


# Links between parental life histories of wild salmon and the telomere lengths of their offspring

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## Abstract

The importance of parental contributions to offspring development and subsequent performance is self-evident at a genomic level; however, parents can also affect offspring fitness by indirect genetic and environmental routes. The life history strategy that an individual adopts will be influenced by both genes and environment; and this may have important consequences for offspring. Recent research has linked telomere dynamics (i.e., telomere length and loss) in early life to future viability and longevity. Moreover, a number of studies have reported a heritable component to telomere length across a range of vertebrates, although the effects of other parental contribution pathways have been far less studied. Using wild Atlantic salmon with different parental life histories in an experimental split-brood in vitro fertilization mating design and rearing the resulting families under standardized conditions, we show that there can be significant links between parental life history and offspring telomere length (studied at the embryo and fry stage). Maternal life history traits, in particular egg size, were most strongly related to offspring telomere length at the embryonic stage, but then became weaker through development. In contrast, paternal life history traits, such as the father's growth rate in early life, had a greater association in the later stages of offspring development. However, offspring telomere length was not significantly related to either maternal or paternal age at reproduction, nor to paternal sperm telomere length. This study demonstrates both the complexity and the importance of parental factors that can influence telomere length in early life.

## KEYWORDS

egg size, life history, parental effects, *Salmo*, telomere

## 1 | INTRODUCTION

Parental contributions to offspring fitness can occur by many routes, involving both direct and indirect genetic and environmental effects (Rossiter, 1996; Wolf & Wade, 2009). Many species show substantial variation in a number of life history traits, such as age and size

at reproduction, which are influenced by both genetic and environmental factors. The realized life history of an individual may in turn affect its pattern of investment in offspring, and so have important consequences for offspring fitness. For example, the relationship between parental age at reproduction and offspring lifespan has been established for a number of organisms, with offspring from

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older parents often displaying reduced longevity (Bouwhuis, Vedder, & Becker, 2015; Fox, Bush, & Wallin, 2003; Garcia-Palomares et al., 2009; Gavrilov & Gavrilova, 1997). The molecular mechanisms underlying any such effects have been little studied. However, recent research has identified the importance of telomeres in patterns of lifetime fitness and longevity in wild animals (Monaghan, 2010). With this in mind, variation in offspring telomere length may provide a useful indicator with which to examine the relationship between various parental life history traits and offspring fitness.

Telomeres cap the ends of eukaryotic chromosomes and play an important role in chromosome protection (for reviews, see Blackburn, 1991; Campisi, S-h, Lim, & Rubio, 2001; Monaghan, 2010). Telomere loss occurs at each cell division as a result of the “end replication problem,” but the amount of loss may also be influenced by conditions within the cell, including levels of oxidative damage to the telomeric DNA (Chan & Blackburn, 2004; Oikawa & Kawanishi, 1999); however, more detailed studies are still needed to test this effect in vivo (Boonekamp, Bauch, Mulder, & Verhulst, 2017). A relatively short telomere length is generally considered indicative of poor biological state, which may be linked to reduced adult performance/lifespan and/or increased disease susceptibility (Bauch, Becker, & Verhulst, 2014; Heidinger et al., 2012; Ilmonen, Kotrschal, & Penn, 2008; Salmón, Nilsson, Watson, Bensch, & Isaksson, 2017). Therefore, both an animal's initial telomere length and its subsequent rate of loss (Boonekamp, Mulder, Salomons, Dijkstra, & Verhulst, 2014) are of potential importance to lifetime fitness and longevity.

There is considerable variation in telomere length, both among and within species (Gorbunova & Seluanov, 2009; Monaghan, 2010); however, the determinants of this variation are still not fully understood. There is increasing evidence that telomere loss (and subsequent telomere length) in wild animals are partially under the influence of ecological conditions. For example, a number of studies have linked telomere length to environmental stressors, such as in utero stress (Haussmann, Longenecker, Marchetto, Juliano, & Bowden, 2011; Marchetto et al., 2016), disturbance (Herborn et al., 2014) and sibling competition (Cram, Monaghan, Gillespie, & Clutton-Brock, 2017). The pattern of growth is also likely to influence telomere loss and several studies have found faster growth and/or larger body size to be associated with reduced telomere length (Noguera, Metcalfe, Boner, & Monaghan, 2015; Pauliny, Devlin, Johnsson, & Blomqvist, 2015; Ringsby et al., 2015).

Direct genetic effects are also likely to be important, and a number of studies have demonstrated a heritable component to telomere length (e.g., Asghar, Bensch, Tarka, Hansson, & Hasselquist, 2015; Njajou et al., 2007; Nordfjall, Svenson, Norrback, Adolfsson, & Roos, 2010; Olsson et al., 2011; Reichert et al., 2015). These studies have mostly found a positive relationship: parents with relatively longer telomeres at the time of reproduction produce offspring with relatively longer telomeres. There is inconsistency within these studies as to whether the effect is stronger through the mother or the father, although there is some indication of taxon-specific differences: human studies mostly report stronger paternal inheritance (e.g., Njajou et al., 2007; Nordfjall et al., 2010), while bird studies

mostly report stronger maternal inheritance (e.g., Asghar et al., 2015; Reichert et al., 2015).

In addition to direct genetic effects, there are also several routes whereby the environment experienced by a parent may affect the telomere length of its offspring (i.e., parental effects). For example, there are links between a mother's physiological state and the quality of her eggs (and hence the developmental environment and condition of her offspring) (Blount, Surai, Nager, et al., 2002; Tobler & Sandell, 2009), which in turn may influence the future telomere length of those offspring. In support of this, Noguera, Metcalfe, Reichert, and Monaghan (2016) found that offspring telomere length in zebra finches was negatively correlated with ovulation order within a clutch: the first laid eggs in a clutch developed into offspring with a relatively longer telomere length, while offspring from the later laid eggs in the same clutch had the shortest telomeres. This effect may be driven by variation in egg composition, as key egg components, such as maternally derived antioxidants, are known to change with laying order in birds (Royle, Surai, & Hartley, 2003).

Atlantic salmon *Salmo salar* provide the opportunity to examine various parental effects on telomere length in offspring. As fertilization is external, their matings can be relatively easily controlled using in vitro fertilizations (IVF). Salmon produce large clutches and have no confounding effects of parental care, as there is none. Additionally, the collection of gametes and offspring for telomere analysis is straightforward. Wild salmon display extensive within-population variation in life history strategies, hence variation in parental state at the time of fertilization (for reviews, see Fleming, 1996; Klemetsen et al., 2003; Jonsson & Jonsson, 2011). Eggs are laid in freshwater, where juveniles can spend up to 6 years (dependent on growth rate) before migrating to sea (Metcalfe & Thorpe, 1990; Økland, Jonsson, Jensen, & Hansen, 1993). They also vary in the number of years spent at sea (categorized as 1 sea winter (1SW) or multisea winter (MSW)) before returning to their native river to reproduce, and this variation is thought to be under both genetic and environmental determination (Barson et al., 2015; Fleming, 1998; Gardner, 1976). Multisea winter fish have mostly spent two (occasionally three or more) years at sea and tend to be much larger in size at the time of spawning (Trust, 2012). Age at reproduction is therefore a combination of the time spent in the two habitats. There is a correlation between female body size and average egg size (Fleming, 1996), and therefore, MSW mothers, in general, produce significantly larger eggs than 1SW mothers. It is also possible for males (very rarely females) to become precociously mature as parr (the freshwater juvenile stage), prior to seaward migration. These precocious male parr adopt an alternative reproductive strategy of “sneaky” matings (Baum, Laughton, Armstrong, & Metcalfe, 2004; Fleming, 1996).

