

Effects of ambient oxygen and size-selective mortality on growth and maturation in guppies

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Growth, onset of maturity and investment in reproduction are key traits for understanding variation in life-history strategies. Many environmental factors affect variation in these traits, but for fish, hypoxia and size-dependent mortality have become increasingly important because of human activities, such as increased nutrient enrichment (eutrophication), climate warming and selective fishing. Here, we study experimentally the effect of oxygen availability on maturation and growth in guppies (*Poecilia reticulata*) from two different selected lines, one subjected to positive and the other negative size-dependent fishing. This is the first study to assess the effects of both reduced ambient oxygen and size-dependent mortality in fish. We show that reduced ambient oxygen led to stunting, early maturation and high reproductive investment. Likewise, lineages that had been exposed to high mortality of larger-sized individuals displayed earlier maturation at smaller size, greater investment in reproduction and faster growth. These life-history changes were particularly evident for males. The widely reported trends towards earlier maturation in wild fish populations are often interpreted as resulting from size-selective fishing. Our results highlight that reduced ambient oxygen, which has received little experimental investigation to date, can lead to similar phenotypic changes. Thus, changes in ambient oxygen levels can be a confounding factor that occurs in parallel with fishing, complicating the causal interpretation of changes in life-history traits. We believe that better disentangling of the effects of these two extrinsic factors, which increasingly affect many freshwater and marine ecosystems, is important for making more informed management decisions.

Key words: Eutrophication, fishing selection, hypoxia, life history, *Poecilia reticulata*, water management

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Introduction

Maturation determines the beginning of the reproductive part of an individual's life cycle and is costly in terms of survival and energy. The age and size at which an individual matures are therefore key life-history traits. Growth determines the relationship between age and size, with the latter also being a key determinant of survival and fecundity. Thus, studying the effects of different extrinsic factors on growth and maturity is important for understanding the variation in life-history strategies (Roff, 1992; Stearns, 1992, 2000).

Many different environmental factors, such as food availability, temperature, oxygen and presence of predators, affect the acquisition and allocation of resources to growth, maturation and reproduction (Berner and Blanckenhorn, 2007; Enberg *et al.*, 2012). Two factors affecting life-history traits are of particular interest in fishes, namely oxygen and size-dependent mortality. Oxygen is one of the most critical physical constraints for aquatic animals (Ross, 2000; Pauly, 2010): water is a dense, viscous medium that contains little oxygen in comparison to air; only small quantities of oxygen can be dissolved, and respiratory areas do not grow as fast as body weight (Pauly, 1981, 2010). Oxygen demand is proportional to the rate of metabolism and increases with, e.g. body size and stress. Low-oxygen conditions occur naturally in many closed water bodies and in the oxygen minimum zones of the World Ocean, but oxygen depletion is also becoming increasingly prevalent in freshwater and marine ecosystems because of increasing eutrophication and temperature (Diaz and Rosenberg, 2008; Doney *et al.*, 2012; Jenny *et al.*, 2016). Importantly, temperature plays a dual role: increasing temperature reduces the solubility of oxygen, while in ectotherms, it also increases the metabolic demand for oxygen (Pörtner and Knust, 2007; Holt and Jørgensen, 2015).

Similar to oxygen depletion, size-dependent mortality occurs naturally but can be influenced by human activities. Size-dependent natural mortality is driven by the presence of predators that commonly prey more heavily on smaller size classes, i.e. it is negatively size selective (Lorenzen, 1996; Sogard, 1997; Gislason *et al.*, 2010). In contrast, fishing most often targets large fish (i.e. it is positively size selective). Fishing pressure has increased since the middle of the past century, mainly targeting large individuals and higher trophic levels (Pauly *et al.*, 2002; Kolding *et al.*, 2016). Importantly, reduced oxygen levels and increased size-selective fishing co-occur in many aquatic ecosystems, for instance in Lake Victoria (Kolding *et al.*, 2008b), on the Swedish west coast (Kattegat and Skagerrak; Cardinale and Svedäng, 2004) and in the northern Benguela system (Utne-Palm *et al.*, 2010).

Reduced oxygen and overexploitation cause reduced abundance and recruitment in demersal fish (Diaz and Rosenberg, 2008). Low oxygen saturation in water is a proximate factor

driving reduced asymptotic maximal size, because the limited oxygen available is allocated to maintenance rather than somatic growth (Pauly, 1981, 2010; van Dam and Pauly, 1995; Chabot and Claireaux, 2008). Little is known about the effect of hypoxia on reproduction, but extreme levels of hypoxia can impair it (Wu *et al.*, 2003; Landry *et al.*, 2007; Chabot and Claireaux, 2008). However, it is predicted that at moderate levels of hypoxia, stunting is caused by earlier maturation and increased reproductive investment at early ages (Kolding, 1993; Kolding *et al.*, 2008a). However, similar changes in maturation and post-maturation growth are expected from evolutionary change caused by fisheries-induced selection (Heino *et al.*, 2015).

Despite the fact that a low oxygen level and fishing may co-occur and drive similar changes in life-history traits, little effort has been made to study their joint effect (Kolding *et al.*, 2008b). Studying the combined effect of several factors is crucial to gain a better understanding and inform management and conservation plans of natural resources and fish populations in particular (Jackson *et al.*, 2016). For instance, Kolding *et al.* (2008b) concluded that low oxygen, rather than overfishing, was the most important threat for Nile perch (*Lates niloticus*) in lake Victoria. Likewise, the reduction in individual size and maturation observed in Nile perch (Mkumbo and Marshall, 2015) and Dagaa (*Rastrineobola argentea*; Sharpe *et al.*, 2012) in Lake Victoria could be driven by hypoxia. Crucially, mitigating actions depend on the driver. If reduced oxygen is the culprit, then changing the environment is needed (Rabalais *et al.*, 2007) and, in the best case, the management response is rapid (Beutel and Horne, 1999). In contrast, if dwarfing reflects evolutionary adaptation to fishing, then the fishing pattern needs to be changed, and even in the best case the response is likely to be slow (Law, 2000; Heino *et al.*, 2015).

