Received: 16 August 2023 | Revised: 15 November 2023 | Accepted: 7 February 2024

DOI: 10.1002/ecy.4303

REPORT

ECOLOGY
ECOLOGICAL SOCIETY OF AMERICA

Growth of brown trout in the wild predicted by embryo stress reaction in the laboratory

Jonas Bylemans[1,2] | **Lucas Marques da Cunha**[1] | **Laetitia G. E. Wilkins**[1,3] | **David Nusbaumer**[1] | **Anshu Uppal**[1] | **Claus Wedekind**[1]

[1]Department of Ecology and Evolution, Biophore, University of Lausanne, Lausanne, Switzerland

[2]University of Savoie Mont Blanc, INRAE, CARRTEL, Thonon-les-Bains, France

[3]Max-Planck Institute for Marine Microbiology, Bremen, Germany

Correspondence
Claus Wedekind
Email: claus.wedekind@unil.ch

Funding information
The Swiss National Science Foundation, Grant/Award Numbers: 31003A_159579, 31003A_182265

Handling Editor: Andrea Kirkwood

Abstract

Laboratory studies on embryos of salmonids, such as the brown trout (*Salmo trutta*), have been extensively used to study environmental stress and how responses vary within and between natural populations. These studies are based on the implicit assumption that early life-history traits are relevant for stress tolerance in the wild. Here we test this assumption by combining two data sets from studies on the same 60 families. These families had been experimentally produced from wild breeders to determine, in separate samples, (1) stress tolerances of singly kept embryos in the laboratory and (2) growth of juveniles during 6 months in the wild. We found that growth in the wild was well predicted by the larval size of their full sibs in the laboratory, especially if these siblings had been experimentally exposed to a pathogen. Exposure to the pathogen had not caused elevated mortality among the embryos but induced early hatching. The strength of this stress-induced change of life history was a significant predictor of juvenile growth in the wild: the stronger the response in the laboratory, the slower the growth in the wild. We conclude that embryo performance in controlled environments can be a useful predictor of juvenile performance in the wild.

KEYWORDS

0+ juvenile, embryo, growth, hatching, larvae, life history, maternal environmental effects, salmonids, stress-induced, trout

INTRODUCTION

Salmonids are not only charismatic fish of high ecological and socioeconomic relevance but also excellent models for experimental research, especially in early developmental stages. External fertilization and the lack of parental care allow for full-factorial in vitro fertilization under controlled conditions. Embryos can then be raised in separate groups or singly in multiwell plates to study, for example, family-specific growth and mortality under different environmental conditions or life-history decisions such as the timing of hatching. This allows for estimating the genetic and maternal environmental effects on embryo mortality (Houde et al., 2013, 2016) or developmental problems (Evans & Neff, 2009). Embryo performance can then be linked to parental characteristics to learn more

Jonas Bylemans and Lucas Marques da Cunha are co-first authors.

about, for example, the information content of sexual signals (Huuskonen et al., 2011; Janhunen et al., 2011; Neff & Pitcher, 2005; Wedekind et al., 2008), the genetic quality of dominant males (Jacob et al., 2007), or the fitness consequences of different female reproductive strategies (Jacob et al., 2010; Kekäläinen et al., 2010). Embryo performance can also be studied under different environmental conditions. Large numbers of independent replicates then allow for estimating the relevance of environmental factors and the evolutionary potential of populations to adapt to them. The latter is typically revealed in two- or three-way interactions between parental and environmental factors on embryo performance. Environmental factors that have been studied on embryos of experimentally produced families include increased temperatures in the context of climate change (Burt et al., 2012; Muñoz et al., 2014), different types of chemical pollutants (Marques da Cunha et al., 2019; Nusbaumer, Marques da Cunha, & Wedekind, 2021), pollution by nanoparticles (Clark et al., 2016; Yaripour et al., 2021), organic pollution (Nusbaumer, Garaud, et al., 2021; Wedekind et al., 2010), and pathogens (Clark et al., 2014; Pompini et al., 2013; von Siebenthal et al., 2009; Wilkins et al., 2017). Such experimental designs can make mandatory ecotoxicological tests on fish more informative (Wedekind et al., 2007). Full-factorial crosses can even be used to estimate the variance components of commercial traits in farmed salmon (Colihueque, 2010; Ødegård et al., 2011), to study the effects of sperm cryopreservation (Nusbaumer et al., 2019), or to describe microbial symbionts on different hosts (Wilkins et al., 2016). Factorial breeding within and between populations and monitoring of embryo performance have also been used to study the causes of phenotypic differentiation among populations (Aykanat et al., 2012) or to determine hybrid vigor as an indicator of inbreeding depression (Clark, Stelkens, & Wedekind, 2013; Stelkens et al., 2014).