In a field study of Atlantic salmon, we recently showed that paternal life history can influence offspring telomere length (at the fry stage), but that there was also a strong influence of environmental conditions experienced by the fry in the wild (McLennan et al., 2016). A second recent study also found that stream temperature influenced telomere lengths in wild juvenile salmonids, partly because of its influence on growth rates (Debes, Visse, Panda, Ilmonen, & Vasemägi,

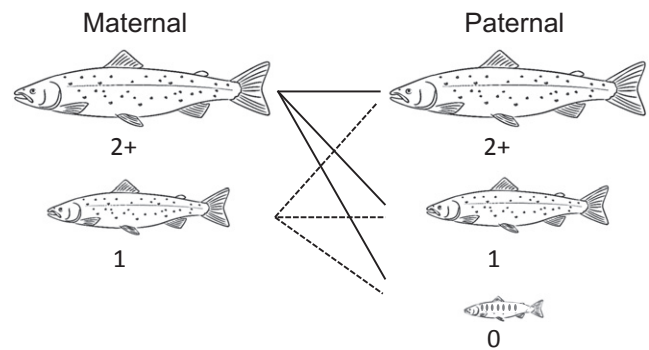
2016). Therefore, in order to detect parental effects on offspring telomere length, it is necessary to control for environmental conditions. Here, we use that approach to evaluate the relationship between parental life history (years spent in freshwater and seawater, overall age at reproduction, egg size) and offspring telomere lengths in Atlantic salmon. More specifically, we test two hypotheses: (i) that larger, better quality eggs will produce offspring with longer telomeres and (ii) parents that are larger (and/or older) at the time of reproduction will produce offspring with shorter telomeres.

## 2 | METHODS

### 2.1 | In vitro fertilization

In vitro fertilization was conducted on wild sexually mature salmon between 28 November and 30 November 2012, using a split-brood IVF design that utilized all readily identifiable parental life history types (Figure 1). In each replication of the mating design, the clutches of two female fish (one 1SW and one MSW) were each divided into three equal portions; each portion was then fertilized with sperm from one of three male fish (one 1SW, one MSW and one precocious male parr) to produce six half-sib families, with contrasting parental life histories. This design was replicated 10 times (using new fish each time) to produce 60 half-sib families in total, based on 20 mothers and 30 fathers. A small sample of tissue was taken from the adipose fin of each parent and flash-frozen for subsequent analysis of parental telomere length. Use of the fin allowed noninvasive measurement of telomeres, and a study on the closely related Brown trout *Salmo trutta* found fin telomere length to correlate significantly with the telomere length of other tissues (Debes et al., 2016). A small sample of sperm was taken from each of the males and flash-frozen for subsequent analysis of paternal gamete telomere length. A small random subsample of eggs was also taken from each of the females and flash-frozen, with the aim of determining maternal gamete telomere length. However, we were unable to recover sufficient nucleic DNA from the eggs, most likely because each egg possessed only one nucleus. These egg samples were still used to calculate average egg size per family. The mass of an Atlantic salmon egg can vary significantly among females, but is relatively uniform within a clutch (Fleming, 1996). To calculate egg mass for this study, 10 of the frozen eggs from each female were weighed separately (to 0.01 g) and the average egg weight for each family was calculated. Lastly, a small sample of scales was removed from each of the parent fish to be used for scalimetry analysis; where the circuli of a fish scale is used to determine freshwater age and sea water age (Shearer, 1992). A detailed outline of our IVF procedure is available in the SI.

In addition to the samples taken from each parent fish used in our split-brood IVF design, we also sampled supplementary parental tissue from an experiment that was being simultaneously conducted at the same field site (for an outline of the experiment see Burton et al. (2016)). Doing so added 42 additional parent fish (21 male and 21 female), which allowed a larger sample size for the analysis of



**FIGURE 1** A schematic diagram of the split-brood in vitro fertilization design, utilizing all available parent types of wild Atlantic salmon. Numbers depict the number of years spent at sea. 2+ = multisea winter parent fish (MSW), 1 = 1 sea winter (1SW) parent fish and 0 = male parr that matured in freshwater and had spent 0 years in sea water

variation in parental telomere length. For each of these supplementary fish, we recorded their body measurements (fork length to 0.5 cm; body mass to 0.1 g), their fin tissue was sampled for telomere analysis, and a sample of scales was taken for subsequent scalimetry analysis to confirm the period that they had spent in freshwater and at sea. All parent fish from the supplementary experiment were 1SW, but they differed in weight, length, sex and years in FW.

### 2.2 | Rearing conditions for offspring

Several hours after fertilization, eggs were transferred to the nearby fish hatchery at Contin, Scotland. They remained in separate family groups at the hatchery, under ambient water temperatures ( $3.82 \pm SD 0.69^\circ\text{C}$ ) until they had reached the more stable “eyed egg” stage of development (~2 months old). During this time, they were checked daily and any dead eggs were removed. Mortality was minimal for all families. On 5 March 2013, when all eggs had passed the eyed stage, and so were safe to move, a small sample of eggs ( $n = 50$ ) was counted for each family and transferred to the aquarium facilities at the University of Glasgow. Five eggs per family were immediately sampled and stored in 95%–100% ethanol for subsequent telomere analysis of the embryo stage. The remainder of the eggs transferred to Glasgow were held as separate family groups, in 2 L compartments of a recirculating stream system. The water temperature was initially held at  $6^\circ\text{C}$ , to match the ambient temperature at the initial hatchery facilities, but was then slowly raised to  $13^\circ\text{C}$  over the first 30 days, as would occur in the wild.

Eggs hatched into alevins (free swimming, but with an attached yolk sac) from March 25th to April 3rd. From this point onwards, a small proportion of water was changed daily to maintain water quality within the stream system. The alevins reached the first-feeding stage (when the majority of the yolk sac had been utilized and they began feeding on exogenous food) over the period April 15–22. Fry were then fed to satisfaction several times per day with commercial pelleted food (EWOS Ltd, stage 1). Food was left in each family

compartment for 30–60 min, after which any uneaten food was removed. Fry remained in the stream system until 8 weeks after first feeding. At this stage, when the fry were approximately 186 days postfertilization (4 June 2013), 10 fry per family were euthanized, measured for fork length and body mass and then preserved in 95%–100% ethanol for subsequent telomere analysis of the fry stage.

This experiment was approved by the University of Glasgow local ethical review panel, and all procedures were carried out under the jurisdiction of a UK Home Office project licence (PPL 60/4292).

## 2.3 | Telomere analysis

DNA was extracted from parent, embryo and fry tissue using the DNeasy Blood and Tissue Kit (Qiagen), as described in McLennan et al. (2016). For the sperm analysis, sperm was diluted 1/200 with sterile PBS solution. One hundred microliter of the diluted sperm solution was added to 100  $\mu$ l of buffer X2 (20 mM Tris-Cl pH 8.0, 20 mM EDTA, 200 mM NaCl, 80 mM DTT, 4% SDS, 250  $\mu$ g/ml proteinase K) and incubated at 55°C until tissue was fully lysed. Each set of DNA extractions conducted also included a negative control which contained all of the reagents, but without any tissue. This was used to check for contamination during the lysis and extraction steps. DNA concentration and purity were measured spectrophotometrically using a Nanodrop 8000.