Here, we test how oxygen level affects maturation schedules and growth in fish populations exposed to different size-selective mortality regimes. We expect that both low oxygen and exposure to positive size-selective fishing result in earlier maturation and reduced growth. This is the first study jointly to assess the effects of reduced ambient oxygen and size-dependent mortality in fish. Thus, little is known about the relative importance of these factors in driving changes in key life-history traits. For this purpose, we used populations of guppies (*Poecilia reticulata*) in laboratory conditions. This model species was also used to demonstrate von Bertalanffy's theory of growth von Bertalanffy (1938), to study the effect of fishing on population dynamics (Silliman and Gutsell, 1958) and to assess the effect of predatory size-selective mortality on life-history traits (Reznick and Ghalambor, 2005). Moreover, similar laboratory experiments have been shown to be useful to inform conservation and management plans (see, e.g. Stockwell and Weeks, 1999; Conover and Munch, 2002; Reznick and Ghalambor, 2005; Diaz Pauli and Heino, 2014).

Materials and methods

We used guppies from a life-history experiment designed to study evolutionary consequences of size-selective fishing. The test fish were first-generation (F1) offspring from six replicate laboratory populations that had experienced size-selective mortality for 3.1 years (approximately four generations). These populations represent two treatments, with three replicates each, as follows: (i) positive size-selected line, in which large individuals [>16 mm standard length (SL)] were removed from the population every sixth week; and (ii) negative-size selected line, in which individuals smaller than 16 mm were removed at equal intervals.

Twenty females per population were housed together in 10 litre tanks and feed *ad libitum* with newly hatched *Artemia salina* in the morning and fish flakes (tetraMin, Tetra) in the afternoon. Tanks were checked twice a day for newborns, which were collected and immediately transferred to 2 litre individual isolation aquaria, where they were randomly assigned to one of two oxygen treatments: (i) high oxygen, with $95 \pm 5\%$ oxygen saturation, corresponding to $\sim 7.9 \pm 0.4$ mg l⁻¹; or (ii) low oxygen, with $64\% \pm 6\%$ oxygen saturation (5.3 ± 0.5 mg l⁻¹). All tanks were covered with a tightly fitting piece of Styrofoam over the whole water surface. The cover prevented surface breathing and minimized gas exchange with the atmosphere. In the high-oxygen treatment, high oxygen saturation was maintained with an air stone. This resulted in a 2×2 full factorial experiment, with oxygen level and inherited background (past size-selective mortality) as the treatments. Ten males and 10 females from each of the six populations were assigned to each oxygen treatment, resulting in a total of $n = 240$ fish (1:1 sex ratio).

Test fish were maintained in individual isolation at a constant temperature of $25 \pm 0.5^\circ\text{C}$ and under a 12 h–12 h light–dark regime. During the first 2 weeks, each fish was fed daily $38 \pm 6 \mu\text{l}$ of 3% solution of living filtered *Artemia salina*. At 2 weeks of age, this was increased to $76 \mu\text{l day}^{-1}$, and at 4 weeks of age it was increased to $114 \mu\text{l}$, which was maintained until the fish reached maturation and the experiment was terminated.

Fish were anaesthetized in a 0.3 g l^{-1} solution of metacaine, measured for SL and weight, and assessed for maturation weekly. Non-invasive assessment of maturation is reliable only in males; this is achieved by following the development of the gonopodium (modified anal fin used in insemination). Initiation of maturation is indicated by the increase from nine to ten segments in the third ray of the anal fin, while complete maturation is marked by the growth of the fleshy hood over the tip of the gonopodium and the number of segments in the third ray being >27 (Turner, 1941; Reznick, 1990). Gonopodium development is correlated with the development of the gonadotrophic zone in the adenohypophysis and the maturation of the testis (Kallman and Schreibman, 1973; Schreibman and Kallman, 1977; Greven, 2011). The initiation of maturation stage is correlated with

initial enlargement of the testis and proliferation of spermatogonia and, possibly, spermatocytes (van den Hurk, 1974; Koya *et al.*, 2003). At the completion stage, there are several layers of spermatogonial cysts, sperm cells and developed testicular ducts with enzyme activity, and spermatozeugmata (sperm bundles) are present (Schreibman *et al.*, 1982; Koya *et al.*, 2003). We consider the initiation of maturation to be a good representation of male maturation ‘decision’ in guppies; it is the time when they commit to maturation, reflecting more accurately the factors that affect maturation than the final maturation stage (Tobin *et al.*, 2010; Harney *et al.*, 2012; Diaz Pauli and Heino, 2013). Therefore, in the present study we assessed the effect of oxygen and size selection on the initiation of maturation, from now on referred to as maturation. Female maturation cannot be assessed non-invasively; therefore, from female fish we obtained only growth data, from which we later estimated maturation (see next subsection). Females were kept in the experiment until 2 weeks after a male from the same brood reached the last stage of maturation.

Statistical analysis

Growth

All analyses were performed in R (version 3.2.4; R Core Team, 2016). To assess treatment effects on individual growth, we used the biphasic growth model of Boukal *et al.* (2014), which is derived from the model by Quince *et al.* (2008), within the ‘nlme’ R package (version 3.1–125; Pinheiro *et al.*, 2016). The model provides a mechanistic description of somatic growth pre- and post-maturation, based on the principles of allometry and energy allocation. Surplus energy acquisition rate, which is equal to maximal potential somatic growth, is related to somatic weight, W , by the coefficient c and the allometric exponent β , as follows:

$$\frac{dW}{dt} = cW^\beta \quad (1)$$

Assuming that juveniles allocate surplus energy only to growth (reproductive investment $r_a = 0$), the juvenile growth curve for weight at age a is as follows:

$$W_a = {}^{1-\beta}\sqrt{W_0^{1-\beta} + c(1-\beta)a} \quad (2)$$

The post-maturation (adult) growth curve takes into account reproductive investment (r) for mature individuals, i.e. for $a \geq a_{\text{mat}}$:

$$W_a = {}^{1-\beta}\sqrt{R^{a-a_{\text{mat}}}(W_0^{1-\beta} + Hb^{1-\beta}a_{\text{mat}}) + \frac{RHb^{1-\beta}}{1-R}(1-R^{a-a_{\text{mat}}})}, \quad (3)$$

where $H = c(1-\beta)b^{-(1-\beta)}$, $R = 1/[1 + (1-\beta)r]$, and W_0 is weight at birth.