Most studies that are based on factorial breeding designs have focused on embryos or larvae and ignored later developmental stages, with few exceptions: Juveniles that resulted from experimental breeding have been raised in captivity to study genotype-by-environmental effects (Evans et al., 2010), genetic aspects of life history (Forest et al., 2016), maternal environmental effects in different temperature environments (Thorn & Morbey, 2018), or effects of enriched versus nonenriched environments (Yaripour et al., 2020). Studies on juveniles released into the wild and recaptured later are scarce and, so far, generally suffer from low sample sizes because of low recapture rates (von Siebenthal et al., 2017; Wedekind et al., 2008). Consequently, the conclusions drawn from laboratory studies on experimentally produced families assume that the observed reactions reveal effects that are also relevant in the wild. This assumption is still poorly backed up.

Here we combine data from two studies on brown trout (*Salmo trutta*) that were done in separate samples from the same 60 families. These families had been experimentally produced in two full-factorial breeding blocks using gametes collected from wild-caught males and females. The breeders were sampled from a stream that represents a mostly pristine environment within the Swiss Plateau (Marques da Cunha et al., 2019; Nusbaumer, Marques da Cunha, & Wedekind, 2021) and a population that shows no signs of elevated inbreeding (Clark, Stelkens, & Wedekind, 2013; Stelkens et al., 2014). One sample of freshly fertilized eggs per each of the 60 families was used in a controlled laboratory environment to study embryo growth and stress tolerance (Wilkins et al., 2017). The other sample was incubated under hatchery conditions and stocked in a natural streamlet at early larval stages following routine procedures of the local fishery authorities. Approximately 6 months later, a significant number of these fish could be successfully sampled at the end of their first summer, as revealed by molecular markers. Parental assignments and molecular sexing could then be applied to study sex-specific inbreeding depression in the wild (Bylemans et al., 2024). Here we combine the findings on the embryos (Wilkins et al., 2017) with the findings on the juveniles (Bylemans et al., 2024) to test how the outcomes of laboratory studies on embryos correlate with juvenile performance in the wild.

MATERIALS AND METHODS

The experiments started with male and female brown trout being caught shortly before the spawning season from the *Rotache* stream (a tributary of the Aare River; see Stelkens et al. [2012] for a description of the genotypes and phenotypes of this and neighboring populations). They were kept in the Fischereistützpunkt Reutigen and regularly checked for ovulation until the eggs of 12 females could be stripped and fertilized with milt of 10 males to create two full-factorial breeding blocks (6×5 each) on the same day. See Wilkins et al. (2017) for a detailed description of the procedure. Fin clips were collected and stored in 70% ethanol at −20°C for genetic analyses.

Before fertilization, total egg weight per female was determined and four eggs per female were frozen in liquid nitrogen for later analyses of the carotenoids astaxanthin, capsanthin, lutein, and zeaxanthin as described in Wilkins et al. (2017). After fertilization and egg hardening (>2 h), photos of each family (eggs in monolayer in individual Petri dishes) were taken to later count the eggs

and determine the average egg weight per female (total egg weight/egg count).

A subset of 24 fertilized eggs per family was transported to the laboratory and raised singly (each egg in its own 2-mL well of a 24-well plate) in a climate chamber under controlled experimental conditions to assess the effects of egg carotenoids on the tolerance of embryos to the bacterium *Pseudomonas fluorescens*. A bacterial strain was used that had previously been found to reduce embryo growth and affect hatching time but would not significantly elevate mortality (Clark, Stelkens, & Wedekind, 2013; Clark, Wilkins, & Wedekind, 2013). The details of the bacterial culture and exposure are provided by Wilkins et al. (2017). Briefly, half of the embryos per family received 0.2 mL standardized water with 2×10^6 bacterial cells per well to a final water volume of 2 mL/well, the other half received 0.2 mL standardized water only (sham controls). The treatment happened either 18 days post fertilization (DPF; first breeding block) or 49 DPF (second breeding block) to also test for virulence effects of the timing of pathogen exposure (as reported in Wilkins et al., 2017).

Shortly before hatching was expected to start, eight embryos per family were sacrificed for a study on carotenoid consumption (Marques da Cunha et al., 2018). All remaining eggs were checked daily to record individual hatching time and to take photos of the freshly hatched larvae. These photos were used to determine hatchling length (L_{hatching}) and hatchling yolk-sac volume (YS_{hatching}). Because hatchling size is likely to vary with the timing of hatching, larval length and yolk-sac volume were again measured for each larva 14 days past hatching (DPH; $L_{14\text{DPH}}$ and $YS_{14\text{DPH}}$, respectively). This could be done in a sample of 815 larvae (mean ± SD per family = 13.6 ± 1.8; after five larvae whose measurements of larval growth per loss of yolk sac over these 14 days exceeded 3 SDs from the global mean had been excluded as outliers). Larval length at 75 DPF, the day when all larvae had hatched (and 11 days after the very first hatching), was calculated by linear extrapolation as

$$L_{75\text{DPF}} = L_{\text{hatching}} + \left(75 - D_{\text{hatching}}\right)\left(L_{14\text{DPH}} - L_{\text{hatching}}\right)/14, \quad (1)$$

where D_{hatching} is the number of days from fertilization to hatching and $L_{14\text{DPH}}$ the larval length at 14 DPH. Analogously, yolk-sac volume at 75 DPF was determined as

$$YS_{75\text{DPF}} = YS_{\text{hatching}} + \left(75 - D_{\text{hatching}}\right)\left(YS_{\text{hatching}} - YS_{14\text{DPH}}\right)/14. \quad (2)$$

The remaining 1925 eggs (mean ± SD number per full-sib family = 32.1 ± 14.8) were incubated from the day of fertilization under routine hatchery conditions in the cantonal Fischereistützpunkt Reutigen at constant 8.5°C and stocked in the Mühlibach streamlet (tributary to the Rotache; 46.804459° N, 7.690544° E) at 105 DPF, that is, in early March at a late yolk-sac stage when emergence from gravel would usually happen and larvae would start exogenous feeding.