Telomere length was measured in all samples using quantitative PCR, and data were analysed using QBASE software for windows (Hellemans, Mortier, De Paepe, Speleman, & Vandesompele, 2007), both as described in McLennan et al. (2016). Atlantic salmon chromosomes are not thought to have a significant amount of interstitial telomere sites (Perez, Moran, & Garcia-Vazquez, 1999), which could potentially add noise to the qPCR measurement. In brief, this qPCR method provides a relative measure of telomere length (RTL) and is calculated as a ratio (T/S) of telomere repeat copy number (T) to a control, single copy gene number (S). The Atlantic salmon glyceraldehyde-3-phosphate dehydrogenase (GAPDH) gene was chosen as the single copy gene (GenBank Accession no.: NM\_001123561). In addition to the samples, each qPCR plate also included a sixfold serial dilution of a reference sample (1.25–40 ng/well), a “golden reference” sample and a nontarget control. The DNA for the serial dilution was a pool of 60 samples drawn from all life stages (embryo, fry and adult). The serial dilution was used to generate a standard curve for calculation of assay efficiencies. The “golden standard” was a pool of DNA from 20 of the experimental samples that included all life stages, and was used as the same reference sample across all plates. The mean assay efficiencies for the telomere and GAPDH were 101.0 and 100.7, respectively, well within the acceptable range (85–115). The average intraplate variation in the  $C_t$  values was 1.04 for the telomere assay and 0.81 for the GAPDH assay. The average interplate variation in the  $C_t$  values was 1.85 for the telomere assay and 0.90 for the GAPDH assay.

## 2.4 | Data analysis

All statistical analyses were conducted using R version 3.4.0 software. We measured/calculated the following four variables, which were used as dependent variables in the analyses: 1—parental relative telomere length (subsequently referred to as *parental RTL*), measured at the time of spawning, 2—sperm relative telomere length (*sperm RTL*), 3—embryonic relative telomere length (*embryo RTL*), measured at the eyed egg stage and 4—fry relative telomere length (*fry RTL*), measured 8 weeks after first feeding. All telomere measurements were log-transformed. Also, note that for variables 3–4, a mean value per half-sib family was used in all subsequent analyses.

Due to the large number of variables in each model, we used a Pearson correlation coefficient matrix (Table S1) to assess potential collinearity between explanatory variables (with problematic collinearity being defined as a coefficient > 0.7). Fry weight and fry length were highly collinear (Pearson  $r = .90$ ,  $p < .001$ ); therefore, only fry weight was used in analyses, where appropriate. Parental weight was highly collinear with parental SW age (paternal = Pearson  $r = .95$ ,  $p < .001$ ; maternal = Pearson  $r = .89$ ,  $p < .001$ ) as MSW fish are generally much larger than 1SW fish; therefore, only parental SW age was used in models, where appropriate. Lastly, paternal total age was not independent of the number of years each father had spent in FW (Pearson  $r = .83$ ,  $p < .001$ ) or at sea (Pearson  $r = .90$ ,  $p < .001$ ), and so for the models investigating variation in embryo RTL and fry RTL we ran two separate models, one using total ages of each parent and another separating these total ages into years spent in fresh water and sea water.

Factors affecting variation in parental RTL (using the supplementary parent data in addition to the parent data from the split-brood IVF experiment) were assessed by general linear models (GLM,  $n = 92$ ) which included sex and age (either total age or separated years spent in FW and SW). The effects of paternal RTL and paternal age (either total age or separated into years spent in FW and SW) on sperm RTL (using only the paternal data from the split-brood IVF experiment) were assessed by GLM ( $n = 30$ ). Details of the full GLM models, containing all considered main effects, are outlined in Table S2.

Variation in embryo RTL and fry RTL was assessed by linear mixed models (LME,  $n = 60$  families) using the lme4 and lmerTest functions (Bates, Maechler, Bolker, & Walker, 2015; Kuznetsova, Brockhoff, & Christensen, 2014). Maternal ID and paternal ID were included as random factors to control for nonindependence of half-siblings, along with the following independent variables, where appropriate: maternal and paternal years in FW and SW (or total age, in the alternative models), maternal relative telomere length (maternal RTL), paternal relative telomere length (paternal RTL), sperm relative telomere length for each father (sperm RTL), average egg weight for each family (egg weight) and average 8-week-old fry weight for each family (fry weight).

For embryo RTL and fry RTL, details of the full LME models containing all considered main effects are given in Table S3. The models were then simplified using backwards model selection, with the least

significant variable being systematically removed from a model until models contained only significant terms (Table 1). For the purpose of exploration, we also took an AIC-based approach for the embryo RTL and fry RTL models, using the MuMin package and associated dredge function. All plausible models with a delta AIC less than 5 are presented in the Results (Table 2) alongside those from the backwards elimination approach.

### 3 | RESULTS

#### 3.1 | Parental and sperm relative telomere lengths

Parental RTL at the time of fertilization did not vary with sex or age (whether expressed as total age or separated into years spent in FW and SW; Table S2). While there was heterogeneity between fathers with respect to sperm RTL, this variation was not explained by paternal age (either total age or separated years spent in FW and SW; Table S2). Sperm RTL did not contribute significantly to any of the models of offspring telomere length (embryo RTL or Fry RTL; Table S3).

#### 3.2 | Embryo relative telomere length

We found that initial egg weight was a significant predictor of embryo RTL: heavier eggs resulted in offspring (embryos) with relatively longer telomeres (Table 1, Figure 2). This was also supported by our AIC approach, as egg size was included in the top two models (Table 2). Neither maternal nor paternal age (either total age or separate FW/SW ages) had any significant effect. However, there was an effect of maternal RTL: mothers with relatively longer telomeres at the time of spawning produced embryos with a relatively longer telomere length (Table 1, Figure 3). However, the effect size of maternal TL was smaller than for egg size, and maternal TL was only included in the second top model in the AIC approach (Table 2).

#### 3.3 | Fry relative telomere length

Fry RTL was negatively correlated with fry growth rate, with fry that had reached a larger body mass having shorter telomeres (Table 1, Figure 4). Although egg size was removed from the fry RTL models during backward model selection, it was identified as a predictor of fry RTL in the AIC approach (Table 2), most likely due to its association with fry size (egg size and fry size being positively correlated: Pearson's  $\rho = 0.56$ ,  $p < .001$ ,  $n = 59$ , based on mean values per family). At a family level, fry RTL was positively related to embryo RTL, as families with a relatively greater telomere length at the embryo stage produced fry with longer telomeres (Table 1, Figure 5). Fry RTL was not significantly affected by maternal age (either total age or separate FW/SW ages), but there was a significant negative effect of paternal years in FW (Table 1), with offspring (fry) from fathers that had spent the least time in freshwater having the longest telomeres (Figure 6). However, paternal years in FW was not included in any of the top models produced by the AIC approach

(Table 2), suggesting that while significant, it was not a robust predictor of fry telomere length.

## 4 | DISCUSSION

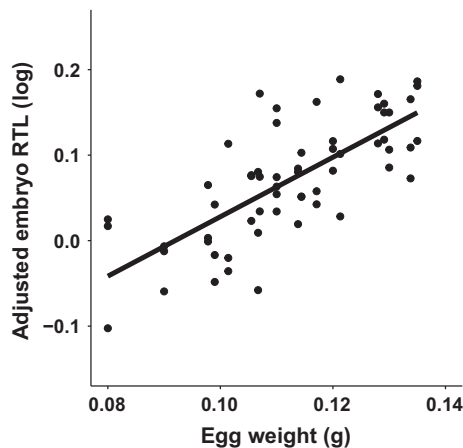
This study demonstrates that rearing the offspring of wild parents under controlled environmental conditions can reveal significant links between parental life history and offspring telomere length. In particular, the size of eggs produced by the mother was an important predictor of telomere length at the embryonic stage (and indirectly at the fry stage, by means of variation in fry weight). The father's early growth rate (and hence time taken to reach the marine phase) had an association in the later stages of development. We found no link between parental age and offspring telomere length in salmon, despite this previously being found to be one of the most pervasive parental effects on offspring telomere length across a range of taxa (e.g., Asghar et al., 2015; Broer et al., 2013; De Meyer et al., 2007; Heidinger et al., 2016). However, it may be the case that our relatively smaller sample size (in comparison to these other studies) did not allow enough power to statistically detect a parental age effect. It is also possible that our cross-sectional approach allowed the selective disappearance of certain individuals, which in turn may have masked potential parental age effects.