Growth curves were estimated for males and females separately. Weight at birth was affected neither by sex ($F_{2,13,1} = 1.68$, $P = 0.19$) nor by size-selection treatment ($F_{4,3,1} = 0.07$, $P = 0.79$) according to a linear mixed-effect model with population as random factor. These linear mixed models were performed with lme4 R package (version 1.1–11; Bates *et al.*, 2015). P -values and degrees of freedom were obtained with the R package 'lmerTest' (version 2.0–29; Kuznetsova *et al.*, 2015). Therefore, weight at birth $W_0 = 0.007$ g was used for both males and females. In males, age at maturation (a_{mat}) was included in the model as a known individual-specific variable (age at which initiation of maturation occurs), but in females it was estimated as a model parameter. Reproductive investment (r) and the coefficient in the allometric growth rate–weight relationship (c) were estimated for both males and females, whereas the allometric exponent in the growth rate–weight relationship (β) was estimated for males but kept constant for females as $\beta = 0.8$ because simultaneous estimation of β and a_{mat} was not possible. Initial exploration of our data showed that $\beta = 0.8$ was the most appropriate value for our data, and similar values have been suggested by Boukal *et al.* (2014).

The parameters were estimated with a non-linear mixed-effect model in the R package 'nlme' (Pinheiro *et al.*, 2016), with fish identity (ID) as random factor for r and c for both males and females. Including fish ID as random factor for β and a_{mat} for males and females, respectively, did not improve the models [for males the change in the Akaike information criterion (ΔAIC) = 6.1, likelihood ratio statistic = 0.09, $P = 0.99$; and for females, ΔAIC = 6.0, likelihood ratio statistic = 0.0002, $P = 1$]. Oxygen, size-selection line and their interaction were tested as fixed effects on r , c and β for males and r , c and a_{mat} for females. The model that yielded the lowest AIC was considered the best approximating model, i.e. the model that best described the data. We also discuss models that differ from the best ranked-model with AIC values >2 ($\Delta_i = \text{AIC}_i - \text{AIC}_{\text{best}}$), as these are considered essentially as good as the best model (Burnham and Anderson, 1998). We also calculated the probabilities of a model being the best model, referred to as Akaike weights (w_i). Notice that the approach chosen here does not involve significance testing of the model parameters.

Maturation

Maturation in males is described by the probabilistic maturation reaction norm (PMRN; Heino *et al.*, 2002), estimated with generalized linear mixed models with binomial error distribution using the lme4 package in R (version 1.1–11; Bates *et al.*, 2015). Fish ID nested within population was included as a random factor, whereas age, weight, oxygen, size-selection line and all their first-order interactions were included as fixed effects. As for the growth models, we used the AIC to select the final model. The logistic curve for the probability of maturation is given by the following equation:

$$\text{logit}(p) \sim c_0 + c_1a + c_2w + c_3o + c_4s + \dots c_n, \quad (4)$$

where $\text{logit}(p) = \log_e[p/(1 - p)]$ is the link function, c_0 is the intercept, and c_1 to c_n are the regression parameters of the model for the different explanatory variables (age a , weight w , oxygen o , size-selection line s , interactions, etc.). To facilitate the interpretation of the model coefficients, weight and age were standardized to zero mean and unity standard deviation (SD). In males, mean age was $\bar{x} \pm \text{SD} = 87.6 \pm 27$ days, and mean weight was $\bar{x} \pm \text{SD} = 0.055 \pm 0.012$ g. The PMRN mid-points (i.e. the estimated age-specific weight at which the probability of maturing is 50%; also referred as W_{p50}) were used to illustrate the estimated reaction norms and are roots of equation (4) for weight w .

For females, maturation cannot be assessed non-invasively, and age at maturation (a_{mat}) was estimated from the biphasic growth model. This implies a definition of maturation that is purely energetic and corresponds to the (assumed) abrupt start of allocating a significant proportion of energy to reproduction; it is not possible to link this definition to male maturation based on different criteria.

Results

Males

Growth in males showed high inter-individual variability (Fig. 1a). Nevertheless, growth models suggested significant effects of both oxygen treatment and parental size-selection line (Fig. 1b and Table 1). No single model was superior, but all highest-ranking models were broadly similar and suggested significant effects of oxygen and/or size selection on all parameters (Table 1). The model that explained the data best (M1) included effects of oxygen and size selection on reproductive investment (r) and on the coefficient c in the growth rate–weight relationship, whereas there was an effect of size-selection line only on the allometric exponent β of the growth rate–weight relationship (Table 1). This model was superior to the model that did not include any treatment effect (M0; $\Delta\text{AIC} = 25.99$, likelihood ratio test statistic = 35.99, $P < 0.001$). Males under low ambient oxygen from each selection line reached lower predicted weights at age 210 days than their counterparts with high ambient oxygen (Fig. 1b and Table 1), but their size-specific maximal potential growth rate was higher (growth rate theoretically attained in the absence of reproduction; Fig. 2a). Likewise, males that were descended from the positive size-selection lines reached higher predicted weights at age 210 days (Fig. 1b) and presented a higher size-specific maximal potential growth rate than those descended from the negative size-selection lines (Fig. 2a). Results are similar for the other models with high probability for explaining our data (M2–M4); these models also showed the effect of our treatments on the growth parameters, particularly with an effect of size-selection line on allometric growth and oxygen in reproductive investment (Table 1). Only one model (M3) included an interaction effect between oxygen and size selection, suggesting that the effect of oxygen on reproductive investment (r) was