In late August (281 DPF, i.e., nearly 6 months after release into the wild), electrofishing was used to sample brown trout juveniles from the Mühlibach streamlet, as reported in Bylemans et al. (2024). The fish were narcoticized (0.075 g/L tricaine methanesulfonate buffered with 0.15 g/L $NaHCO_3$) and photographed on a weighing scale to later extract fork length and weight. Fin clips were collected and stored in 70% ethanol at 4°C for genetic analyses. The adult breeders and 375 wild-caught juveniles were genotyped at 13 microsatellite loci and a sex-linked locus as described in Palejowski et al. (2022). The parental assignment was based on the full-likelihood approach implemented in Colony version 2.0.6.5 (Jones & Wang, 2010) with a threshold of 0.98. In total 301 of the 375 juveniles could be assigned to 56 of the 60 experimental families (range 1–17, mean ± SD = 5.3 ± 3.1; see Bylemans et al. [2024], who also provide a comparison between the juveniles identified as captive bred and as wild-born).

Statistical analyses were done in JMP Pro17. *F*-tests were used to compare means. Linear mixed-effect models (LMMs) were used to evaluate the relationship between laboratory-based measures (means per sib group and treatment) and the sizes of their full sibs caught from the wild. Dam and sire identities were included as random effects. Despite the full-factorial breeding, the limited number of recaptured juveniles per family did not allow for the inclusion of dam × sire interaction effects in the LMMs or to reliably estimate family-specific recapture rates. Treatment effects were tested using the mean difference in response variables (control—exposed to *P. fluorescens*) as predictors of juvenile size in the wild. The breeding block was added as a fixed factor to additional models (Appendix S1) because the timing of the experimental exposure to *P. fluorescens* could potentially affect embryo growth and the timing of hatching. Collinearity between predictor variables was evaluated using Pearson correlation coefficients to avoid the inclusion of highly correlated predictor variables (i.e., $|r| > 0.5$) within the same model (Dormann et al., 2013). Spearman correlation coefficients (r_s) were used to test for correlations between recapture rates in the wild and embryo performance in the laboratory, based on means per parent (after averaging over sib-group families).

TABLE 1 Linear mixed model on juvenile length as predicted by (a) larval length at 75 days post fertilization (DPF), that is, about 6 months before the juvenile were caught), and (b) yolk-sac volume per family at 75 dpf (using means per treatment and family each).

Effects	df	*F*	Variance component	*p*
(a) Larval length				
Fixed effects				
Larval length	1, 22.55	6.3		0.02
Random effects[a]				
Dam			20.0 ± 10.1	0.05
Sire			2.8 ± 3.0	0.35
Residual			79.9 ± 6.7	
(b) Yolk sac				
Fixed effects				
Yolk-sac volume	1, 14.2	10.4		0.006
Random effects[a]				
Dam			13.4 ± 8.4	0.11
Sire			4.0 ± 3.4	0.25
Residual			79.9 ± 6.7	

Note: Parental identities were included as random factors.
[a]REML unbounded variance components ± SE, Wald *p*-values.

RESULTS

Juvenile body length was on average 7.1 times larger than larval body length at 75 DPF and was well predicted by mean larval length and mean yolk-sac volume of their full sibs at 75 DPF (Table 1; Figure 1a; Appendix S1: Figure S1). Juvenile sex did not significantly affect these correlations (Appendix S1: Table S1). Juvenile body length was also positively correlated to mean egg weight (LMM; $F_{1,8.975} = 6.1$, $p = 0.04$; Appendix S1: Table S2a, Figure S2) but not significantly to the carotenoid content of the eggs (Appendix S1: Table S2b).

In separate tests of larvae that had or had not been stressed with *P. fluorescens*, the average days of hatching in the laboratory did not predict juvenile body length in the wild (Appendix S1: Table S3). However, the experimental stress induced a change in the mean hatching date that was a good predictor of growth in the wild: the stronger the stress response in the laboratory, the less the juveniles grew in the wild (Figure 1b; Table 2). Adding breeding blocks and mean larval length as factors to the LMM did not change this conclusion (Appendix S1: Table S4).

Larval length at 75 DPF was a significant predictor of juvenile size about 6 months later, whether the larvae had been raised under stress conditions (LMM; $F_{1,38.11} = 6.5$, $p = 0.01$; Appendix S1: Table S5a) or under nonstress conditions (LMM; $F_{1,21.41} = 5.0$, $p = 0.04$; Appendix S1: Table S5b). The stress-induced reduction in larval length at 75 DPF was not a significant predictor of juvenile size in the wild (Appendix S1: Table S5c).