The telomere length of salmon embryos correlated positively with the size of the egg from which they had hatched. Neither the paternal nor the maternal age had a significant effect on embryo telomere length, but there was a relationship with maternal telomere length, as mothers with a relatively longer telomere length at the time of reproduction produced offspring with relatively longer telomeres. The effects of maternal telomere length and egg size may share a common cause. Telomere length is considered an index of an individual's physiological state and there are positive links between a mother's physiological state and the quality of her eggs (Blount, Surai, Houston, & Møller, 2002; Tobler & Sandell, 2009). It is possible that these effects arose through variation in egg provisioning (Donelson, Munday, & McCormick, 2009; Van Leeuwen et al., 2015) and nongenetic maternal effects arising through differential egg provisioning have been reported in a range of species (Costantini, 2010; Noguera et al., 2016; Royle et al., 2003; Tobler & Sandell, 2009). Intraspecific variation in egg proximate composition (i.e., per cent of water, lipid, protein and carbohydrate) is fairly limited in fish (Kamler, 1992); therefore, larger eggs will generally have more of all macronutrient components than small eggs. This is supported by the fact that larger salmon eggs generally result in larger offspring at emergence (for reviews Fleming, 1996; Jonsson & Jonsson, 2011). It is reasonable to assume that larger eggs could also contain a greater reserve of antioxidants. Oxidative damage may influence telomere loss, and antioxidants have been shown to help mitigate rates of telomere loss during development (Kim & Velando, 2015; Noguera, Monaghan, & Metcalfe, 2015). Therefore, it may be that mothers are influencing the telomere length of their offspring through the provisioning of their eggs (e.g., with antioxidants).

**TABLE 1** Summary of the four final linear mixed-effect models explaining variation in embryo relative telomere length and fry relative telomere length

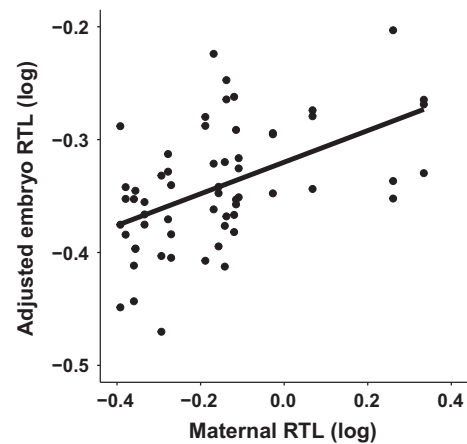
	Independent variable	Parameter estimate	SE	df	T	p
Embryo RTL (including parental age)	Intercept	-0.331	0.08	8.15	-3.91	.004
	Egg weight	3.595	0.74	7.79	4.85	.001
	Maternal RTL	0.140	0.06	10.62	2.28	.044
Embryo RTL (including parental years in FW & SW)	Intercept	-0.331	0.08	8.15	-3.91	.004
	Egg weight	3.595	0.74	7.79	4.85	.001
	Maternal RTL	0.140	0.06	10.62	2.28	.044
Fry RTL (including parental age)	Intercept	0.214	0.08	36.75	2.82	.007
	Embryo RTL	0.424	0.10	45.34	4.10	<.001
	Fry weight	-0.370	0.15	37.94	-2.54	.015
Fry RTL (including parental years in FW & SW)	Intercept	0.302	0.08	39.39	3.84	<.001
	Embryo RTL	0.407	0.11	45.56	3.74	<.001
	Fry weight	-0.372	0.14	35.76	-2.65	.011
	Paternal FW 2 year	-0.082	0.04	22.20	-2.26	.034
	Paternal FW 3 year	-0.117	0.04	20.95	-3.14	.005

All telomere measurements were log-transformed. The main effects initially included in each of the models are outlined in Table S1. Terms were sequentially removed if not significant. Mother ID and father ID were included as random effects to control for nonindependence of half-siblings. See Methods for definitions.



**FIGURE 2** The relationship between egg weight (g) and embryo relative telomere length ( $N = 60$  families). Embryo telomere lengths have been plotted as partial residuals as a function of egg weight when all other significant independent variables = 0. See methods and Table 1 for statistical analysis

The results of the present study show some contrasts with McLennan et al. (2016), which found no effect of egg size on offspring telomere length in salmon embryos or fry. The differences may relate to the environmental conditions under which the offspring developed. The present study was conducted in the laboratory using controlled constant temperatures, standardized fish densities and freely available food. In contrast, the experiment in McLennan et al. (2016) involved temperature variation at both the embryo and the fry stage, as well as the fry being reared in the wild



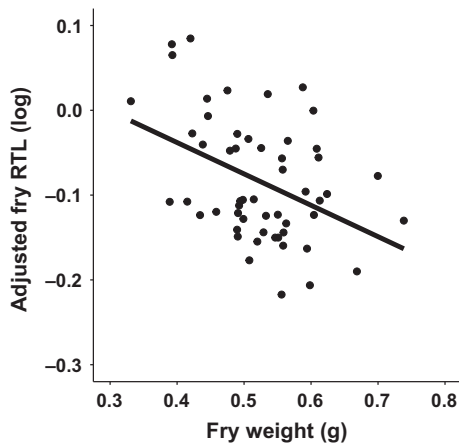
**FIGURE 3** The relationship between maternal relative telomere length and embryo relative telomere length ( $N = 60$  families). Embryo telomere lengths have been plotted as partial residuals as a function of maternal telomere length when all other significant independent variables = 0. See methods and Table 1 for statistical analysis

(with consequent spatial and temporal variability in food supply, temperature, competition and predator levels). Telomere lengths in that study were found to be related to both local temperature and predator levels (see also Debes et al. (2016) for similar patterns in wild trout), and these additional sources of variation may have obscured any effects of egg size. It should be noted that in the present study we used fresh egg weight as the measure of egg size, whereas in McLennan et al. (2016) we used dry egg weight, but this seems

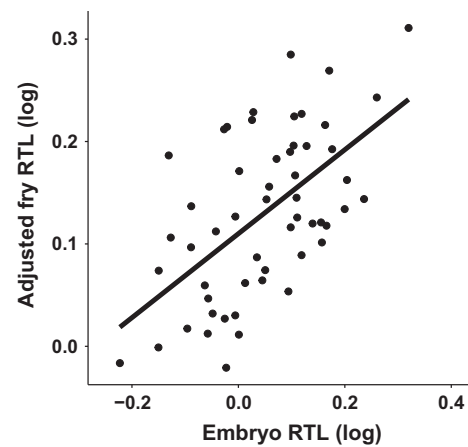
**TABLE 2** Akaike's information criterion ranking of models explaining variation in embryo relative telomere length and fry relative telomere length