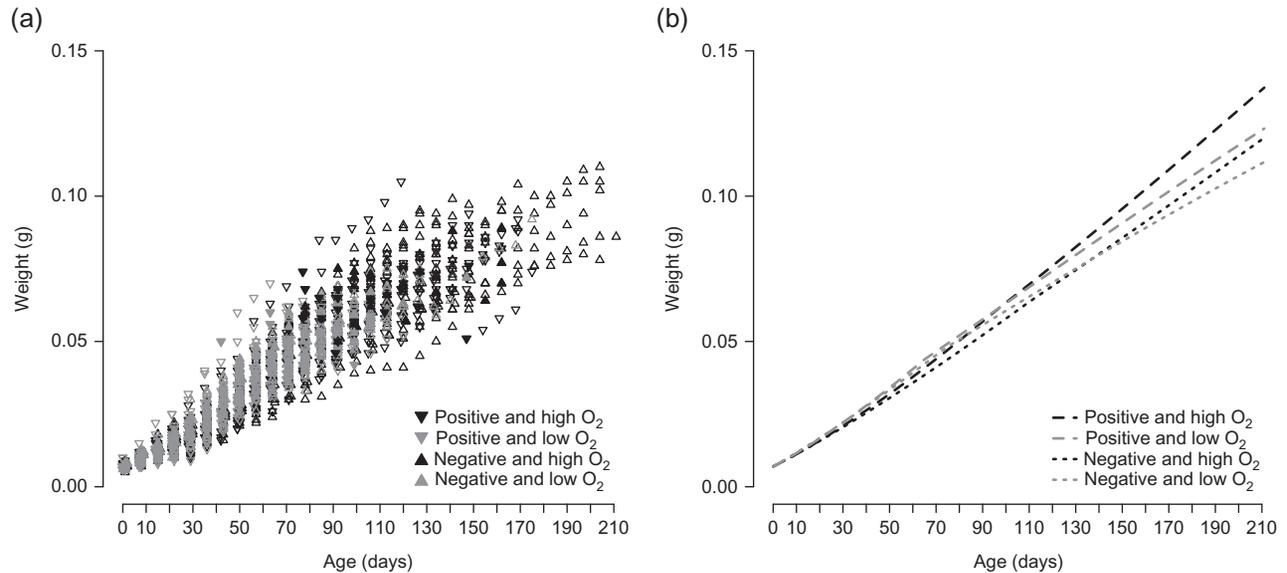


Figure 1: Growth trajectories for males from raw data (a) and biphasic growth model estimates (b). In (a), symbol type represents the size-selection treatment and colour the oxygen treatment. Filled symbols depict the observations when initiation of maturation was scored. In (b), colour represents oxygen treatments, respectively, and line type refers to size-selection line. Growth curves are based on the best-ranked model (M1), and growth parameter values are given in Table 1.

reversed for the negatively compared with positively size-selected lines (Table 1).

In high-oxygen conditions, males from the lines exposed to negative size-selective mortality matured at 0.065 ± 0.010 g (mean \pm SD) and 111 ± 25 days old, whereas those from positive size-selective mortality matured at 0.060 ± 0.008 g and 97 ± 23 days (Fig. 3). In the presence of low oxygen availability, males matured at 0.048 ± 0.008 g and 75 ± 19 days old and at 0.044 ± 0.007 g and 66 ± 12 days old for negative and positive size-selection, respectively. Thus, both low oxygen and positive size-selective mortality resulted in earlier maturation at smaller size, but the effect of oxygen was larger than that of size-selective mortality.

Mean age and size at maturation are also influenced by growth. Maturation tendency can be expressed independently from growth by calculating age- and size-dependent maturation probabilities, i.e. PMRNs. Nearly horizontal PMRNs (Fig. 3) show that maturation is primarily determined by size, with only a weak, positive effect of age. The size (weight) at 50% maturation probability at a given age was significantly smaller in low-oxygen conditions and for positive size-selection lines (Fig. 3). The oxygen availability had the strongest effect, with the odds of maturation in the presence of low oxygen ~ 61 times higher than in high-oxygen conditions [estimate \pm SE = 4.11 ± 0.9 in log(odds), $z = 4.68$, d.f. = 1, $P < 0.001$]. This is in line with the results obtained from analysis of growth curves showing that males in the presence of low oxygen also invested more in reproduction (higher r) than those reared in high oxygen. Descending from the positive size-selection line had a

weaker positive effect, increasing the odds of maturation compared with negative size-selection by a factor of 3.1 [estimate \pm SE = 1.12 ± 0.5 in log(odds), $z = 2.33$, d.f. = 1, $P = 0.02$].

The effect of oxygen availability on maturation was strong also in comparison to the effect of growth. An increase in weight by 1 SD (0.012 g) corresponded to an increase in odds of maturing by a factor of 11.0 [estimate \pm SE = 2.41 ± 0.5 in log(odds), $z = 5.12$, d.f. = 1, $P < 0.001$]. Age influenced maturation only through its interaction with weight; the effect was weak but significant [odds ratio = 0.59 for 1 SD increase in weight and age, estimate \pm SE = -0.53 ± 0.1 in log(odds), $z = -3.75$, d.f. = 1, $P < 0.001$], which resulted in a decreasing PMRN for older ages (Fig. 3).

