Received: 10 November 2020 | Accepted: 4 June 2021

DOI: 10.1111/1365-2435.13860

RESEARCH ARTICLE

Functional Ecology | BRITISH ECOLOGICAL SOCIETY

Climate change-induced deprivation of dietary essential fatty acids can reduce growth and mitochondrial efficiency of wild juvenile salmon

Libor Závorka[1,2] | Amelie Crespel[2] | Neal J. Dawson[2] | Magdalene Papatheodoulou[2] | Shaun S. Killen[2] | Martin J. Kainz[1]

[1]WasserCluster Lunz – Inter-University Centre for Aquatic Ecosystem Research, Lunz am See, Austria

[2]Institute of Biodiversity, Animal Health & Comparative Medicine, Graham Kerr Building, College of Medical, Veterinary & Life Sciences, University of Glasgow, Glasgow, UK

Correspondence
Libor Závorka
Email: liborzavorka@email.cz; libor.zavorka@wcl.ac.at

Funding information
L.Z. was funded by the Austrian Science Fund, FWF, Project M2742 BBL.

Handling Editor: Sonya Auer

Abstract

1. Omega-3 long-chain polyunsaturated fatty acids (*n* − 3 LC-PUFA) are essential micronutrients for optimal functioning of cellular metabolism and for somatic growth of all vertebrates including fishes. In addition, *n* − 3 LC-PUFA could also play a key role in response of fishes and other ectothermic vertebrates to changing temperatures.
2. An important, but largely overlooked, consequence of climate change is the reduced availability of dietary *n* − 3 LC-PUFA in aquatic food webs. Changes in availability of dietary *n* − 3 LC-PUFA have recently been proposed as a major driver of novel adaptations and diversification of consumers. Yet, there is only limited knowledge about how *n* − 3 LC-PUFA depletion in aquatic food webs will affect the performance of wild fishes.
3. Here we combine biochemistry and physiology at the cellular level with physiological and cognitive processes at the whole-animal level to test how ecologically relevant deprivation of *n* − 3 LC-PUFA affects performance of wild juvenile Atlantic salmon *Salmo salar*.
4. We found that juvenile salmon had a limited capacity to maintain the fatty acid profile of both muscle and brain under an *n* − 3 LC-PUFA-deficient diet. Despite these findings, brain tissues showed remarkable functional stability in mitochondrial metabolism, and we found no effect of diet on learning ability. However, we found that mitochondrial efficiency in muscles and the somatic growth were reduced under an *n* − 3 LC-PUFA-deficient diet. Importantly, we discovered that the somatic growth of juvenile salmon within both treatments decreased with increasing rate of DHA synthesis and retention.
5. Since DHA is essential for functioning of cellular metabolism, which together with body size are traits closely related to fitness of wild fishes, we suggest that the trade-off between growth rate and accumulation of DHA could play a critical role in resilience of juvenile salmon to the ongoing rapid environmental change.

KEYWORDS
aerobic metabolism, animal behaviour, animal cognition, ATP production, ecosystem functioning, omega-3 fatty acids

1 | INTRODUCTION

An important but poorly understood consequence of climate change is the reduced availability of omega-3 long-chain polyunsaturated fatty acids (*n* − 3 LC-PUFA), eicosapentaenoic acid (EPA, 20:5 *n* − 3) and docosahexaenoic acid (DHA, 22:6 *n* − 3), in aquatic food webs (Fuiman, 2018; Hixson & Arts, 2016; Taipale et al., 2018). This issue is especially pressing in temperate headwater streams vulnerable to rapid decrease of these micronutrients because increasing temperature (Hixson & Arts, 2016) and frequency of extreme weather events, such as torrential rains (Kanno et al., 2014; Woodward et al., 2015). These factors increase the influx of terrestrial matter that is deficient in *n* − 3 LC-PUFA (Brett et al., 2017; Tiwari et al., 2017) and hamper the production of *n* − 3 LC-PUFA by primary producers in the stream (Hixson & Arts, 2016; Taipale et al., 2018). Therefore, climate change will not only affect the thermal environment (Pinsky et al., 2019), but also decrease the availability of dietary *n* − 3 LC-PUFA for consumers in small head water streams, including juvenile Atlantic salmon *Salmo salar*. Understanding the impacts of an *n* − 3 LC-PUFA-deprived diet on the performance and thermal response of stream-dwelling fishes can help us better predict their resilience to changing climate.

It has been shown that dietary *n* − 3 LC-PUFA, especially DHA, influence various ecologically important traits of fishes such as somatic growth (Tocher, 2010) and development of brain and cognitive skills (Pilecky et al., 2021). However, this evidence is largely based on domesticated fishes and laboratory models, whose genotype and phenotype substantially differ from their wild counterparts (Albert et al., 2012; Johnsson et al., 2014). A handful of studies on fish of wild origin is available from marine environments (e.g. Gourtay et al., 2020), where access to dietary *n* − 3 LC-PUFA is rarely a limiting factor for consumers (Colombo et al., 2017). The tissue content of *n* − 3 LC-PUFA determines numerous cellular processes in ectotherms, including functioning of mitochondrial metabolism (Martin et al., 2013; Salin et al., 2021), which limits the performance of organs and the whole organism (Dawson, Alza, et al., 2020; Salin et al., 2015), including their thermal performance (Salin et al., 2015). Therefore, the link between the diet quality, fatty acid composition of the tissue and mitochondrial metabolism provides a promising, but rarely explored venue for a more mechanistic understanding of the diet quality impact on performance of cells and whole-animal and potential feedbacks to the entire ecosystems under changing climate.

Aquatic prey of juvenile fishes is rich in EPA and alpha-linoleic acid (ALA; 18:3 *n* − 3) that are produced by periphyton at the base of the stream food web, but terrestrial resources contain only ALA and are almost deprived of EPA (Twining et al., 2019). The conversion of EPA to DHA has been shown to be more effective and contributes 10 times more to the building of neuronal structures than the less efficient conversion of ALA to DHA (Pilecky et al., 2021). Juvenile salmon and other small stream fishes have only limited direct access to dietary DHA, but they can occasionally acquire it, for example, by consumption of eggs (Näslund et al., 2015). The elevated metabolic costs (i.e. the additional energy spend) for internal synthesis of DHA from ALA, for example, during periods of floods characterized by low availability of *n* − 3 LC-PUFA micronutrients (Argerich et al., 2016; Arts et al., 2009), may lead to a reduction of somatic growth (Tocher, 2010). Yet, under scarcity of dietary *n* − 3 LC-PUFA this costly physiological adaptation can be important to maintain proper functioning and growth of brain (Ishizaki et al., 2005; Lund et al., 2014). Here, we posit that dietary *n* − 3 LC-PUFA deprivation caused by climate change as a result of reduced primary production and elevated flux of terrestrial subsidies should force fish into a trade-off between maintenance of *n* − 3 LC-PUFA tissue content and somatic growth. Reduced content of *n* − 3 LC-PUFA in brain and muscle should then have negative consequences for their thermal tolerance, mitochondrial efficiency, aerobic metabolism, and cognition.

