0.2)	0.7 (± 0.8)
		moderate	3	0.4 (± 2.1)	6.1 (± 1.9)
		high	2	1.0 (± 0.7)	1.2 (± 1.4)
	high	none	2	-0.6 (± 1.6)	-10.8 (± 1.5)
			3	-7.1 (± 6.4)	-17.3 (± 5.3)
		moderate	2	2.7 (± 2.7)	5.0 (± 4.4)
			3	-5.5 (± 13.7)	-3.2 (± 14.2)
		high	1	-8.8 (± 6.8)	-14.9 (± 6.3)
			2	-10.8 (± 2.3)	-17.3 (± 2.3)

Figure captions

Figure 1. Description of a probabilistic maturation reaction norm (PMRN). A) The PMRN describes the genetic propensity to mature as a function of an individual's age and length. Changes in growth (for example, caused by changes in population abundance) produce changes in the observed ages and lengths at maturation that are due to phenotypic plasticity. An individual with faster growth (dashed line) has a tendency to mature at an earlier age and larger body length (black circle) than an individual with a slower growth trajectory (dotted line) who tends to mature at a later age and smaller body length (black square). The midpoint of the PMRN represents the length for a given age at which the probability to mature is 50% (solid black line). Contour lines for probabilities of 25% and 75% are also shown (grey dashed lines above and below the midpoint). B) Changes in the position of the PMRN (grey solid line) relative to the original PMRN (black solid line) reflect an underlying evolutionary change in the timing of maturation. In this case, the observed maturation ages and lengths (grey circle and square) in the population have shifted as a result of changes in the genetic tendency to mature rather than plastic changes in growth. Shifts in the timing of maturation further influence growth trajectories after maturation (grey dashed and dotted lines).

Figure 2. Eco-genetic model scenarios used to evaluate the accuracy of empirically estimating probabilistic maturation reaction norms. A) Maximum growth potential in density-dependent and -independent growth scenarios. B) The probability distribution ($P(x)$) of genotypic traits (maturation reaction norm slope and intercept) when evolvability is high ($CV_G=0.12$), moderate ($CV_G=0.06$), and when evolution is constrained ($CV_G=0$). C) Size-selectivity curves for high ($E_{max} = 1$), moderate ($E_{max} = 0.5$), and no harvest ($E_{max} = 0$); the dotted line shows the minimum size limit for the fishery (32 cm).

Figure 3. Comparison between estimated (white bars) and known (grey bars) probabilistic maturation reaction norm midpoints. Growth is either A) density independent or B) density dependent. Only ages for which midpoints could be estimated are shown. Fishing began in year 100 and continued annually at three different harvest levels (none – $E_{\max} = 0$; moderate – $E_{\max} = 0.5$, high – $E_{\max} = 1$) and for a population with two different evolvabilities (moderate – $CV_G = 0.06$; high – $CV_G = 0.12$). Midpoints are shown for two time periods (before fishing, years 50-55) and after fishing began (after fishing, years=170-175). Values are means of 5 independent simulation runs and error bars are the standard deviations around the means.

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