Females

As with males, inter-individual variability in female growth was high but contained significant effects related to oxygen availability and parental size-selection line (Fig. 4a). The best-ranked model (F1) showed an effect of oxygen level, selection line and their interaction on age at maturation, and an effect of oxygen and size selection on reproductive investment and on the growth coefficient (Table 2). Females reared in low-oxygen conditions showed lower predicted weight at age 190 days relative to females reared in high-oxygen conditions (Fig. 4b). Similar to the males, this was probably a result of a higher investment in reproduction and earlier age at maturation (Table 2), rather than size-specific maximal potential growth rate that was higher in low oxygen (Fig. 2b). Although females from the positive size-selection

Table 1: Male biphasic growth model estimates for reproductive investment, r , growth coefficient, c , and allometric exponent, β

Model	Effects	Support		Parameter estimates			
		Δ_i	w_i	Treatment	r	c ($g^{1-\beta} \text{ day}^{-1}$)	β
M1	$r \sim$ size selection + O_2 $c \sim$ size selection + O_2 $\beta \sim$ size selection	0	0.28	High O_2 and negative size selection	0.0006	0.0009	0.16
				High O_2 and positive size selection	0.0009	0.0013	0.25
				Low O_2 and negative size selection	0.0025	0.0010	0.16
				Low O_2 and positive size selection	0.0028	0.0014	0.25
M2	$r \sim O_2$ $c \sim$ size selection $\beta \sim$ size selection + O_2	0.07	0.27	High O_2 and negative size selection	0.0007	0.0009	0.17
				High O_2 and positive size selection	0.0007	0.0013	0.25
				Low O_2 and negative size selection	0.0023	0.0009	0.14
				Low O_2 and positive size selection	0.0023	0.0013	0.22
M3	$r \sim$ size selection * O_2 $c \sim$ size selection + O_2 $\beta \sim$ size selection	0.11	0.26	High O_2 and negative size selection	0.0002	0.0009	0.16
				High O_2 and positive size selection	0.0014	0.0013	0.24
				Low O_2 and negative size selection	0.0029	0.0010	0.16
				Low O_2 and positive size selection	0.0020	0.0014	0.24
M4	$r \sim O_2$ $c \sim$ size selection + O_2 $\beta \sim$ size selection	0.81	0.19	High O_2 and negative size selection	0.0007	0.0009	0.16
				High O_2 and positive size selection	0.0007	0.0013	0.24
				Low O_2 and negative size selection	0.0026	0.0010	0.16
				Low O_2 and positive size selection	0.0026	0.0014	0.24
M0	$r \sim 1$ $c \sim 1$ $\beta \sim 1$	25.99	0.00	n.a.	0.0013	0.001	0.18

Support for a particular model is given by the change in the Akaike information criterion (AIC) relative to the model with the lowest AIC (Δ_i), and by the Akaike weights (w_i). All models follow equations (2) and (3) but differ in which of the parameters (if any) are affected by the treatment(s) as well as the presence of treatment interactions (denoted with '*' in the model formulae). Results are shown for the four best-ranked non-linear mixed-effect models (M1–M4; the model with the lowest AIC and all models for which $\Delta_i < 2$) as well as for the null model (M0) without any effects of experimental treatments (formula '~1' means that the parameter is unaffected by the treatments). n.a. means "not applicable".

line reached a higher predicted weight at 190 days (Fig. 4b), their size-specific maximal potential growth rate was lower than that of females in negative size-selection lines (Fig. 2b and Table 2). Females from positive size-selection lines presented lower reproductive investment and older age at maturation (Table 2) relative to females from negative size-selection lines. The model showing these treatment effects (F1) was superior to the null model considering no treatment effects (F0; $\Delta AIC = 48.11$, likelihood ratio test statistic = 62.10, $P < 0.001$). Similar results were obtained with the second-ranked model (F2; Table 2). Both best-ranked models suggest an interaction effect between oxygen and size selection, either for age at maturation (F1) or for reproductive investment (F2; Table 2).

Age at maturation for females could not be observed directly, but the estimates from the growth model showed a pattern similar to the one obtained for males (Table 2). Mean age at maturation was lower in low-oxygen conditions compared with high-oxygen conditions. However, females from lines with negative size-selective mortality had a lower age at

maturation than those from lines with positive size-selective mortality. The highest mean age at maturation was for females in high-oxygen conditions for females from lines with positive size-selective mortality (65 days). These estimates are lower than the observations for males (treatment-specific mean 66–111 days), but the estimates are not directly comparable because they are based on different ways of defining and estimating maturation.

Discussion

The oxygen saturation in the ambient water and prior ancestral exposure to size-selective mortality affected maturation, growth and reproductive investment in similar ways. A reduced ambient oxygen led to stunting, early maturation and high reproductive investment. Fish exposed to high mortality of larger-sized individuals displayed earlier maturation at smaller size, greater investment in reproduction and faster growth. These results were clearer for male guppies than for females.