Recapture rates per dam could not be predicted from average larval length ($r_s = -0.39$, $n = 12$, $p = 0.21$), yolk-sac volume ($r_s = 0.06$, $p = 0.86$), or stress-induced difference in hatching time ($r_s = -0.36$, $p = 0.25$), and none of the analogous correlations for recapture rates per sire were significant (average larval length: $r_s = 0.33$, $n = 10$, $p = 0.35$; yolk-sac volume: $r_s = 0.22$, $p = 0.54$; stress-induced difference in hatching time: $r_s = -0.30$, $p = 0.39$).

DISCUSSION

The high recapture rates that Bylemans et al. (2024) reported made it possible, arguably for the first time, to statistically link a large-scale laboratory study on fish embryos (Wilkins et al., 2017) with the performance of their juvenile siblings in the wild. All parental fish were well represented among the recaptured juveniles. Bylemans et al. (2024) reported that the recapture rates could not be predicted from inbreeding coefficients. We found that the recapture rates were also not correlated to any larval size measures or stress-induced hatching in the laboratory. However, we found that juvenile growth in the wild could be well predicted by measures taken in the laboratory. Egg size, larval length, and yolk-sac volumes were all significantly correlated with juvenile size (after taking possible parental effects into account). These correlations did not seem to be sex-specific, although Bylemans et al. (2024) found that the female juveniles suffered more inbreeding depression and were on average smaller than males. Because Wilkins et al. (2017) had quantified egg carotenoid content, we could also test for correlations between carotenoids and juvenile growth but did not find them to be significant. However, with only 12 dams, our sample size was limited for this type of analysis. It also remains to be shown what other maternal environmental effects could play a role here. Females may differ, for example, in how they supply their eggs with innate immunity proteins and antibodies (Li & Leatherland, 2012), and variance in maternal stress can influence glucocorticoid levels in eggs that then affect offspring development (Sopinka et al., 2017).

Wilkins et al. (2017) experimentally challenged embryos by exposing half of them to a bacterial pathogen that did not cause increased mortality but reduced growth and induced precocious hatching. This pathogen-related effect on hatching time was a good predictor of juvenile growth in the wild. Juveniles grew less if their

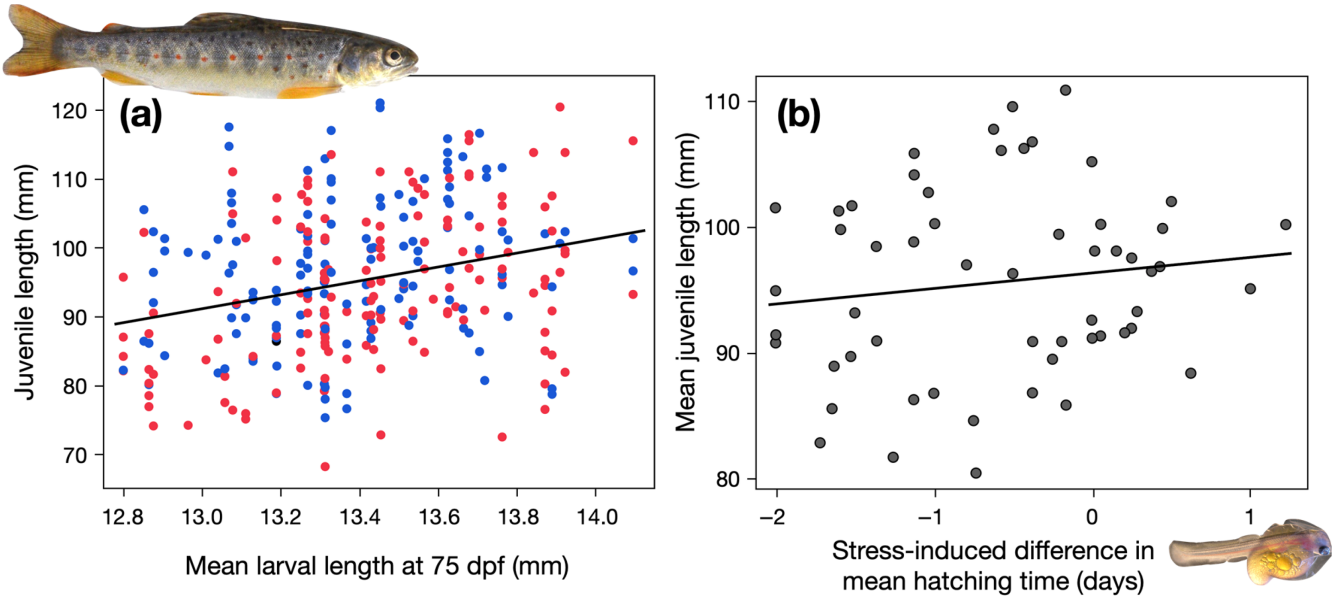

FIGURE 1 Juvenile length after 6 months in wild predicted by larval characteristics in laboratory. (a) Juvenile length versus mean larval length per family at 75 days post fertilization (DPF), for male (blue symbols) and female juveniles (red symbols). (b) Mean juvenile length per family versus stress-induced difference in mean hatching date of their full sibs in the laboratory. Regression lines illustrate direction of correlations. See Table 1 for statistics that take parental effects into account. The photos show a freshly hatched larva still partly in its egg membrane (photo credit: Manuel Pompini) and an average-sized juvenile after its first spring and summer in the wild (photo credit: Claus Wedekind).