	Model <sub>i</sub>	<i>k</i>	$\Delta\text{AICc}$	$\omega_i$
Embryo RTL (including parental age)	Egg weight	4	0	0.659
	Egg weight + Mother RTL	5	1.4	0.327
Embryo RTL (including parental FW/SW years)	Egg weight	4	0	0.650
	Egg weight + Mother RTL	5	1.4	0.322
Fry RTL (including parental age)	Egg weight + Embryo RTL	5	0	0.353
	Embryo RTL + Fry weight	5	0.89	0.227
	Egg weight + Embryo RTL + Fry weight	6	1.24	0.190
	Embryo RTL	4	2.17	0.119
Fry RTL (including parental FW/SW years)	Egg weight + Embryo RTL	5	0	0.349
	Embryo RTL + Fry weight	5	0.89	0.224
	Egg weight + Embryo RTL + Fry weight	6	1.24	0.188
	Embryo RTL	4	2.17	0.118

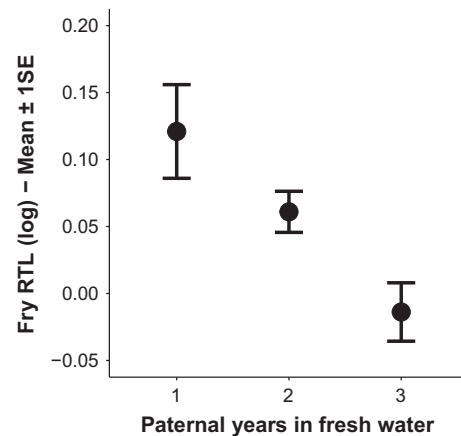
All telomere measurements were log-transformed. For each model, we show number of parameters (*k*), the difference in Akaike's information criterion between that model and the top-ranked model ( $\Delta\text{AICc}$ ) and the Akaike weight of that model ( $\omega_i$ ). All models with a  $\Delta\text{AICc} < 5$  are presented. We outline this AIC ranking for the purposes of data exploration. Final models were not based on this mode of inference.



**FIGURE 4** The relationship between the average fry weight at 186 days postfertilization and average fry relative telomere length for a given family ( $N = 60$  families). Fry telomere lengths have been plotted as partial residuals as a function of fry weight when all other significant independent variables = 0. Plot shows the relationship from the model that included parental years in FW and SW as independent variables. Plot from the alternative model, which used parental age as an independent variable, can be viewed in the supplementary information (Figure S1). See Methods and Table 1 for statistical analysis



**FIGURE 5** The relationship between the average embryo relative telomere length for a given family and its average fry relative telomere length at 186 days postfertilization ( $N = 60$  families). Fry telomere lengths have been plotted as partial residuals as a function of embryo telomere length when all other significant independent variables = 0. The plot shows the relationship from the model that included parental years in FW and SW as independent variables. The plot from the alternative model, which used parental age as an independent variable, can be viewed in the supplementary information (Figure S2). See Methods and Table 1 for statistical analysis



**FIGURE 6** The relationship between paternal years in freshwater and fry relative telomere length. Fathers had spent either 1 ( $N = 6$  families), 2 ( $N = 32$  families) or 3 ( $N = 20$  families) years in freshwater, prior to sea migration. See Methods and Table 1 for statistical analysis

unlikely to be an important difference as the two are usually highly correlated and are used interchangeably as an index of egg size (e.g., Einum & Fleming, 2000; Heinimaa & Heinimaa, 2004). It is also possible that egg quality differed between the two experiments, since although they were based on the same population of wild salmon they were conducted in different years. Maternal diet (e.g., intake of dietary antioxidants) and physiochemical condition of the water (e.g., temperature and pH) are known to affect egg quality (Brooks, Tyler, & Sumpter, 1997); therefore, it is possible that the two cohorts of

mothers experienced different conditions at sea, which influenced the relative quality of their eggs, with potential consequences for offspring telomere length.

There was a positive relationship between telomere length at the embryo and fry stages of development. Families that had relatively longer telomeres at the embryo stage also produced fry with a relatively longer telomere length. One of the main pathways by which telomere length can change is by cell proliferation (i.e., growth). However, because food rations were similar between groups, we saw relatively little variation in body size at the fry stage, compared to what would be observed in the wild. Telomere loss may also be linked to variation in oxidative damage. While rearing conditions in this study were relatively stable (constant temperature, low constant water flow, high food availability) so that oxidative stress might have been relatively low, there may still be heritable differences in antioxidant defences that generate these persistent among-family differences in telomere lengths (e.g., see Kahar, Debes, Vuori, Vähä, & Vasemägi, 2016). Although we have identified a strong relationship between embryo telomere length and fry telomere length, we would expect this relationship to be weaker in the wild, where there is much greater variation in environmental conditions and individual state. This may explain why we found no significant association between embryo telomere length and fry telomere length in wild salmon fry in McLennan et al. (2016).

In contrast to the positive relationship between embryo and fry telomere length, there was a negative relationship between fry weight and fry telomere length. Therefore, for a given embryo telomere length, families that had faster growing (and hence larger) fry had relatively shorter telomeres at the fry stage. This is similar to results obtained by McLennan et al. (2016) and Debes et al. (2016), both of which found that larger wild salmonid fry of a given age had shorter telomeres. Fry may achieve an increase in body size by cell division or cell growth (or both) (Arendt, 2007). While cell growth does not cause any change in telomere length, cell division will lead to some telomere shortening, so an increase in cell proliferation rate (to achieve a larger body size) could have a detrimental effect on telomere length, which may explain this correlation. Although egg size was removed from the fry RTL models during our backward model selection approach, it was identified as a predictor of fry RTL in the AIC approach, most likely because larger salmon eggs generally result in larger offspring at emergence (for reviews, see Fleming, 1996; Jonsson & Jonsson, 2011). Being larger as a fry may have negative consequences for telomere length, but this disadvantage may be offset by the potential benefits to a larger size, such as improved competitiveness.

Fry telomere length was significantly associated with the number of years the father had spent in freshwater. Fathers that had spent a longer period in the river prior to sexual maturation produced fry with shorter telomeres. However, it should be noted that the effect size of paternal years in FW was relatively small, and it was not included in any of the top models during the AIC approach. Therefore, it may be the case that while statistically significant, it was not an overly important predictor of fry telomere length. It is currently

unclear why early paternal years in FW water (and hence early growth rate) might influence offspring telomere length. One possible mechanism could relate to telomerase expression, which is capable of elongating telomeres (Blackburn, 2005). Many species downregulate telomerase expression in postembryonic somatic tissue; however, a number of fish studies have found telomerase to be active in a diversity of somatic cells at various postembryonic life stages (Hartmann et al., 2009; Hatakeyama et al., 2016; Lund, Glass, Tolar, & Blazar, 2009; Yip et al., 2017). Moreover, studies suggest that telomerase activity is positively linked to fish growth rate (Hatakeyama et al., 2016; Peterson, Mok, & Au, 2015; Yap, Yeoh, Brenner, & Venkatesh, 2005). Therefore, it is possible that fathers who have a slower growth rate in early life also experience a down-regulation in telomerase expression, which could affect germline telomere length and therefore the telomere length of their offspring. However, future studies (ideally longitudinal, to avoid the risk of selective disappearance) would be needed to allow a more accurate prediction of the effect of parental growth rates on offspring telomere dynamics (length and loss), and these would benefit from the simultaneous assessment of telomerase activity.