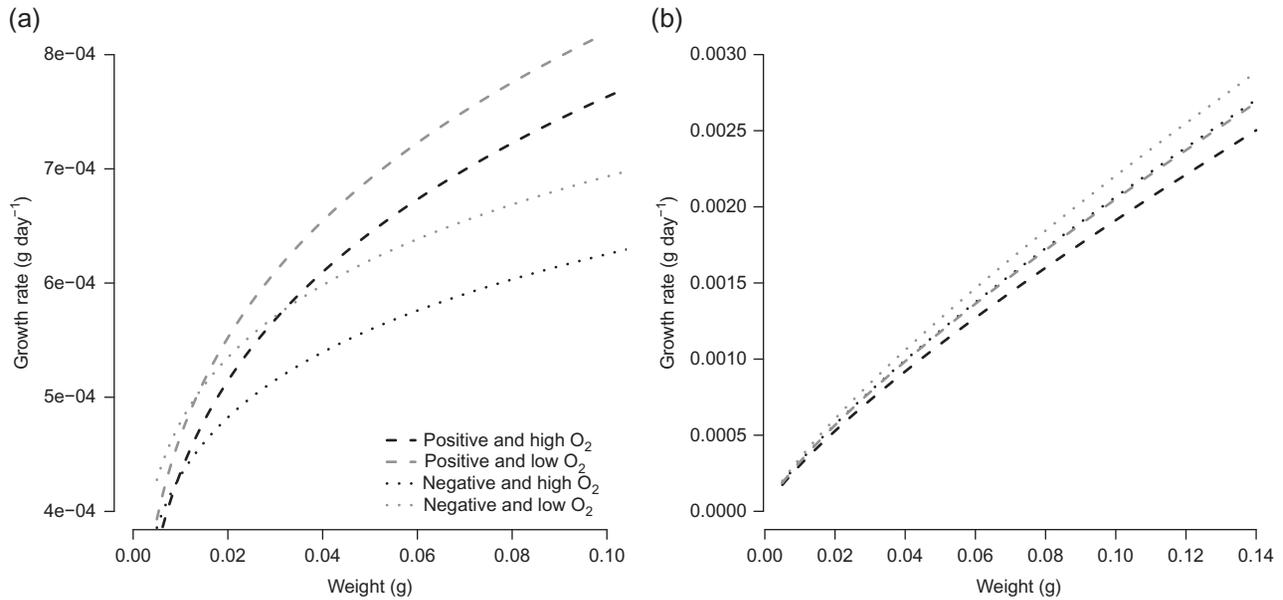


Figure 2: Maximal potential size-specific growth rates for males (a) and females (b) in high (black lines) and low (grey lines) oxygen treatments and that belonged to the positive size-selection lines (dashed lines) or the negative size-selection lines (dotted lines). Growth rates are based in males (a) on the allometric exponent β and the coefficient c in the growth rate–weight relationship estimated with the best-ranked model (M1, Table 1), whereas in females (b), growth rates are based on the allometric coefficient c in the growth rate–weight relationship estimated with the best-ranked model (F1) and the exponent β had the value of 0.8 for all treatments (Table 2). Realized growth rates are lower when energy is allocated to reproduction; the predicted growth curves in Figs 1b and 4b account for this, for males and females, respectively.

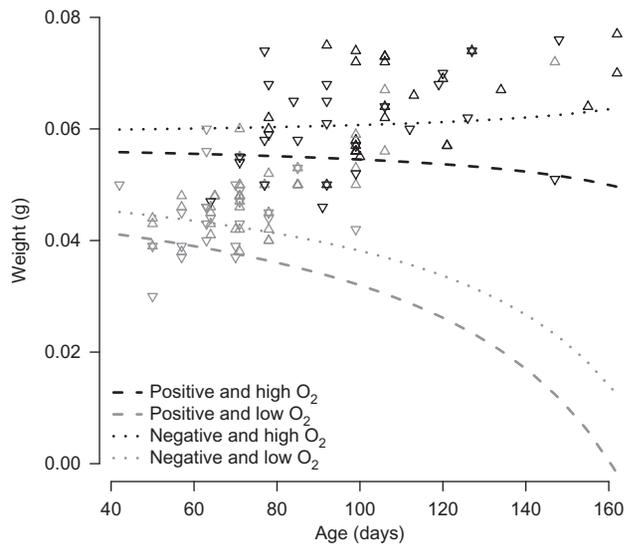


Figure 3: Weight- and age-based probabilistic maturation reaction norms for males represented by the midpoints (weight with 50% maturation probability, W_{p50}) in conditions of high (black line) and low (grey line) oxygen and for positive (dashed line) and negative (dotted line) size-selected lines. Black and grey triangles (negative size-selection line) and inverted triangles (positive size-selection line) represent the observed weights (in grams) and ages (in days) at maturation for high and low oxygen, respectively.

Oxygen

Exposure to low oxygen saturation resulted in a smaller size at age and higher investment in reproduction relative to exposure to normoxic conditions, as expected if reduced oxygen supply triggers the shift from somatic growth to maturation (Pauly, 1984; Kolding, 1993; Kolding *et al.*, 2008a). Both males and females also matured at an earlier age and smaller size when reared in low-oxygen relative to high-oxygen conditions. The low-oxygen treatment was not severe enough to hamper fish maturation as observed in some other studies (e.g. Wu *et al.*, 2003; Landry *et al.*, 2007; Chabot and Claireaux, 2008).

Low oxygen resulted in faster juvenile size-specific maximal growth rate. Iles (1973) predicted such an increase in the juvenile growth rate of wild tilapia owing to low oxygen availability, although his prediction might be a result of lack of standardization of the growth rates. In any case, it should be noticed that a reduction in growth rate associated with low oxygen levels is detectable only after maturation (Pauly, 1981; van Dam and Pauly, 1995). Other studies of adult growth in guppies did see a decrease in growth rate owing to oxygen limitation (Weber and Kramer, 1983). The lack of decrease in growth rate in our experiment was not attributable to surface respiration, because our experimental set-up prevented it. Aquatic surface respiration is initiated in