TABLE 2 Linear mixed model on juvenile length as predicted by stress-induced difference in hatching date under laboratory conditions, that is, difference between mean hatching date under control conditions and mean hatching date after exposure to *Pseudomonas fluorescens*.

Effects	df	*F*	Variance component	*p*
Fixed effects				
Difference in hatching date	1154.9	5.4		0.02
Random effects[a]				
Dam			30.4 ± 14.5	0.04
Sire			3.4 ± 2.9	0.23
Residual			79.0 ± 6.7	

Note: Parental identities were included as random factors in all models.
[a]REML unbounded variance components ± SE, Wald *p*-values.

siblings had shown a stronger reaction to the experimental stress in the laboratory than other sib groups. The analogous effects of pathogen-induced reduction in growth were not statistically significant, but the correlation between larval size and juvenile size seemed strongest in the pathogen-exposed group.

The timing of hatching has been demonstrated to be a good indicator of perceived stress in salmonids and other taxa. The nearby presence of an infected egg and even water-borne cues emitted from infected eggs can induce early hatching in brown trout (Pompini et al., 2013), other salmonids (Wedekind, 2002), and other lower vertebrates (Warkentin et al., 2001). Induced early hatching could also be observed in response to other types of stress such as simulated danger of desiccation (Wedekind & Müller, 2005), a simulated or actual attack by a predator (Gomez et al., 2023; Warkentin, 2005), or exposure to chemical stressors (Lieke et al., 2021). The response of infected embryos can, however, differ in direction. Sometimes, exposure to pathogens induces early hatching (Pompini et al., 2013; Warkentin et al., 2001; Wedekind, 2002), sometimes it delays hatching (Clark et al., 2014; Nusbaumer, Marques da Cunha, & Wedekind, 2021). This difference in reaction is not understood yet but could be linked to the virulence of an infection.

Measuring family-specific fitness is notoriously difficult (Carlson & Seamons, 2008) and often based on strong assumptions, especially in laboratory studies. We found that key variables that are typically used in laboratory studies on embryos, such as larval growth or stress-induced change in life history, were good predictors of how siblings of these embryos grew in the wild during their first spring and summer. Our findings support the implicit assumption of numerous studies, namely that the effects of environmental challenges, as measured under laboratory conditions, can serve as

valuable predictors of family-specific performance in the wild and, hence, of the evolutionary potential of fish populations.

AUTHOR CONTRIBUTIONS

Jonas Bylemans, Lucas Marques da Cunha, Laetitia G. E. Wilkins, and Claus Wedekind designed the project. Lucas Marques da Cunha, Laetitia G. E. Wilkins, David Nusbaumer, Anshu Uppal, and Claus Wedekind did the experimental breeding, Lucas Marques da Cunha, and Laetitia G. E. Wilkins raised the embryos in the laboratory, and Lucas Marques da Cunha, David Nusbaumer, and Anshu Uppal sampled the juveniles from the wild. Jonas Bylemans and Lucas Marques da Cunha were responsible for the genotyping and parental assignments. Jonas Bylemans and Claus Wedekind performed the analyses and wrote the manuscript. All authors revised and approved the final manuscript for publication.

ACKNOWLEDGMENTS

We thank U. Gutmann and B. Bracher for catching and handling the adult fish and for raising and stocking the F1 into the wild, L. Garaud, M. Hobil, C. Küng, E. Longange, L. Menin, D. Ortiz, E. Pereira-Alvarez, and V. Vocat-Mottier for help in the laboratory and/or for discussion, T. Bösch, J. Kast, and the Ecogenics team for genotyping the fish, and K. Cottingham, A. Kirkwood, and two anonymous reviewers for comments on the manuscript. The project was approved by the Veterinärdienst of the Bern canton (BE118/14) and by the fisheries inspectorate of the Bern canton. The Swiss National Science Foundation provided funding (31003A_159579 and 31003A_182265). Open access funding provided by Universite de Lausanne.

CONFLICT OF INTEREST STATEMENT

The authors declare no conflicts of interest.

DATA AVAILABILITY STATEMENT

Paternal characteristics and embryo performance data (Wilkins et al., 2018) are avalable in Dryad at https://doi.org/10.5061/dryad.sj416. Data for dams, sires, sibling groups, and juveniles (Wedekind et al., 2024a) are available in Dryad at https://doi.org/10.5061/dryad.2ngf1vhv4. Code (Wedekind et al., 2024b) is available in Zenodo at https://doi.org/10.5281/zenodo.8065568.

ORCID

Claus Wedekind https://orcid.org/0000-0001-6143-4716

REFERENCES

Aykanat, T., C. A. Bryden, and D. D. Heath. 2012. "Sex-Biased Genetic Component Distribution among Populations: Additive Genetic and Maternal Contributions to Phenotypic Differences among Populations of Chinook Salmon." *Journal of Evolutionary Biology* 25: 682–690.