To test our predictions, we exposed juvenile salmon (offspring of wild parents) to two experimental diets, one with a content of optimal *n* − 3 LC-PUFA conducive for their proper development (hereafter control diet) and the other with reduced *n* − 3 LC-PUFA content simulating the decreased availability of these micronutrients in degraded aquatic food web (hereafter *n* − 3 LC-PUFA-deprived diet). The treatment lasted 8 weeks which is a time period comparable to pulses of *n* − 3 LC-PUFA poor terrestrial resources at headwater streams during seasonal floods (Argerich et al., 2016). Our experimental design allowed us to test whether an *n* − 3 LC-PUFA-deprived diet: (a) leads to a reduction of *n* − 3 LC-PUFA and particularly DHA in brain and muscle tissues; (b) decreases efficiency of mitochondrial metabolism and increases maximum metabolic rate (MMR) particularly at elevated temperature; (c) reduces brain size and deteriorates learning ability particularly at elevated temperature and (d) has a negative effect on somatic growth of wild fish.

2 | MATERIALS AND METHODS

2.1 | Experimental fish origin and rearing

Experimental fish were offspring of wild parents (14 families originating from unique crossing of 14 females and 14 males) collected during the spawning migration between November and December 2018 in a permanent trap at Loch na Croic on the river Black Water, which is situated within the catchment of River Conon in Northern

Scotland, UK. Atlantic salmon and resident brown trout *Salmo trutta* are the dominant fish species in the system (Auer et al., 2018). Fertilized eggs developed until hatching at the Contin hatchery, which is supplied by water from the Black Water River. Juvenile salmon were transported post-hatching on 9 April 2019 to the aquarium facility at the University of Glasgow. From the onset of external feeding until the start of the experiment individuals were kept in flow-through stream system supplied with UV-treated, recirculating freshwater kept at 13°C. They were fed daily until apparent satiation with a mixture of frozen bloodworms and commercial pellet food with high content of fish oil (EWOS).

On 19 November 2019 (i.e. 7 months post-hatching), 80 individuals of similar size were anaesthetized in a benzocaine solution and measured for fork length (FL) to the nearest millimetre and body mass (bM) to the nearest 0.01 g (mean ± *SD*, FL = 55 ± 5 mm, bM = 1.92 ± 0.61 g). Fish were then tagged with Visible Implant Elastomer (VIE; Northwest Marine Technology Inc.) for individual identification and distributed among ten rearing tanks (32 L, 40 × 40 × 30 cm, 8 individuals per tank). Tanks were evenly designated to the two feeding treatments with five replicated rearing tanks and 40 individuals in each treatment. One tank from each diet treatment (16 individuals in total) was used to test design of learning test and these individuals were removed from the study. During the experiment salmon were fed on isonitrogenous and isocaloric fish pellet feeds (GARANT^{TM}, Austria) formulated to provide the same amount of energy, lipid and protein sufficient to meet somatic requirements for salmonids (Murray et al., 2014; Tocher, 2015) but differing in their *n* − 3 LC-PUFA concentration (ESM 1). We evaluated total fatty acid profiles and mitochondrial metabolism in salmon muscle and brain, as well as maximum metabolic rate of individuals (MMR), their somatic growth rate, and learning ability. All traits were measured on all individuals available with exception for measurements of mitochondrial metabolism, which were based on a subset of nine individuals randomly selected from each treatment group (Table 1). We measured MMR, learning ability and mitochondrial metabolism at low (13°C) and elevated (18°C) temperatures to evaluate the response of these traits to acute warming. These temperatures are near below and above the physiological optimum reported for Atlantic salmon (Elliott & Hurley, 1997). Fish were exposed to a progressive increase of temperature over the course of 24 hr and then remained at 18°C1 for another 48 hr until the test. Following the test, the temperature of the rearing tank was progressively cooled over 24 hr back to 13°C. Three-day exposure to temperature increase by 6°C is ecologically relevant as it can occur in salmon streams and Atlantic salmon are able to physiologically cope with the temperature change of a similar intensity (Gallant et al., 2017).

2.2 | Maximum metabolic rate and somatic growth rate

Maximum metabolic rate of individuals was estimated from the rate of oxygen uptake after exhaustive exercise using intermittent flow respirometry (Killen et al., 2021). The metabolic assays were conducted between 6 and 15 January 2020, after 7 weeks of exposure to the dietary treatments. MMR of each fish was measured twice, once at the acclimation temperature, that is, 13°C and once at elevated temperature, that is, 18°C. Assays took place in 16 glass chambers (0.10204 L) in recirculation loops using a peristaltic pump (also used to achieve good water mixing in the chambers) and gas-tight PVC tubing (volume of the loop between 8 to 15 ml) submerged into a thermoregulated water bath (92 L, 80 × 40 × 29 cm). Fish from two rearing tanks were thus measured simultaneously. Bacterial oxygen consumption was kept at a minimum by using a UV filter sterilizer and it was evaluated daily before and after the fish were placed in the respirometry chambers. One chamber was also kept empty during the measurements to estimate the evolution of the bacterial consumption over time. Measured oxygen uptake was then adjusted by a linear increase in bacterial metabolism over the time of the assay. The whole system was also fully bleached between each trial. Flush pumps, connected to a timer, flushed oxygenated water through the chambers for 2 min with 8-min intervals between flushes, during which the oxygen uptake of individuals was measured. Oxygen concentration within each glass chamber was recorded every 2 s by a fibre optic sensor inserted into probe holders inside the loop and using a four-channel FireSting O2system (PyroScience GmbH). The level of oxygen within the chambers never dropped under 60% air saturation. Oxygen probes were calibrated on a daily basis. Prior to

TABLE 1 Overview of chronology of the measurements and sample size of the experimental fish

Process	Date range	Sample size *n* − 3 [PUFA-deprived/control diet]
Tagging and start of dietary treatment	09/11/2019	32/32
MMR	06/01/2020–15/01/2020	31/30
Learning test—training	22/11/2019–11/01/2020	32/32
Learning test—scoring	26/12/2019–13/01/2020	32/31
Mitochondrial metabolism	26/01/2020–06/02/2020	9/9
Fatty acids profile analysis—brain	14/01/2020–06/02/2020	18/15
Fatty acids profile analysis—muscle	14/01/2020–06/02/2020	28/24
SGR	09/11/2019–06/02/2020	28/24

measurement, individuals were fasted for 48 hr. MMR was measured as the oxygen uptake immediately after an exhaustive exercise protocol where fish were individually chased until exhaustion (i.e. no response to taping of the tail fin) for 3 min in a circular tank containing aerated freshwater (Killen et al., 2021). MMR was calculated using rolling regression slopes of 2 min every 2 s, after a wait period of 20 s. The slope was multiplied by the volume of the respirometry chamber and tubing after deduction of the fish volume (assuming a fish density of 1 g/ml) was used to calculate the maximum oxygen uptake (mg O_2/hr). After respirometry, all individuals were measured for FL to the nearest millimetre and bM to the nearest 0.01 g. Three individuals died before the respirometry (1 and 2 from the control and *n* − 3 LC-PUFA-deprived diet treatment respectively). Due to a technical issue with the respirometry setup, data from 16 individuals (eight from each diet treatment group) were recorded at 13℃, but not at 18℃.