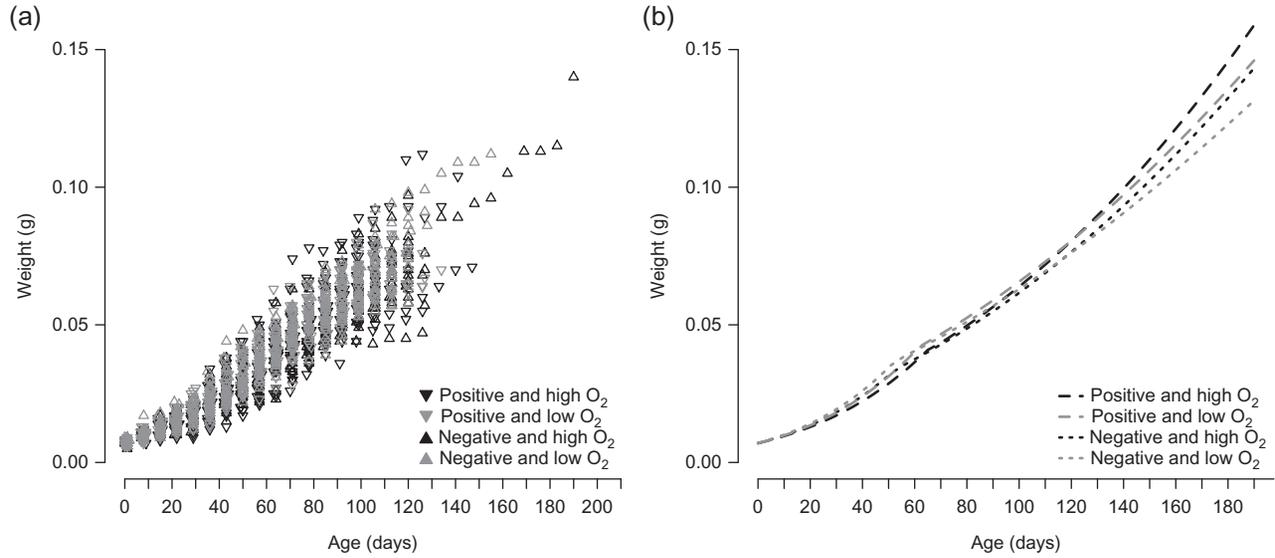


Figure 4: Growth trajectories for females from raw data (a) and biphasic growth model estimates (b). In (a), inverted triangles represent the positive size-selected line and upright triangles the negative size-selected line, while black refers to high oxygen and grey to low oxygen. In (b), black and grey lines represent high- and low-oxygen treatments, respectively; and dashed lines refer to females that belonged to the positive size-selection line whereas dotted lines refer to the negative size-selection line. Growth curves are based on the best-ranked model (F1), and growth parameter values are given in Table 2.

Table 2: Female biphasic growth model estimates for reproductive investment, r , growth coefficient, c , and age at maturation, a_{mat}

Model	Effects	Support		Parameter estimates			
		Δ_i	w_i	Treatment	r	c ($g^{1-\beta} day^{-1}$)	a_{mat} (days)
F1	$r \sim$ size selection + O_2 $c \sim$ size selection + O_2 $a_{mat} \sim$ size selection * O_2	0	0.48	High O_2 and negative size selection	0.011	0.013	53.7
				High O_2 and positive size selection	0.009	0.012	65.2
				Low O_2 and negative size selection	0.014	0.014	53.6
				Low O_2 and positive size selection	0.012	0.013	62.8
F2	$r \sim$ size selection * O_2 $c \sim O_2$ $a_{mat} \sim$ size selection + O_2	0.95	0.30	High O_2 and negative size selection	0.010	0.012	54.4
				High O_2 and positive size selection	0.009	0.012	63.7
				Low O_2 and negative size selection	0.012	0.013	53.5
				Low O_2 and positive size selection	0.014	0.013	62.9
F0	$r \sim 1$ $c \sim 1$ $a_{mat} \sim 1$	48.11	0.00	n.a.	0.011	0.013	60.6

Results are shown for the two best-ranked non-linear mixed-effect models [F1–F2, i.e. the model with the lowest Akaike information criterion (AIC) and the only other model for which $\Delta_i < 2$] as well as for the null model (F0) without any effects of experimental treatments. See Table 1 for further explanation. n.a. means "not applicable".

guppies at ~30% oxygen saturation (Kramer and Mehegan, 1981); hence, even if it had been allowed in our study, it might not have been important. Thus, our modest reduction in oxygen availability led to a slightly faster juvenile growth rate and triggered earlier maturation and increased reproductive allocation, which resulted in stunting in both males and females, despite higher maximal potential growth rates.

Size-selective mortality

Positive size-selective mortality implies a higher mortality risk for large individuals relative to small individuals. In the present study, the size limit for culling was set at 16 mm SL, slightly less than normal guppy maturation length (Magurran, 2005). Positive size-dependent mortality favours fast life-

history strategies involving early maturation, high investment in reproduction and, in many cases, a faster growth rate before maturation (Charlesworth, 1994; Law, 2000; Réale *et al.*, 2010; Enberg *et al.*, 2012).

Our results are in agreement with these expectations, particularly in the case of male guppies. Males descending from lines exposed to positive size-selective mortality had a higher probability of maturing at a given age and size, which led to maturation at a smaller size and younger age compared with males from the lines subjected to negative size selection. Males also had a higher investment in reproduction. Our estimates are comparable with earlier studies on guppies and other poeciliids for reproductive investment (Baatrup and Junge, 2001; Schlupp *et al.*, 2006) and size and age at maturation (Reznick and Bryga, 1987; Magurran, 2005); it should be noticed that most studies considered completion of maturation, rather than initiation of maturation (but see Diaz Pauli and Heino, 2013). Similar directional changes in maturation and reproductive investment have been observed in several exploited fish populations (Heino *et al.*, 2015) and in other selection experiments (van Wijk *et al.*, 2013; Uusi-Heikkilä *et al.*, 2015).

Males presented faster maximal potential and realized growth rates in lines exposed to positive size-dependent mortality. Studies on the effect of (positive) size-selective fishing mortality have often concluded that growth rates decreased rather than increased, but in most cases such reduction was a secondary effect from increased allocation to reproduction (reviewed by Enberg *et al.*, 2012; Heino *et al.*, 2015) and applies to post-maturation growth. This contrasts with the simplistic expectation that killing large fish should always favour smaller fish and thus slower growth. Although this expectation is largely warranted for adult fish, expectations for juvenile growth are less straightforward (Enberg *et al.*, 2012). Dunlop *et al.* (2009) concluded that one key factor that determines whether positively size-selective fishing favours an increased or decreased juvenile growth rate is the size limit at which the harvesting takes place. When the minimal size is set smaller than the size at maturation, as in our experiment, juvenile growth is expected to accelerate to reach maturation earlier in life (Dunlop *et al.*, 2009). Positive size selection also led to faster juvenile growth rate in zebrafish (*Danio rerio*; Uusi-Heikkilä *et al.*, 2015).