Burt, J. M., S. G. Hinch, and D. A. Patterson. 2012. "Parental Identity Influences Progeny Responses to Incubation Thermal Stress in Sockeye Salmon *Onchorhynchus nerka*." *Journal of Fish Biology* 80: 444–462.

Bylemans, J., L. Marques da Cunha, S. Sarmiento Cabello, D. Nusbaumer, A. Uppal, and C. Wedekind. 2024. "Sex-Specific Effects of Inbreeding in Juvenile Brown Trout." *Molecular Ecology* 33: e17298.

Carlson, S. M., and T. R. Seamons. 2008. "A Review of Quantitative Genetic Components of Fitness in Salmonids: Implications for Adaptation to Future Change." *Evolutionary Applications* 1: 222–238.

Clark, E. S., M. Pompini, L. Marques da Cunha, and C. Wedekind. 2014. "Maternal and Paternal Contributions to Pathogen Resistance Dependent on Development Stage in a Whitefish (Salmonidae)." *Functional Ecology* 28: 714–723.

Clark, E. S., M. Pompini, A. Uppal, and C. Wedekind. 2016. "Genetic Correlations and Little Genetic Variance for Reaction Norms May Limit Potential for Adaptation to Pollution by Ionic and Nanoparticulate Silver in a Whitefish (Salmonidae)." *Ecology and Evolution* 6: 2751–62.

Clark, E. S., R. B. Stelkens, and C. Wedekind. 2013. "Parental Influences on Pathogen Resistance in Brown Trout Embryos and Effects of Outcrossing within a River Network." *PLoS One* 8: e57832.

Clark, E. S., L. G. E. Wilkins, and C. Wedekind. 2013. "MHC Class I Expression Dependent on Bacterial Infection and Parental Factors in Whitefish Embryos (Salmonidae)." *Molecular Ecology* 22: 5256–69.

Colihueque, N. 2010. "Genetics of Salmonid Skin Pigmentation: Clues and Prospects for Improving the External Appearance of Farmed Salmonids." *Reviews in Fish Biology and Fisheries* 20: 71–86.

Dormann, C. F., J. Elith, S. Bacher, C. Buchmann, G. Carl, G. Carré, J. R. G. Marquéz, et al. 2013. "Collinearity: A Review of Methods to Deal with it and a Simulation Study Evaluating their Performance." *Ecography* 36: 27–46.

Evans, M. L., and B. D. Neff. 2009. "Non-additive Genetic Effects Contribute to Larval Spinal Deformity in Two Populations of Chinook Salmon (*Oncorhynchus tshawytscha*)." *Aquaculture* 296: 169–173.

Evans, M. L., B. D. Neff, and D. D. Heath. 2010. "Quanitative Genetic and Translocation Experiments Reveal Genotype-by-Environment Effects on Juvenile Life-History Traits in Two Populations of Chinook Salmon (*Oncorhynchus tshawytscha*)." *Journal of Evolutionary Biology* 23: 687–698.

Forest, A. R., C. A. Semeniuk, D. D. Heath, and T. E. Pitcher. 2016. "Additive and Non-additive Genetic Components of the Jack Male Life History in Chinook Salmon (*Oncorhynchus tshawytscha*)." *Genetica* 144: 477–485.

Gomez, E. K., A. Chaiyasarikul, B. Guell, and K. M. Warkentin. 2023. "Developmental Changes in Red-Eyed Treefrog Embryo Behavior Increase Escape-Hatching Success in Wasp Attacks." *Behavioral Ecology and Sociobiology* 77: 52.

Houde, A. L., C. C. Wilson, and T. E. Pitcher. 2016. "Genetic Architecture and Maternal Contributions of Early-Life Survival in Lake Trout *Salvelinus namaycush*." *Journal of Fish Biology* 88: 2088–94.

Houde, A. L. S., C. C. Wilson, and B. D. Neff. 2013. "Genetic Architecture of Survival and Fitness-Related Traits in Two Populations of Atlantic Salmon." *Heredity* 111: 513–19.

Huuskonen, H., J. Kekäläinen, B. Panda, T. Shikano, and R. Kortet. 2011. "Embryonic Survival and Larval Predator-Avoidance Ability in Mutually Ornamented Whitefish." *Biological Journal of the Linnean Society* 103: 593–601.

Jacob, A., G. Evanno, B. A. von Siebenthal, C. Grossen, and C. Wedekind. 2010. "Effects of Different Mating Scenarios on Embryo Viability in Brown Trout." *Molecular Ecology* 19: 5296–5307.

Jacob, A., S. Nusslé, A. Britschgi, G. Evanno, R. Müller, and C. Wedekind. 2007. "Male Dominance Linked to Size and Age, but Not to 'Good Genes' in Brown Trout (*Salmo trutta*)." *BMC Evolutionary Biology* 7: 207.