Specific growth rate of individuals was calculated following equation from (Brett & Groves, 1979) using initial and final FL as measured at tagging and final tissue sampling respectively, and time interval between the two measurements in days.

2.3 | Learning tests

Focal fish were trained within their rearing tank to associate a visual stimulus with a food reward, which was then used to determine the success probability of approaching the rewarded stimulus in a maze test (see ESM 4 for details of fish training and experimental setup). Fish were scored individually in three 10-min consecutive trials with 20 min acclimation period in between the trials during each scoring day. In the first two trials in each scoring day, the salmon were presented with two stimuli—red circle (i.e. rewarded stimulus) and blue circle (i.e. unrewarded stimulus). To test the effect of egocentric spatial learning (i.e. left-right decision), the salmon were presented with two blue circles in the third trial (Rodriguez et al., 1994). In the third trial, individuals were rewarded only if they approached the stimulus that was on the side where the red circle had been presented in the previous two trials. Potential lateralization effects were tested on the first scoring day with the reward stimulus being presented haphazardly on the left or the right side in horizontal position on the water surface, that is, like the training technique used in the rearnig tank (ESM 4). The main learning test was then conducted on scoring days 2 to 4 (i.e. the total number of trials was 9) with the stimulus presented in the vertical position encircling an entrance to a maze chamber (ESM 4) and the rewarded stimulus being always on the right side. Individuals were not fed for 24 hr prior the learning tests to standardize the hunger level and to increase the motivation to approach the reward stimulus. Water temperature during the learning tests was 13℃. To test the effect of acute warming on learning performance half of the individual from each dietary treatment was tested at 18℃ in the second scoring day and the other half in the third scoring day. Records from all nine learning trials were available in 48 individuals, while due to mortality and technical failure for 15 individuals (i.e. nine control and eight *n* − 3 PUFA-deprived diet) were available only six records from 2 scoring days and one individual (i.e. control diet) was not tested at all.

2.4 | Fatty acids profile analysis

Samples of brain and white muscle tissue were collected for fatty acid analysis between 14 January and 5 February 2020 immediately after individuals were killed with an overdose of benzocaine. The time span of the final sampling was due to the fact that subset of individuals was used for the measurements of mitochondrial metabolism (see below). Tissue samples were snap frozen by submerging them in liquid nitrogen, freeze-dried and then stored at—80℃ until further analysis. The final bM and FL of all individuals were measured before the tissue samples collection. Brains were dissected and brain mass was measured to the nearest 0.001 g. The sample of white muscle tissue separated from the skin and bone tissue was collected from the area above the lateral line under the dorsal fin. Due to the final mortality of 12 individuals (eight and four raised on the control and *n* − 3 LC-PUFA-deprived diet respectively) the final sample size was lower than the initial number. In addition, brain from a subsample of 19 individuals (9 and 10 raised on the control and *n* − 3 LC-PUFA-deprived diet respectively) was fixed in 4% buffered (pH 6.9) paraformaldehyde solution for immunocyto-chemical analysis (unpublished data), and thus could not be used for evaluation of fatty acids profile. The final subsample was thus an end result of mortality and random subsampling and can be considered representative of the larger population of fish in the study.

The lipid content of freeze-dried samples was quantified following the method described in Böhm et al. (2014), see (ESM 2) for details. All fatty acids were measured and reported as relative contents of total fatty acid methyl esters. Concentrations of 45 fatty acids were calculated using calibration curves based on known standard concentrations (see ESM 2 for the full list). Total fatty acids correspond to the sum of their polar and neutral phase; however, it should be noted that majority of functionally important fatty acids in the brain is from polar lipids (Ebm et al., 2021) which also applies to DHA in the muscle tissues (ESM 2). We calculated descriptive statistics for the main groups of fatty acids (Table 2), but for further analysis we used only the 15 most dominant fatty acids with the average relative concentration higher than 1%. We also calculated bioaccumulation factors (Kainz et al., 2006) of ALA, EPA and DHA as:

$$\text{Bioacummulation factor} = \frac{n-3\ \text{PUFA}\,[\%]\ \text{in tissue of an individual}}{n-3\ \text{PUFA}\,[\%]\ \text{in diet}}.$$

The bioaccumulation factor is an indicator of the internal synthesis or retention of *n* − 3 LC-PUFA by the consumers. Specifically, the retention or internal synthesis of *n* − 3 LC-PUFA increases with increasing value of the bioaccumulation factor and it is positive if the factor is >1.

TABLE 2 Summary of final models reported in Results. Models for muscle and brain tissue had identical structure. 15FAs –% of 15 dominant fatty acids, diet—dietary treatment, bM—body mass at the time of measurement, bM_i—initial body mass, *T*–temperature, F_b DHA—bioaccumulation factor of DHA, position—position of the rewarded stimulus, trial—trial number, socRank—social rank of individual within the rearing tank, resBrain—residual brain mass, ID—individual identification

			Explanatory variables	
Model group	**Model type**	**Response variables**	**Fixed factor**	**Random factor**
Fatty acid	MANOVA	15 FAs	diet + bM	—
	LM	SFA or MUFA or *n* − 3 PUFA or *n* − 6 PUFA or *n* − 3:*n* − 6 or DHA	diet + bM	—
	LM	F_b ALA or EPA or DHA	diet + bM	—
Mitochondrial metabolism	LM	RCR or LEAK	*T* + diet + bM	
MMR	LMM	MMR	*T* + diet + bM	1|ID
Growth	LM	SGR FL	F_b DHA (brain or muscle) + diet + bM_i	—
Learning	GLMM	Success rate on day 1 (i.e. lateralization)	position + diet + trial + soc. Rank + resBrain + bM	1|ID
	GLMM	Success rate on day 2–4	diet + trial + *T* + socRank + resBrain + bM	1|ID
	LM	resBrain	diet	—