Males from the lines exposed to positive size-selective mortality had larger predicted size at age 210 days (the maximal age in the experiment). This occurred because of their high maximal potential growth rate and despite their earlier maturation and higher investment in reproduction. This result is contrary to theoretical expectations (Heino *et al.*, 2015) and other experimental studies (Walsh *et al.*, 2006; van Wijk *et al.*, 2013; Uusi-Heikkilä *et al.*, 2015). A possible explanation is that because we killed our fish soon after maturation, we have little information on how their realized growth and reproductive allocation would have developed

through their adulthood, which was estimated in former studies (Walsh *et al.*, 2006; van Wijk *et al.*, 2013; Heino *et al.*, 2015; Uusi-Heikkilä *et al.*, 2015). The ultimate size at adulthood is affected by the maximal potential somatic growth rate as well as the continued investment in reproduction in this iteroparous species and might have resulted in smaller individuals later in life in positive size-selected lines. Our estimates of realized growth rate are similar to those of Auer *et al.* (2010). The values of β estimated from our model are in the lower range of the great variation in the values of the allometric exponent β (Killen *et al.*, 2010; Boukal *et al.*, 2014), which is associated with determinate/indeterminate growth. Male poeciliids are typically considered to have determinate growth, although they do not completely cease growth after maturation (Snelson, 1982). Nevertheless, because fish were killed well before reaching their maximal sizes, our estimates of β might be downward biased. In practice, the estimations of β and reproductive investment (r) are confounded, and the truncated adult lifespan might have aggravated this problem.

Whether the differences between size-selected lines represent evolutionary (i.e. genetic) change is ambiguous, as our experimental set-up only controlled for environmental differences among the fish subjected to the oxygen treatments, but not those of their parents. It is generally accepted that lines should be maintained for at least two generations in common garden conditions to be able to discern genetic changes clearly using phenotypic data (Reznick and Ghalambor, 2005). The differences could therefore represent parental effects, genetic differences or (perhaps most likely) a combination of both. Nevertheless, the phenotypic changes were in agreement with the predictions from life-history theory.

Estimates for reproductive investment, growth rate and age at maturation in females are comparable with values obtained in other studies (Magurran, 2005; Auer *et al.*, 2010; Rocha *et al.*, 2011). Still, as maturation in females could not be determined visually, the study of life-history changes in them was not as thorough as with males. Exposure to positive size-selective mortality led to estimated maturation at older rather than younger ages, and to a lower investment in reproduction. This is opposite to what was observed in males in the present study and earlier selection experiments (Walsh *et al.*, 2006; Uusi-Heikkilä *et al.*, 2015). However, these results refer to age at maturation inferred with the growth model and which might be inaccurate, rather than to directly observed maturation, as with males. In addition, females in the positive size-selected line presented lower maximal potential size-specific growth rates, but higher realized growth, contrary to what was observed in males. The estimation of maximal growth rate was based on only one parameter (c , the coefficient in the growth rate–weight relationship), while the allometric exponent β was kept constant. For males, it was the allometric exponent β that showed the strongest effect of size-selection line and the parameter that affected growth rate the most. If the growth model for females is performed to estimate β by keeping c constant at

$0.01 \text{ g}^{1-\beta} \text{ day}^{-1}$, the results remain very similar (results not shown). Nevertheless, the differences between positively and negatively size-selected lines were smaller for females than for males, despite being significant in all cases.

Interplay of effects on life-history traits and implications

Manipulation of the oxygen level resulted in bigger changes in reproductive investment and maturation compared with manipulation of size-selective mortality in parental generations. Positive culling led to an estimated increase in reproductive investment of 33% relative to negative culling (in high-oxygen conditions), whereas low oxygen led to an increase of >100% relative to high oxygen. Similar results were obtained for age and size at maturation; the odds of maturing were 60 times higher in the presence of low oxygen compared with high oxygen, but only three times higher for positive lines compared with negative lines.

However, direct comparison of the importance or strength of these two different drivers is difficult for two reasons. First, the two treatments are conceptually very different; the oxygen treatment was affecting the ambient environment of the very same fish that we observed during the experiment, whereas the size-selective mortality treatment represented conditions that the parental generations of the test fish had experienced over the course of 3 years (approximately four generations). The actual treatment levels are in both cases somewhat arbitrary (i.e. the specific oxygen saturation level and the duration and intensity of past size selection). Second, the mechanisms through which the treatments affect life histories are different. Oxygen is a strong proximate driver of phenotypic change in maturation and growth, triggering direct plastic responses (Pauly, 1984; Kolding *et al.*, 2008a), whereas the effect of size-selective mortality on life histories occurs through both genetic change (evolution) and phenotypic plasticity, including inter-generational plasticity (parental effects). Although hypoxia could also lead to evolutionary changes in life history (Riesch *et al.*, 2010), this was not considered in our experiment that followed only a single generation of fish.

Our results do not suggest strong interactions between ambient oxygen and prior size selection in controlled laboratory conditions; that is, that the effects of oxygen level would depend on adaptations to contrasting size-selectivity regimes. For males, only one of the four top-ranking growth models included an interaction between size selection and oxygen (affecting a single parameter), whereas for females, both top-ranking models contained a single interaction each. These findings provide some evidence for the oxygen-depletion-induced increase in reproductive investment being stronger in the lines that had been subjected to negative size-selective mortality. Most effects, however, were simply additive.

We believe it is essential to consider both proximate and ultimate factors to gain a better understanding of life-history

variation and how populations evolve under the influence of these factors. Hypoxia and size-dependent mortality, including that induced by fishing, not only co-occur, but can also drive similar life-history changes. Thus, investigation of the interplay of fishing- and hypoxia-induced changes is necessary to make ecosystem-based predictions on the sustainability of the fishery (Kolding *et al.*, 2008b). To our knowledge, this is the first study to look at the combined effect of oxygen and size-dependent mortality on life-history traits. Despite being an experimental study, our results illustrate the risks of trying to infer the process from patterns. This is a well-known problem, much discussed in the context of using observational field data to study life-history changes in exploited fish populations (e.g. Dieckmann and Heino, 2007; Kraak, 2007; Browman *et al.*, 2008; Jørgensen *et al.*, 2008; Kuparinen and Merilä, 2008). The potential role of low oxygen levels in driving phenotypic change, however, has until now been overlooked (e.g. Sharpe *et al.*, 2012). We encourage the performance of further studies to link these factors to changes in life-history, behavioural and physiological traits, and that the confounding effect of oxygen should be considered along with other environmental factors when studying the effects of size-selective fishing in exploited populations.

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