Both this study and that of McLennan et al. (2016) found that fry telomere length was shorter when fathers had spent only 1 year at sea compared with the fathers who had spent longer at sea. There was, however, a difference in the telomere length of fry from the precocial parr fathers. In McLennan et al. (2016) (where paternal years at sea was positively linked to offspring telomere length) precocious parr fathers produced offspring with the shortest telomeres, whereas in the current study offspring from precocious parr fathers had a similar telomere length to offspring from MSW fathers. A possible explanation of this difference is that the relatively high mortality of fry in the wild (90%, compared with only 10% in captivity) which could lead to a nonrandom distribution of surviving fry with respect to telomere length, although further experiments would be needed to examine this possibility.

In this study, we have shown that parental, in particular maternal, life history is associated with offspring telomere length. As we did not experimentally manipulate the parental life histories, it is hard to determine the extent to which these parental effects are due to environmental versus genetic factors, but both are likely to be involved. Nonetheless, it is evident that parents may affect offspring telomere length by indirect genetic and environmental routes.

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## DATA ACCESSIBILITY

Data deposited in the Dryad repository: <https://doi.org/10.5061/dryad.5b6f7>.

## AUTHOR CONTRIBUTIONS

D.M., S.M., J.D.A., P.M. and N.B.M. contributed to experimental design. D.M. and S.M. carried out the I.V.F. manipulations, while D.C.S. analysed fish scales to determine parental life histories. The telomere assay was optimized by D.M. and W.B., and D.M. performed the laboratory analysis. D.M. performed the data analysis, with additional comments from P.M. and N.B.M. This article was written by D.M., with additional comments from W.B., P.M. and N.B.M.

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## REFERENCES

- Arendt, J. (2007). Ecological correlates of body size in relation to cell size and cell number: Patterns in flies, fish, fruits and foliage. *Biological Reviews*, *82*, 241–256. <https://doi.org/10.1111/j.1469-185X.2007.00013.x>
- Asghar, M., Bensch, S., Tarka, M., Hansson, B., & Hasselquist, D. (2015). Maternal and genetic factors determine early life telomere length. *Proceedings of the Royal Society of London B: Biological Sciences*, *282*, 20142263.
- Barson, N. J., Aykanat, T., Hindar, K., Baranski, M., Bolstad, G. H., Fiske, P., ... Karlsson, S. (2015). Sex-dependent dominance at a single locus maintains variation in age at maturity in salmon. *Nature*, *528*, 405–408. <https://doi.org/10.1038/nature16062>
- Bates, D., Maechler, M., Bolker, B., & Walker, S. (2015). Fitting linear mixed-effects models using lme4. *Journal of Statistical Software*, *67*, 1–48.
- Bauch, C., Becker, P. H., & Verhulst, S. (2014). Within the genome, long telomeres are more informative than short telomeres with respect to fitness components in a long-lived seabird. *Molecular Ecology*, *23*, 300–310. <https://doi.org/10.1111/mec.12602>
- Baum, D., Laughton, R., Armstrong, J. D., & Metcalfe, N. B. (2004). Altitudinal variation in the relationship between growth and maturation rate in salmon parr. *Journal of Animal Ecology*, *73*, 253–260. <https://doi.org/10.1111/j.0021-8790.2004.00803.x>
- Blackburn, E. H. (1991). Structure and function of telomeres. *Nature*, *350*, 569–573. <https://doi.org/10.1038/350569a0>
- Blackburn, E. H. (2005). Telomeres and telomerase: Their mechanisms of action and the effects of altering their functions. *FEBS Letters*, *579*, 859–862. <https://doi.org/10.1016/j.febslet.2004.11.036>
- Blount, J. D., Surai, P. F., Houston, D. C., & Møller, A. P. (2002). Patterns of yolk enrichment with dietary carotenoids in gulls: The roles of pigment acquisition and utilization. *Functional Ecology*, *16*, 445–453. <https://doi.org/10.1046/j.1365-2435.2002.00648.x>
- Blount, J. D., Surai, P. F., Nager, R. G., Houston, D. C., Møller, A. P., Trewby, M. L., & Kennedy, M. W. (2002). Carotenoids and egg quality in the lesser black-backed gull *Larus fuscus*: A supplemental feeding study of maternal effects. *Proceedings of the Royal Society of London B*, *269*, 29–36. <https://doi.org/10.1098/rspb.2001.1840>
- Boonekamp, J. J., Bauch, C., Mulder, E., & Verhulst, S. (2017). Does oxidative stress shorten telomeres? *Biology Letters*, *13*, 20170164. <https://doi.org/10.1098/rsbl.2017.0164>
- Boonekamp, J. J., Mulder, G. A., Salomons, H. M., Dijkstra, C., & Verhulst, S. (2014). Nestling telomere shortening, but not telomere length, reflects developmental stress and predicts survival in wild birds. *Proceedings of the Royal Society B: Biological Sciences*, *281*, 20133287. <https://doi.org/10.1098/rspb.2013.3287>
- Bouwhuis, S., Vedder, O., & Becker, P. H. (2015). Sex-specific pathways of parental age effects on offspring lifetime reproductive success in a long-lived seabird. *Evolution*, *69*, 1760–1771. <https://doi.org/10.1111/evo.12692>
- Broer, L., Codd, V., Nyholt, D. R., Deelen, J., Mangino, M., Willemsen, G., ... de Geus, E. J. (2013). Meta-analysis of telomere length in 19 713 subjects reveals high heritability, stronger maternal inheritance and a paternal age effect. *European Journal of Human Genetics*, *21*, 1163–1168. <https://doi.org/10.1038/ejhg.2012.303>
- Brooks, S., Tyler, C. R., & Sumpter, J. P. (1997). Egg quality in fish: What makes a good egg? *Reviews in Fish Biology and Fisheries*, *7*, 387–416. <https://doi.org/10.1023/A:1018400130692>
- Burton, T., Robertsen, G., Stewart, D. C., McKelvey, S., Armstrong, J. D., & Metcalfe, N. B. (2016). Maternal age at maturation underpins contrasting behavior in offspring. *Behavioral Ecology*, *27*, 1280–1287. <https://doi.org/10.1093/beheco/arw073>
- Campisi, J., Kim, S.-H., Lim, C.-S., & Rubio, M. (2001). Cellular senescence, cancer and aging: The telomere connection. *Experimental Gerontology*, *36*, 1619–1637. [https://doi.org/10.1016/S0531-5565\(01\)00160-7](https://doi.org/10.1016/S0531-5565(01)00160-7)
- Chan, S. R. W. L., & Blackburn, E. H. (2004). Telomeres and telomerase. *Philosophical Transactions of the Royal Society B: Biological Sciences*, *359*, 109–121. <https://doi.org/10.1098/rstb.2003.1370>
- Costantini, D. (2010). Complex trade-offs in the pigeon (*Columba livia*): Egg antioxidant capacity and female serum antioxidant status in relation to diet quality. *Journal of Comparative Physiology B*, *180*, 731–739. <https://doi.org/10.1007/s00360-010-0456-z>
- Cram, D. L., Monaghan, P., Gillespie, R., & Clutton-Brock, T. (2017). Effects of early-life competition and maternal nutrition on telomere lengths in wild meerkats. *Proceedings of the Royal Society B: Biological Sciences*, *284*, 20171383. <https://doi.org/10.1098/rspb.2017.1383>
- De Meyer, T., Rietzschel, E. R., De Buyzere, M. L., De Bacquer, D., Van Criekinge, W., De Backer, G. G., ... Bekaert, S. (2007). Paternal age at birth is an important determinant of offspring telomere length. *Human Molecular Genetics*, *16*, 3097–3102. <https://doi.org/10.1093/hmg/ddm271>
- Debes, P. V., Visse, M., Panda, B., Ilmonen, P., & Vasemägi, A. (2016). Is telomere length a molecular marker of past thermal stress in wild fish? *Molecular Ecology*, *25*, 5412–5424. <https://doi.org/10.1111/mec.13856>
- Donelson, J. M., Munday, P. L., & McCormick, M. I. (2009). Parental effects on offspring life histories: When are they important? *Biology Letters*, *5*, 262–265. <https://doi.org/10.1098/rsbl.2008.0642>
- Einum, S., & Fleming, I. A. (2000). Selection against late emergence and small offspring in Atlantic salmon (*Salmo salar*). *Evolution*, *54*, 628–639. <https://doi.org/10.1111/j.0014-3820.2000.tb00064.x>
- Fleming, I. A. (1996). Reproductive strategies of Atlantic salmon: Ecology and evolution. *Reviews in Fish Biology and Fisheries*, *6*, 379–416. <https://doi.org/10.1007/BF00164323>
- Fleming, I. A. (1998). Pattern and variability in the breeding system of Atlantic salmon (*Salmo salar*), with comparisons to other salmonids.