2.5 | Mitochondrial metabolism

Mitochondrial metabolism from the white muscle and brain of juvenile salmon was measured in 2 ml of respiration solution using a high-resolution respirometer (Oxygraph-2k with O2k-Fluorescence module; Oroboros Instruments, Innsbruck, Austria) at 13 and 18°C under continuous stirring. Samples were prepared as described in Salin et al. (2016) using the shredded tissue technique. White muscle tissue was dissected, removing skin and bone tissue, from an area ~1 cm above the lateral line under the dorsal fin. Brain was bisected and one half of the brain was used for mitochondrial analysis and other for analysis of fatty acids. Muscle (40 mg wet mass) or brain tissue (10 mg wet mass) were added to the chamber and allowed to sit for 5 min. Respiration rate was measured as the rate of decline in O_2 concentration. Mitochondrial respiration protocols to determine LEAK (Ln) and respiratory control ratio (RCR) were adapted from Dawson, Millet, et al. (2020), for details see ESM 3.

2.6 | Statistical analysis

All analyses were conducted in R v.3.6.2 (http://www.R-project.org/). See summary of the final models reported in result section in (Table 2). We used following categorical variables dietary treatment (i.e. two levels: control or *n* − 3 PUFA-deprived diet), temperature (i.e. two levels: 13 and 18°C), social rank of individual within the rearing tank (i.e. two levels: dominant or subdominant), trial (i.e. three levels), position of the rewarded stimulus (i.e. two levels: left or right). Success probability in the learning test was evaluated by GLMM for data with binomial distribution. Residual brain mass was calculated as a residuals from linear regression model between log transformed brain mass and log transformed final fork length of the fish (Kotrschal et al., 2013). Repeatability of MMR (adjusted for temperature, diet treatment, and body mass) and success in the learning test (adjusted for the trial and diet treatment) was quantified using the intra-class correlation coefficient extracted from linear mixed models with individual identity as a random factor (Nakagawa & Schielzeth, 2010). We used simple linear models and did not evaluate repeatability across the measurements of mitochondrial metabolism, because of the low sample size which prevented us to calculate sufficiently robust estimate of inter-individual variance (Dingemanse & Dochtermann, 2013). Non-significant interactions were removed from the models. Significance of the final models was evaluated using ANOVA tables using Type II sums of squares. Variables were log-transformed when needed to approach the normal distribution of residuals. Differences among groups were analysed using Tukey's HSD post-hoc test.

3 | RESULTS

Dietary treatment influenced the concentration of fatty acids in muscle (MANOVA $F_{15,35} = 68.33$, $p < 0.001$, Figure 1a, Table 3) and brain (MANOVA $F_{15,16} = 11.44$, $p < 0.001$, Figure 1a, Table 3) tissue of juvenile salmon. We also found an effect of body mass on fatty acid profile of the tissues, but this covariable was significant only in the model for muscle (MANOVA $F_{1,49} = 3.61$, $p = 0.001$) and not the brain (MANOVA $F_{15,16} = 1.96$, $p = 0.100$) tissue. Dietary treatment had no effect on amount of total lipids in fish tissues, but it has significantly affected concentration of SFA, MUFA, *n* − 3 PUFA, *n* − 6 PUFA and *n* − 3:*n* − 6 ratio in muscle and brain of juvenile salmon (Table 3). We found that fish did not bioaccumulate ALA and EPA as concentration of these fatty acids was lower in the tissues of the fish then in their diet. In contrast, fish in both

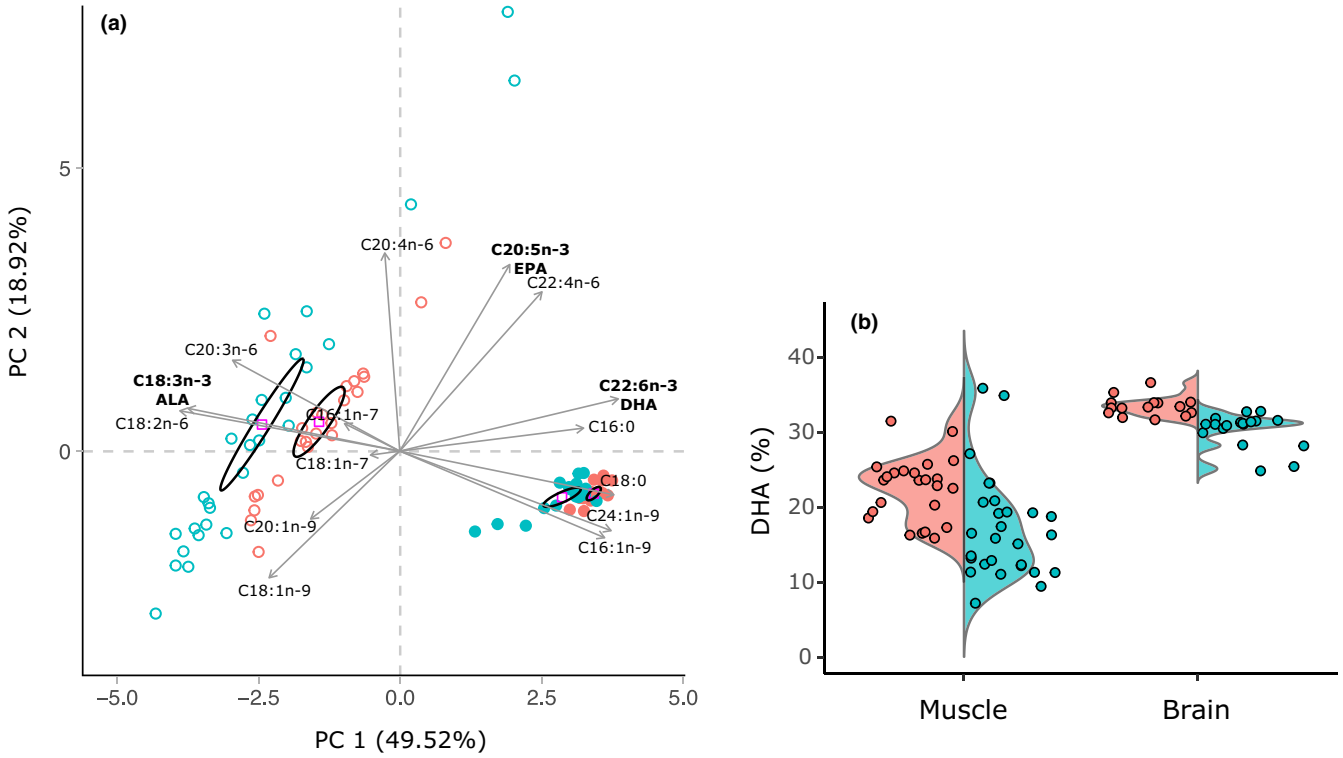


FIGURE 1 (a) Overall fatty acid profile and (b) DHA content (%) in muscle (empty circles) and brain tissues (filled circles). Black ellipses in PCA indicate 95% CI around the mean (empty purple square) of each group. Red and green symbols indicate the control and *n* − 3 LC-PUFA-deprived dietary treatment respectively