Janhunen, M., J. Kekläinen, R. Kortet, P. Hyvarinen, and J. Piironen. 2011. "No Evidence for an Indirect Benefit from Female Mate Preference in Arctic Charr *Salvelinus alpinus*, but Female Ornamentation Decreases Offspring Viability." *Biological Journal of the Linnean Society* 103: 602–611.

Jones, O. R., and J. L. Wang. 2010. "COLONY: A Program for Parentage and Sibship Inference from Multilocus Genotype Data." *Molecular Ecology Resources* 10: 551–55.

Kekäläinen, J., G. Rudolfsen, M. Janhunen, L. Figenschou, N. Peuhkuri, N. Tamper, and R. Kortet. 2010. "Genetic and Potential Non-genetic Benefits Increase Offspring Fitness of Polyandrous Females in Non-resource Based Mating System." *BMC Evolutionary Biology* 10: 20.

Li, M., and J. F. Leatherland. 2012. "The Interaction between Maternal Stress and the Ontogeny of the Innate Immune System during Teleost Embryogenesis: Implications for Aquaculture Practice." *Journal of Fish Biology* 81: 1793–1814.

Lieke, T., C. E. W. Steinberg, S. Bittmann, S. Behrens, S. H. Hoseinifar, T. Meinelt, K. Knopf, and W. Kloas. 2021. "Fulvic Acid Accelerates Hatching and Stimulates Antioxidative Protection and the Innate Immune Response in Zebrafish Larvae." *Science of the Total Environment* 796: 148780.

Marques da Cunha, L., A. Uppal, E. Seddon, D. Nusbaumer, E. L. M. Vermeirssen, and C. Wedekind. 2019. "No Additive Genetic Variance for Tolerance to Ethynylestradiol Exposure in Natural Populations of Brown Trout (*Salmo trutta*)." *Evolutionary Applications* 12: 940–950.

Marques da Cunha, L., L. G. E. Wilkins, L. Menin, D. Ortiz, V. Vocat-Mottier, and C. Wedekind. 2018. "Consumption of Carotenoids Not Increased by Bacterial Infection in Brown Trout Embryos (*Salmo trutta*)." *PLoS One* 13: e0198834.

Muñoz, N. J., A. P. Farrell, J. W. Heath, and B. D. Neff. 2014. "Adaptive Potential of a Pacific Salmon Challenged by Climate Change." *Nature Climate Change* 5: 163–66.

Neff, B. D., and T. E. Pitcher. 2005. "Genetic Quality and Sexual Selection: An Integrated Framework for Good Genes and Compatible Genes." *Molecular Ecology* 14: 19–38.

Nusbaumer, D., L. Garaud, L. Ançay, and C. Wedekind. 2021. "Sex-Specific Stress Tolerance in Embryos of Lake Char (*Salvelinus umbla*)." *Frontiers in Ecology and Evolution* 9: 768263.

Nusbaumer, D., L. Marques da Cunha, and C. Wedekind. 2019. "Sperm Cryopreservation Reduces Offspring Growth." *Proceedings of the Royal Society of London Series B-Biological Sciences* 286: 20191644.

Nusbaumer, D., L. Marques da Cunha, and C. Wedekind. 2021. "Testing for Population Differences in Evolutionary Responses to Pesticide Pollution in Brown Trout (*Salmo trutta*)." *Evolutionary Applications* 14: 462–475.

Ødegård, J., M. Baranski, B. Gjerde, and T. Gjedrem. 2011. "Methodology for Genetic Evaluation of Disease Resistance in Aquaculture Species: Challenges and Future Prospects." *Aquaculture Research* 42: 103–114.

Palejowski, H., J. Bylemans, V. Ammann, L. Marques da Cunha, D. Nusbaumer, I. Castro, A. Uppal, K. B. Mobley, S. Knörr, and C. Wedekind. 2022. "Sex-Specific Life History Affected by Stocking in Juvenile Brown Trout." *Frontiers in Ecology and Evolution* 10: 869925.

Pompini, M., E. S. Clark, and C. Wedekind. 2013. "Pathogen-Induced Hatching and Population-Specific Life-History Response to Waterborne Cues in Brown Trout (*Salmo trutta*)." *Behavioral Ecology and Sociobiology* 67: 649–656.

Sopinka, N. M., P. M. Capelle, C. A. D. Semeniuk, and O. P. Love. 2017. "Glucocorticoids in Fish Eggs: Variation, Interactions with the Environment, and the Potential to Shape Offspring Fitness." *Physiological and Biochemical Zoology* 90: 15–33.

Stelkens, R. B., G. Jaffuel, M. Escher, and C. Wedekind. 2012. "Genetic and Phenotypic Population Divergence on a Microgeographic Scale in Brown Trout." *Molecular Ecology* 21: 2896–2915.

Stelkens, R. B., M. Pompini, and C. Wedekind. 2014. "Testing the Effects of Genetic Crossing Distance on Embryo Survival within a Metapopulation of Brown Trout (*Salmo trutta*)." *Conservation Genetics* 15: 375–386.