- Canadian Journal of Fisheries and Aquatic Science*, 55(Supplement 1), 59–76. <https://doi.org/10.1139/d98-009>
- Fox, C. W., Bush, M. L., & Wallin, W. G. (2003). Maternal age affects offspring lifespan of the seed beetle, *Callosobruchus maculatus*. *Functional Ecology*, 17, 811–820. <https://doi.org/10.1111/j.1365-2435.2003.00799.x>
- García-Palomares, S., Navarro, S., Pertusa, J. F., Hermenegildo, C., García-Perez, M. A., Rausell, F., ... Tarin, J. J. (2009). Delayed fatherhood in mice decreases reproductive fitness and longevity of offspring. *Biology of Reproduction*, 80, 343–349. <https://doi.org/10.1095/biolreprod.108.073395>
- Gardner, M. L. G. (1976). A review of factors which may influence the sea-age and maturation of Atlantic salmon *Salmo salar* L. *Journal of Fish Biology*, 9, 289–327. <https://doi.org/10.1111/j.1095-8649.1976.tb04680.x>
- Gavrilov, L. A., & Gavrilova, N. S. (1997). Parental age at conception and offspring longevity. *Reviews in Clinical Gerontology*, 7, 5–12. <https://doi.org/10.1017/S0959259897000026>
- Gorbunova, V., & Seluanov, A. (2009). Coevolution of telomerase activity and body mass in mammals: From mice to beavers. *Mechanisms of Ageing and Development*, 130, 3–9. <https://doi.org/10.1016/j.mad.2008.02.008>
- Hartmann, N., Reichwald, K., Lechel, A., Graf, M., Kirschner, J., Dorn, A., ... Englert, C. (2009). Telomeres shorten while Tert expression increases during ageing of the short-lived fish *Nothobranchius furzeri*. *Mechanisms of Ageing and Development*, 130, 290–296. <https://doi.org/10.1016/j.mad.2009.01.003>
- Hatakeyama, H., Yamazaki, H., Nakamura, K.-I., Izumiyama-Shimomura, N., Aida, J., Suzuki, H., ... Ishikawa, N. (2016). Telomere attrition and restoration in the normal teleost *Oryzias latipes* are linked to growth rate and telomerase activity at each life stage. *Aging (Albany NY)*, 8, 62–75. <https://doi.org/10.18632/aging.100873>
- Hausmann, M. F., Longenecker, A. S., Marchetto, N. M., Juliano, S. A., & Bowden, R. M. (2011). Embryonic exposure to corticosterone modifies the juvenile stress response, oxidative stress and telomere length. *Proceedings of the Royal Society of London B: Biological Sciences*, 279, 1447–1456.
- Heidinger, B. J., Blount, J. D., Boner, W., Griffiths, K., Metcalfe, N. B., & Monaghan, P. (2012). Telomere length in early life predicts lifespan. *Proceedings of the National Academy of Sciences of the United States of America*, 109, 1743–1748. <https://doi.org/10.1073/pnas.1113306109>
- Heidinger, B. J., Herborn, K. A., Granroth-Wilding, H., Boner, W., Burthe, S., Newell, M., ... Monaghan, P. (2016). Parental age influences offspring telomere loss. *Functional Ecology*, 30, 1531–1538. <https://doi.org/10.1111/1365-2435.12630>
- Heinimaa, S., & Heinimaa, P. (2004). Effect of the female size on egg quality and fecundity of the wild Atlantic salmon in the sub-arctic River Tenö. *Boreal Environment Research*, 9, 55–62.
- Hellems, J., Mortier, G., De Paepe, A., Speleman, F., & Vandesompele, J. (2007). qBase relative quantification framework and software for management and automated analysis of real-time quantitative PCR data. *Genome Biology*, 8, R19. <https://doi.org/10.1186/gb-2007-8-2-r19>
- Herborn, K. A., Heidinger, B. J., Boner, W., Noguera, J. C., Adam, A., Daunt, F., & Monaghan, P. (2014). Stress exposure in early post-natal life reduces telomere length: An experimental demonstration in a long-lived seabird. *Proceedings of the Royal Society of London B: Biological Sciences*, 281, 20133151. <https://doi.org/10.1098/rspb.2013.3151>
- Ilmonen, P., Kotrschal, A., & Penn, D. J. (2008). Telomere attrition due to infection. *PLoS ONE*, 3, e2143. <https://doi.org/10.1371/journal.pone.0002143>
- Jonsson, B., & Jonsson, N. (2011). *Ecology of Atlantic salmon and Brown trout: Habitat as a template for life histories*, 1st ed. Dordrecht, The Netherlands: Springer. <https://doi.org/10.1007/978-94-007-1189-1>
- Kahar, S., Debes, P. V., Vuori, K. A., Vähä, J.-P., & Vasemägi, A. (2016). Heritability, environmental effects, and genetic and phenotypic correlations of oxidative stress resistance-related enzyme activities during early life stages in Atlantic salmon. *Evolutionary Biology*, 43, 215–226. <https://doi.org/10.1007/s11692-016-9368-5>
- Kamler, E. (1992). *Early life history of fish: An energetics approach*. London, UK: Chapman & Hall. <https://doi.org/10.1007/978-94-011-2324-2>
- Kim, S.-Y., & Velando, A. (2015). Antioxidants safeguard telomeres in bold chicks. *Biology Letters*, 11, 20150211. <https://doi.org/10.1098/rsbl.2015.0211>
- Klemetsen, A., Amundsen, P. A., Dempson, J. B., Jonsson, B., Jonsson, N., O'Connell, M. F., & Mortensen, E. (2003). Atlantic salmon *Salmo salar* L., brown trout *Salmo trutta* L. and Arctic charr *Salvelinus alpinus* (L.): A review of aspects of their life histories. *Ecology of Freshwater Fish*, 12, 1–59. <https://doi.org/10.1034/j.1600-0633.2003.00010.x>
- Kuznetsova, A., Brockhoff, P. B., & Christensen, R. H. B. (2014). lmerTest: Tests for random and fixed effects for linear mixed effect models (lmer objects of lme4 package). R package version 2.0-11.
- Lund, T. C., Glass, T. J., Tolar, J., & Blazar, B. R. (2009). Expression of telomerase and telomere length are unaffected by either age or limb regeneration in *Danio rerio*. *PLoS ONE*, 4, e7688. <https://doi.org/10.1371/journal.pone.0007688>
- Marchetto, N. M., Glynn, R. A., Ferry, M. L., Ostojic, M., Wolff, S. M., Yao, R., & Hausmann, M. F. (2016). Prenatal stress and newborn telomere length. *American Journal of Obstetrics and Gynecology*, 215, 94–95.
- McLennan, D., Armstrong, J. D., Stewart, D. C., McKelvey, S., Boner, W., Monaghan, P., & Metcalfe, N. B. (2016). Interactions between parental traits, environmental harshness and growth rate in determining telomere length in wild juvenile salmon. *Molecular Ecology*, 25, 5425–5438. <https://doi.org/10.1111/mec.13857>
- Metcalfe, N. B., & Thorpe, J. E. (1990). Determinants of geographical variation in the age of seaward-migrating salmon, *Salmo salar*. *Journal of Animal Ecology*, 59, 135–145. <https://doi.org/10.2307/5163>
- Monaghan, P. (2010). Telomeres and life histories. The long and the short of it. *Annals of the New York Academy of Sciences*, 1206, 130–142. <https://doi.org/10.1111/j.1749-6632.2010.05705.x>
- Njajou, O. T., Cawthon, R. M., Damcott, C. M., Wu, S.-H., Ott, S., Garant, M. J., ... Hsueh, W.-C. (2007). Telomere length is paternally inherited and is associated with parental lifespan. *Proceedings of the National Academy of Sciences of the United States of America*, 104, 12135–12139. <https://doi.org/10.1073/pnas.0702703104>
- Noguera, J. C., Metcalfe, N. B., Boner, W., & Monaghan, P. (2015). Sex-dependent effects of nutrition on telomere dynamics in zebra finches (*Taeniopygia guttata*). *Biology Letters*, 11, 20140938. <https://doi.org/10.1098/rsbl.2014.0938>
- Noguera, J. C., Metcalfe, N. B., Reichert, S., & Monaghan, P. (2016). Embryonic and postnatal telomere length decrease with ovulation order within clutches. *Scientific Reports*, 6, 25915. <https://doi.org/10.1038/srep25915>
- Noguera, J. C., Monaghan, P., & Metcalfe, N. B. (2015). Interactive effects of early and later nutritional conditions on the adult antioxidant defence system in zebra finches. *Journal of Experimental Biology*, 218, 2211–2217. <https://doi.org/10.1242/jeb.120956>
- Nordfjäll, K., Svenson, U., Norrback, K.-F., Adolfsson, R., & Roos, G. (2010). Large-scale parent-child comparison confirms a strong paternal influence on telomere length. *European Journal of Human Genetics*, 18, 385–389. <https://doi.org/10.1038/ejhg.2009.178>
- Oikawa, S., & Kawanishi, S. (1999). Site-specific DNA damage at GGG sequence by oxidative stress may accelerate telomere shortening. *FEBS Letters*, 453, 365–368. [https://doi.org/10.1016/S0014-5793\(99\)00748-6](https://doi.org/10.1016/S0014-5793(99)00748-6)
- Økland, F., Jonsson, B., Jensen, A. J., & Hansen, L. P. (1993). Is there a threshold size regulating seaward migration of brown trout and