treatments bioaccumulated large amount of DHA and the bioaccumulation factor for both muscle and brain were significantly higher in salmon fed *n* − 3 LC-PUFA-deprived diet then in the control group (Table 3). Despite the higher bioaccumulation was the relative concentration of DHA significantly lower in both muscle ($F_{1,49}$ = 17.67, p < 0.001, Figure 1b) and brain ($F_{1,49}$ = 34.67, p < 0.001, Figure 1b) tissue of *n* − 3 PUFA-deprived diet group then in the control group.

Respiratory control ratio Ln of muscle mitochondrial was lower in salmon fed *n* − 3 LC-PUFA-deprived diet then in the control group ($F_{32,1}$ = 7.04, p = 0.012, Figure 2a), but it was not affected by temperature ($F_{32,1}$ = 2.74, p = 0.108) or body mass of individuals ($F_{32,1}$ = 0.67, p = 0.419). Mitochondrial LEAK Ln in muscle was higher in salmon fed *n* − 3 LC-PUFA-deprived diet then in the control group ($F_{32,1}$ = 4.98, p = 0.033, Figure 2c), but it was not affected by temperature ($F_{32,1}$ = 0.16, p = 0.694) or body mass ($F_{32,1}$ = 0.51, p = 0.479). In contrast to muscles, we found no significant effect of diet treatment on RCR Ln ($F_{32,1}$ = 0.91, p = 0.348; Figure 2b), and LEAK Ln ($F_{32,1}$ = 0.210, p = 0.660; Figure 2d) in brain. Brain mitochondrial responded strongly to the temperature as RCR Ln was higher ($F_{32,1}$ = 6.45, p = 0.016) and LEAK Ln was lower ($F_{32,1}$ = 17.37, p < 0.001) at 13°C then at 18°C. Salmon body mass did not affect RCR Ln ($F_{32,1}$ = 0.10, p = 0.759) and LEAK Ln ($F_{32,1}$ = 0.96, p = 0.333) of brain mitochondria. We have conducted all analyses of the mitochondrial activity also using Lomy method (for comparison of Ln and Lomy methods see ESM 3).

There was no effect of diet treatment on residual MMR, that is, after correcting for body mass (χ^2 = 0.02, p = 0.903), but residual MMR increased in both treatments with increasing temperature (χ^2 = 6.00, p = 0.014; Figure 3). Inter-individual differences in residual MMR were repeatable across the two measurements (R_{adj} = 0.464, 95% CI [0.223; 0.694]).

Juvenile salmon fed on *n* − 3 LC-PUFA-deprived diet grew significantly less than the salmon from the control group ($F_{48,1}$ = 6.94, p = 0.011; Figure 4). The growth rate of individuals across both dietary treatments decreased with increasing bioaccumulation factor of DHA in muscle ($F_{48,1}$ = 23.74, p < 0.001; Figure 4a) and brain ($F_{29,1}$ = 4.28, p = 0.048; Figure 4b) tissue.

Success rate in the main learning test (i.e. testing day 2–4) was not affected by diet (χ^2 = 2.20, p = 0.138; Figure 5a), temperature (χ^2 = 1.60, p = 0.205), social status of individual (χ^2 = 0.18, p = 0.670) or their body mass (χ^2 = 0.13, p = 0.715). However, success probability increased with increasing relative brain mass of individuals (χ^2 = 15.68, p < 0.0001; Figure 5b) and it was affected by the trial number (χ^2 = 6.69, p = 0.035; Figure 5a) as individuals, regardless of dietary treatment, were more successful in the second than in the third trial (post-hoc test comparison of 2nd and 3rd trial: p = 0.041), but the probability of success in the first trial did not differ from the second and third one. Individual differences in success probability were highly repeatable across the trials (R_{adj} = 0.729, 95% CI [0.449, 0.966]). Residual brain mass did not differ between the two dietary treatments ($F_{1,49}$ = 1.599, p = 0.2121; Figure 5c). Success rate on

TABLE 3 Differences in the lipid composition of muscle and brain tissue of the juvenile salmon and their experimental diet in *n* − 3 LC-PUFA-deprived (−*n* − 3) and control (+*n* − 3) treatment, F_b—bioaccumulation factor. Descriptive statistics report mean ± *SD* and the *p*-value of statistical differences is based on models listed in Table 2. Significant differences (false discovery rate method adjusted $p < 0.05$) are indicated by asterisk

	Muscle			Brain			Diet	
	−*n* − 3	+*n* − 3	diff. *p*	−*n* − 3	+*n* − 3	diff. *p*	−*n* − 3	+*n* − 3
Total lipids content [mg/g]	53.55 ± 21.62	54.98 ± 22.29	0.793	349.53 ± 41.28	334.70 ± 27.62	0.246	263.21 ± 10.12	250.31 ± 10.14
SFA [%]	20.87 ± 2.73	23.35 ± 1.46	0.001*	28.44 ± 0.94	29.39 ± 0.81	0.001*	13.54 ± 1.07	20.87 ± 0.12
MUFA [%]	34.76 ± 10.89	29.71 ± 6.23	0.008*	31.67 ± 2.29	29.10 ± 1.95	0.001*	59.82 ± 2.49	44.88 ± 0.27
n − 3 PUFA [%]	23.27 ± 7.42	29.51 ± 4.62	0.001*	33.43 ± 2.14	36.74 ± 1.21	0.001*	7.55 ± 1.71	16.58 ± 0.07
n − 6 PUFA [%]	20.97 ± 2.77	17.23 ± 2.09	0.001*	6.40 ± 0.91	4.73 ± 0.35	0.001*	18.96 ± 1.86	17.47 ± 0.25
n − 3:*n* − 6	1.11 ± 0.34	1.72 ± 0.32	0.001*	5.34 ± 0.92	7.81 ± 0.58	0.001*	0.39 ± 0.06	0.95 ± 0.02
F_b ALA	0.51 ± 0.14	0.60 ± 0.10	0.047	0.10 ± 0.02	0.10 ± 0.01	0.743	–	–
F_b EPA	0.49 ± 0.26	0.64 ± 0.14	0.001*	0.56 ± 0.05	0.62 ± 0.04	0.001*	–	–
F_b DHA	19.24 ± 7.77	4.53 ± 0.85	0.001*	34.07 ± 2.50	6.79 ± 0.26	0.001*	–	–

the first testing day was not affected by the left or right position of rewarded stimulus, that is, lateralization did not affect success rate in the learning test ($\chi^2 = 1.97$, $p = 0.160$).