Thorn, M. W., and Y. E. Morbey. 2018. "Egg Size and the Adaptive Capacity of Early Life History Traits in Chinook Salmon (*Oncorhynchus tshawytscha*)." *Evolutionary Applications* 11: 205–219.

von Siebenthal, B. A., A. Jacob, and C. Wedekind. 2009. "Tolerance of Whitefish Embryos to *Pseudomonas fluorescens* Linked to Genetic and Maternal Effects, and Reduced by Previous Exposure." *Fish & Shellfish Immunology* 26: 531–35.

von Siebenthal, B. A., M. Pompini, R. Müller, and C. Wedekind. 2017. "Pros and Cons of a Fluorescent Pigment Mass-Marking Technique: A 5-Year Long Study on Grayling (*Thymallus thymallus* L.)." *Fisheries Management and Ecology* 24: 173–75.

Warkentin, K. M. 2005. "How Do Embryos Assess Risk? Vibrational Cues in Predator-Induced Hatching of Red-Eyed Treefrogs." *Animal Behaviour* 70: 59–71.

Warkentin, K. M., C. R. Currie, and S. A. Rehner. 2001. "Egg-Killing Fungus Induces Early Hatching of Red-Eyed Treefrog Eggs." *Ecology* 82: 2860–69.

Wedekind, C. 2002. "Induced Hatching to Avoid Infectious Egg Disease in Whitefish." *Current Biology* 12: 69–71.

Wedekind, C., J. Bylemans, L. Marques da Cunha, S. Sarmiento Cabello, D. Nusbaumer, and A. Uppal. 2024a. "Data from: Sex-Specific Effects of Inbreeding in Juvenile Brown Trout." Dryad, Dataset. https://doi.org/10.5061/dryad.2ngf1vhv4.

Wedekind, C., J. Bylemans, L. Marques da Cunha, S. Sarmiento Cabello, D. Nusbaumer, and A. Uppal. 2024b. "Data from: Sex-Specific Effects of Inbreeding in Juvenile Brown Trout." Zenodo, Software. https://doi.org/10.5281/zenodo.8065568.

Wedekind, C., M. O. Gessner, F. Vazquez, M. Maerki, and D. Steiner. 2010. "Elevated Resource Availability Sufficient to

Turn Opportunistic into Virulent Fish Pathogens." *Ecology* 91: 1251–56.

Wedekind, C., A. Jacob, G. Evanno, S. Nusslé, and R. Müller. 2008. "Viability of Brown Trout Embryos Positively Linked to Melanin-Based but Negatively to Carotenoid-Based Colours of their Fathers." *Proceedings of the Royal Society B-Biological Sciences* 275: 1737–44.

Wedekind, C., and R. Müller. 2005. "Risk-Induced Early Hatching in Salmonids." *Ecology* 86: 2525–29.

Wedekind, C., B. von Siebenthal, and R. Gingold. 2007. "The Weaker Points of Fish Acute Toxicity Tests and how Tests on Embryos Can Solve some Issues." *Environmental Pollution* 148: 385–89.

Wilkins, L. G. E., L. Fumagalli, and C. Wedekind. 2016. "Effects of Host Genetics and Environment on Egg-Associated Microbiotas in Brown Trout (*Salmo trutta*)." *Molecular Ecology* 25: 4930–45.

Wilkins, L. G. E., L. Marques da Cunha, L. Menin, D. Ortiz, V. Vocat-Mottier, M. Hobil, D. Nusbaumer, and C. Wedekind. 2017. "Maternal Allocation of Carotenoids Increases Tolerance to Bacterial Infection in Brown Trout." *Oecologia* 185: 351–363.

Wilkins, L. G. E., L. Marques da Cunha, L. Menin, D. Ortiz, V. Vocat-Mottier, M. Hobil, D. Nusbaumer, and C. Wedekind. 2018. "Data from: Maternal Allocation of Carotenoids Increases Tolerance to Bacterial Infection in Brown Trout." Dryad, Dataset https://doi.org/10.5061/dryad.sj416.

Yaripour, S., H. Huuskonen, T. Rahman, J. Kekälinen, J. Akkanen, M. Magris, P. V. Kipriianov, and R. Kortet. 2021. "Pre-Fertilization Exposure of Sperm to Nano-Sized Plastic Particles Decreases Offspring Size and Swimming Performance in the European Whitefish (*Coregonus lavaretus*)." *Environmental Pollution* 291: 118196.

Yaripour, S., J. Kekäläinen, P. Hyvärinen, S. Kaunisto, J. Piironen, A. Vainikka, M.-L. Koljonen, J. Koskiniemi, and R. Kortet. 2020. "Does Enriched Rearing during Early Life Affect Sperm Quality or Skin Colouration in the Adult Brown Trout?" *Aquaculture* 529: 735648.

SUPPORTING INFORMATION

Additional supporting information can be found online in the Supporting Information section at the end of this article.

How to cite this article: Bylemans, Jonas, Lucas Marques da Cunha, Laetitia G. E. Wilkins, David Nusbaumer, Anshu Uppal, and Claus Wedekind. 2024. "Growth of Brown Trout in the Wild Predicted by Embryo Stress Reaction in the Laboratory." *Ecology* e4303. https://doi.org/10.1002/ecy.4303