- Atlantic salmon. *Journal of Fish Biology*, 42, 541–550. <https://doi.org/10.1111/j.1095-8649.1993.tb00358.x>
- Olsson, M., Pauliny, A., Wapstra, E., Uller, T., Schwartz, T., & Blomqvist, D. (2011). Sex differences in sand lizard telomere inheritance: Paternal epigenetic effects increases telomere heritability and offspring survival. *PLoS ONE*, 6, e17473. <https://doi.org/10.1371/journal.pone.0017473>
- Pauliny, A., Devlin, R. H., Johnsson, J. I., & Blomqvist, D. (2015). Rapid growth accelerates telomere attrition in a transgenic fish. *BMC Evolutionary Biology*, 15, 159. <https://doi.org/10.1186/s12862-015-0436-8>
- Perez, J., Moran, P., & Garcia-Vazquez, E. (1999). Cloning and physical mapping of Atlantic salmon (*Salmo salar* L.) telomeric sequences. *Heredity*, 82, 409–414. <https://doi.org/10.1038/sj.hdy.6884980>
- Peterson, D. R., Mok, H. O., & Au, D. W. (2015). Modulation of telomerase activity in fish muscle by biological and environmental factors. *Comparative Biochemistry and Physiology Part C: Toxicology & Pharmacology*, 178, 51–59.
- Reichert, S., Rojas, E. R., Zahn, S., Robin, J. P., Criscuolo, F., & Massemin, S. (2015). Maternal telomere length inheritance in the king penguin. *Heredity*, 114, 10–16. <https://doi.org/10.1038/hdy.2014.60>
- Ringsby, T. H., Jensen, H., Pärn, H., Kvalnes, T., Boner, W., Gillespie, R., ... Monaghan, P. (2015). On being the right size: Increased body size is associated with reduced telomere length under natural conditions. *Proceedings of the Royal Society B: Biological Sciences*, 282, 20152331. <https://doi.org/10.1098/rspb.2015.2331>
- Rossiter, M. (1996). Incidence and consequences of inherited environmental effects. *Annual Review of Ecology and Systematics*, 27, 451–476. <https://doi.org/10.1146/annurev.ecolsys.27.1.451>
- Royle, N. J., Surai, P. F., & Hartley, I. R. (2003). The effect of variation in dietary intake on maternal deposition of antioxidants in zebra finch eggs. *Functional Ecology*, 17, 472–481. <https://doi.org/10.1046/j.1365-2435.2003.00752.x>
- Salmón, P., Nilsson, J. F., Watson, H., Bensch, S., & Isaksson, C. (2017). Selective disappearance of great tits with short telomeres in urban areas. *Proceedings of the Royal Society B: Biological Sciences*, 284, 20171349. <https://doi.org/10.1098/rspb.2017.1349>
- Shearer, W. M. (1992). *Atlantic salmon scale reading guidelines*. Copenhagen, Denmark: International Council for the Exploration of the Sea Copenhagen.
- Tobler, M., & Sandell, M. I. (2009). Sex-specific effects of prenatal testosterone on nestling plasma antioxidant capacity in the zebra finch. *Journal of Experimental Biology*, 212, 89–94. <https://doi.org/10.1242/jeb.020826>
- Trust TAS (2012). *The Atlantic Salmon Trust*. Retrieved from <http://www.atlanticsalmontrust.org/>
- Van Leeuwen, T. E., McLennan, D., McKelvey, S., Stewart, D. C., Adams, C. E., & Metcalfe, N. B. (2015). The association between parental life history and offspring phenotype. *Journal of Experimental Biology*, 219, 374–382.
- Wolf, J. B., & Wade, M. J. (2009). What are maternal effects (and what are they not)? *Philosophical Transactions of the Royal Society B: Biological Sciences*, 364, 1107–1115. <https://doi.org/10.1098/rstb.2008.0238>
- Yap, W. H., Yeoh, E., Brenner, S., & Venkatesh, B. (2005). Cloning and expression of the reverse transcriptase component of pufferfish (*Fugu rubripes*) telomerase. *Gene*, 353, 207–217. <https://doi.org/10.1016/j.gene.2005.04.038>
- Yip, B. W., Mok, H. O., Peterson, D. R., Wan, M. T., Taniguchi, Y., Ge, W., & Au, D. W. (2017). Sex-dependent telomere shortening, telomerase activity and oxidative damage in marine medaka *Oryzias melastigma* during aging. *Marine Pollution Bulletin*, 124, 701–709. <https://doi.org/10.1016/j.marpolbul.2017.1001.1021>

## SUPPORTING INFORMATION

Additional Supporting Information may be found online in the supporting information tab for this article.

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