4 | DISCUSSION

Our results show that dietary deprivation of *n* − 3 LC-PUFA induced by experimental treatment simulating subsidies from a degraded aquatic food web caused, in comparison to a control diet, a change in the fatty acid composition and, importantly, a decrease of DHA content in the muscle and brain of juvenile salmon. DHA is a key micronutrient for optimal functioning of cellular membranes and vital tissues of vertebrates (Pilecky et al., 2021). The reduced tissue content of DHA coincided with reduced mitochondrial efficiency of ATP production in muscle, but it did not affect maximum metabolic rate. Intriguingly, the functioning of mitochondrial in brain and learning ability of the fish were unaffected, despite the substantial decrease of DHA content in the brain. Our findings indicate that juvenile salmon have a capacity to mitigate at least some potentially negative effects of temporarily reduced dietary intake of *n* − 3 LC-PUFA on functioning of brain. However, the DHA bioaccumulation factor in the salmon tissue was negatively correlated with the growth rate of individuals, indicating that individual adaptation to dietary deprivation of *n* − 3 LC-PUFA comes at an energetic cost related to internal synthesis or retention of DHA, which translates to reduced somatic growth.

The DHA content in the experimental diets was likely much lower than that individuals would need for proper long-term functioning of their tissues, but this is also typical for the aquatic and terrestrial prey of stream fishes (Twining et al., 2019). The bioaccumulation factor of DHA in both treatments was much higher than one and about five times higher in salmon fed n-3 LC-PUFA-deprived diet than in the control group. In contrast, differences in bioaccumulation factor of EPA and ALA between the diet treatments were not significant and for both molecules had value lower than one. This suggests that dietary EPA and ALA were not retained in the salmon tissues, but instead likely used for the synthesis of DHA by fish in both dietary treatments (Kainz et al., 2006). Besides internal synthesis of DHA (Murray et al., 2014; Taipale et al., 2018) individuals also could attain high DHA via dietary retention before the experiment and through the maternal provisioning of eggs from which they hatched (Fuiman, 2018). Despite a higher bioaccumulation factor, the DHA content was lower in both muscle and brain tissues *n* − 3 of the salmon fed on the *n* − 3 LC-PUFA-deprived diet. Feeding on *n* − 3 LC-PUFA-deprived diet was associated with slower body growth, and body growth in both dietary treatments decreased with an increasing DHA bioaccumulation factor. This finding corresponds to previous studies on domesticated fishes (Lazzarotto et al., 2015; Murray et al., 2014; Taipale et al., 2018) and demonstrates that maintaining high DHA contents in tissues despite limited dietary supply is costly also for wild fish. Our results thus suggest that the physiological adaptation to mitigate dietary shortage of *n* − 3 LC-PUFA

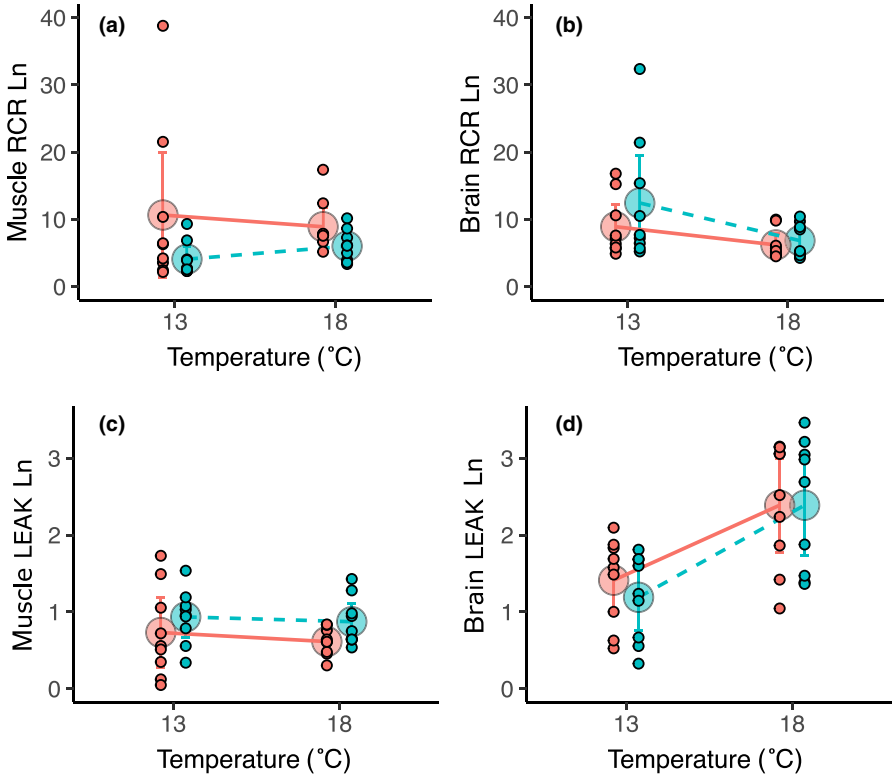


FIGURE 2 Respiratory control ratio RCR in muscle (a) and brain (b), and mitochondrial LEAK in muscle (c) and brain (d) brain

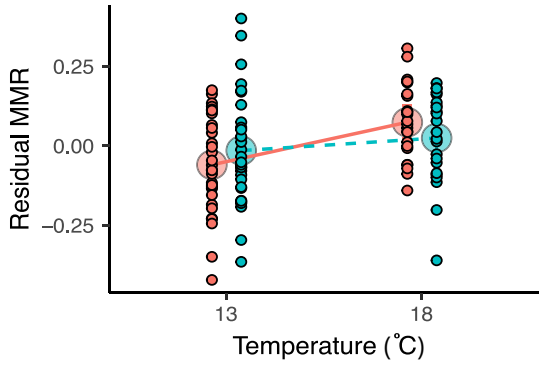


FIGURE 3 Residual maximum metabolic rate (MMR) (i.e. measured as residuals from linear regression between MMR and body mass of individuals at the time of respirometry). Large circles and whiskers indicate mean ± 95% CI of each group (green: *n* − 3 LC-PUFA-deprived diet, red: control diet)

can be limited even in consumers such as juvenile salmonids, which often prey on resources with relatively low *n* − 3 LC-PUFA, for example, terrestrial invertebrates (Evangelista et al., 2014; Syrjänen et al., 2011).

Omega-3 LC-PUFA-deprived diet lowered mitochondrial efficiency of ATP production in muscle tissue as indicated by lower RCR, which was likely caused by the increment of mitochondrial membrane LEAK. However, the effect of diet on mitochondrial in muscle did not translate to differences in MMR or its response to acute warming. This was unexpected because muscle tissue represents the majority of fish biomass, and thus muscle oxygen consumption should relate to overall oxygen uptake of the fish, especially during or after strenuous physical activity (Norin & Malte, 2012). However, the association of oxygen uptake by muscle mitochondrial and whole organism is also driven by other factors such as mitochondrial production of harmful reactive oxygen species (Salin et al., 2015). Previous studies provide equivocal results about the importance of dietary *n* − 3 LC-PUFA for mitochondrial functioning. For example, mitochondrial LEAK in muscle of domesticated trout have been reported to either increase (Martin et al., 2013), but also decrease (Guderley et al., 2008) under *n* − 3 PUFA-deprived diet. While the mechanism behind different resilience of the mitochondrial metabolism remains to be explored, it could explain why we observed no effect of *n* − 3 LC-PUFA deprivation on mitochondrial metabolism in the brain, despite significant decrease of DHA in its cellular membranes.

The functional resilience of the brain to *n* − 3 LC-PUFA-deprived diet was also apparent at the level of cognition, as we found no effect of dietary treatment on learning. This is in contrast with some previous studies, which showed for example that *n* − 3 LC-PUFA-deprived diet reduced responsiveness to visual stimulus in southern flounder *Paralichthys lethostigma* (Oberg & Fuiman, 2015) or deteriorate antipredator behaviour in pike perch *Sander lucioperka* (Lund et al., 2014). The discrepancy could be caused by relatively short dietary deprivation of *n* − 3 LC-PUFA in our study. Nonetheless, other dimensions of behaviour that we did not examine may still be affected and so more study is warranted given the obvious differences in tissue concentrations. Individual differences in success probability in the learning test were highly repeatable across the nine trials and

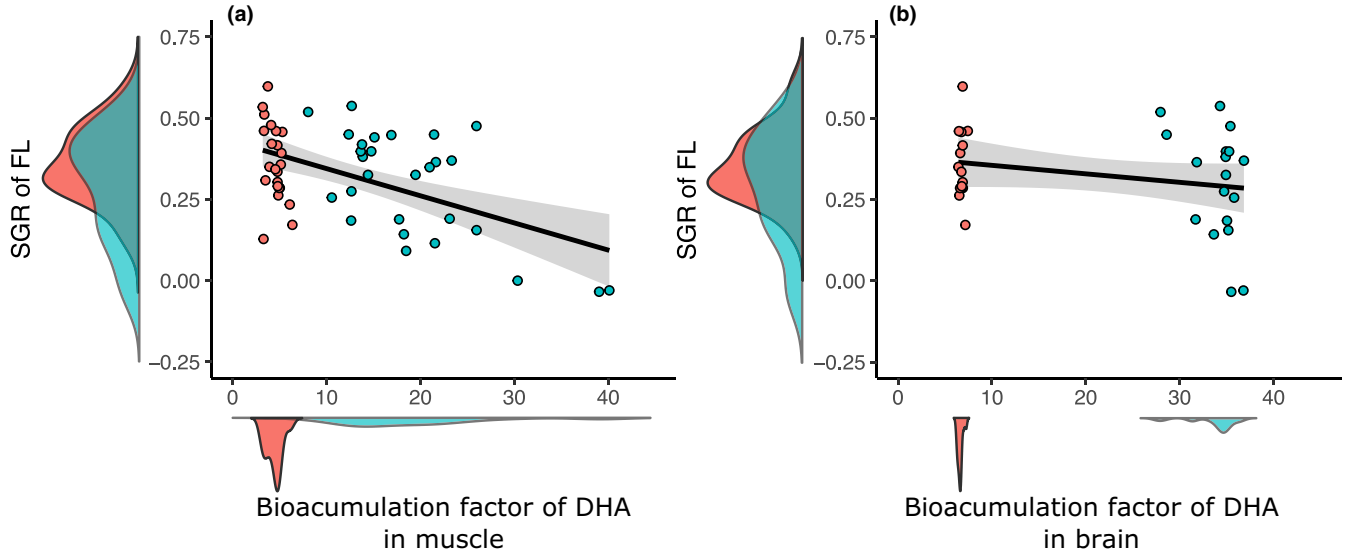
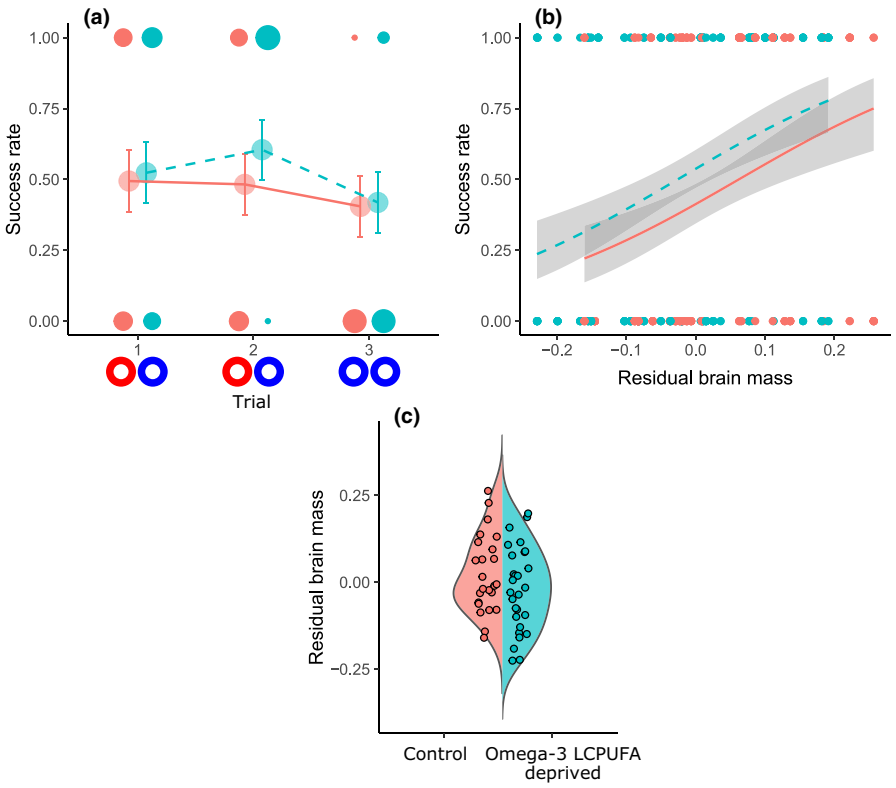


FIGURE 4 The relationship between specific growth rate of fork length SGR FL and bioaccumulation factor of DHA in (a) muscle and (b) brain tissues. Density plots along the axis correspond to the distribution of corresponding variables. Red and green symbols indicate the control and *n* − 3 LC-PUFA-deprived diet respectively

FIGURE 5 (a) Success probability in the learning test, blue and the red circles bellow the plot indicate the visual stimuli in each trial. Large circles and whiskers indicate mean ± 95% CI of each group. Size of the circles at the level of 0 and 1 values of the *y* axis corresponds to the proportion of unsuccessful and successful trials; (b) the relationship between the residual brain size and success probability in the learning test and; (c) effect of dietary treatment on the residual brain mass. Red and green symbols indicate the control and *n* − 3 LC-PUFA-deprived diet respectively



positively associated to the residual brain mass. Intra-specific variation in brain size have been shown to be positively related to neuronal abundance (Marhounová et al., 2019) and learning skills in fishes (Kotrschal et al., 2013). While the brain mass did not differ between the treatment groups, the link between brain size and success rate indicates that the learning test was cognitively challenging and the lack of the effect of dietary treatments is evidence that the experimental diet had no influence on learning ability in this study.

In this unique laboratory experiment, we linked biochemistry and physiology at the cellular level to physiological and cognitive processes at the individual level to provide one of the first evidences that short-term dietary deprivation of *n* − 3 LC-PUFA in freshwater ecosystems can lead to reduction of DHA content, mitochondrial efficiency and body growth of wild fishes. Anadromous fish, such as Atlantic salmon, may mitigate the negative effects of dietary *n* − 3 LC-PUFA deprivation in their nursery stream by accelerating

smoltification (Bell et al., 1997) and migration to the *n* − 3 LC-PUFA rich marine feeding grounds (Colombo et al., 2017). However, the freshwater juvenile life stage is a major selection bottleneck (Elliott, 1994) and diet quality induces a strong selection pressure on the physiological phenotype of juvenile salmonids (Auer et al., 2018). It is thus possible that the long-term reduction of dietary *n* − 3 LC-PUFA availability in aquatic ecosystems caused by homeoviscous adaptation of primary producers to increasing water temperature, for example, diatoms in periphyton on stream bottom (Hixson & Arts, 2016; Taipale et al., 2018) will eventually cause a broader range of negative impacts on physiology, behaviour, and fitness than what we observed in this short (i.e. 8 weeks) feeding experiment. It has been shown that ~16℃ is a breakpoint for decrease of *n* − 3 LC-PUFA primary production in aquatic ecosystems (Hixson & Arts, 2016). This means that increasing temperature in temperate regions—where Atlantic salmon and other salmonid fishes are distributed—will reduce primary production of *n* − 3 LC-PUFA mainly from spring to autumn but in lower latitudes also during the winter (IPCC, 2013). Our study was conducted in winter and temperatures used (i.e. 13 and 18℃) correspond to the winter conditions at the southern edge of the species distribution (Jonsson & Jonsson, 2011; O'Briain, 2019), but salmon populations from higher latitudes will likely experience these temperatures mainly from spring to autumn. Prey consumption and growth rate of Atlantic salmon is not strongly affected by the local adaptation to the thermal conditions in the stream of origin and both peak at temperatures between 16 and 21℃ (Jonsson et al., 2001). Our results suggest that juvenile salmon will, at these temperatures, likely experience lower mitochondrial efficiency and slower growth rate caused by reduced availability of dietary *n* − 3 LC-PUFA. Mitochondrial efficiency (Salin et al., 2015) and growth rate (Morgan & Metcalfe, 2001) are suggested to be related to fitness of wild fishes, thus our findings highlight the need for further studies on ecological significance of dietary essential fatty acids for populations of wild fishes.

ACKNOWLEDGEMENTS

Authors are grateful to Neil Evans, Magnus Lovén Wallerius, Samuel-Karl Kämmer, and Katharina Winter for their assistance with collection and processing of samples.

AUTHORS' CONTRIBUTIONS

L.Z. conceived, designed and coordinated the study, carried out the behavioural measurements, tissue samples collection and analysis, statistical analyses, and drafted the manuscript; A.C. carried out the measurements of respirometry and mitochondrial metabolism and collected tissue samples; N.J.D. carried out the measurements of mitochondrial metabolism; M.P. collected the experimental fish and participated on their husbandry; S.S.K. conceived the study, participated to the study design and metabolic measurements; M.J.K. conceived the study, assisted with tissue analysis; All authors critically revised the manuscript and gave final approval for publication and agree to be held accountable for the work performed therein.

DATA AVAILABILITY STATEMENT

Data are archived at figshare.com https://doi.org/10.6084/m9.figshare.13061684 and in Supporting Information (Závorka et al., 2021).

ORCID

Libor Závorka https://orcid.org/0000-0002-0489-3681
Amelie Crespel https://orcid.org/0000-0002-6351-9008
Neal J. Dawson https://orcid.org/0000-0001-5389-8692
Magdalene Papatheodoulou https://orcid.org/0000-0003-1759-1858
Shaun S. Killen https://orcid.org/0000-0003-4949-3988
Martin J. Kainz https://orcid.org/0000-0002-2388-1504

REFERENCES

Albert, F. W., Somel, M., Carneiro, M., Aximu-Petri, A., Halbwax, M., Thalmann, O., Blanco-Aguiar, J. A., Plyusnina, I. Z., Trut, L., Villafuerte, R., Ferrand, N., Kaiser, S., Jensen, P., & Pääbo, S. (2012). A comparison of brain gene expression levels in domesticated and wild animals. *PLoS Genetic*, *8*, e1002962. https://doi.org/10.1371/journal.pgen.1002962

Argerich, A., Haggerty, R., Johnson, S. L., Wondzell, S. M., Dosch, N., Corson-Rikert, H., Ashkenas, L. R., Pennington, R., & Thomas, C. K. (2016). Comprehensive multiyear carbon budget of a temperate headwater stream. *Journal of Geophysical Research: Biogeosciences*, *121*, 1306–1315. https://doi.org/10.1002/2015JG003050

Arts, M. T., Brett, M. T., & Kainz, J. M. (Eds). (2009). *Lipids in Aquatic ecosystems* (p. 377). Springer-Verlag. https://doi.org/10.1007/978-0-387-89366-2

Auer, S. K., Anderson, G. J., McKelvey, S., Bassar, R. D., McLennan, D., Armstrong, J. D., Nislow, K. H., Downie, H. K., McKelvey, L., Morgan, T. A. J., Salin, K., Orrell, D. L., Gauthey, A., Reid, T. C., & Metcalfe, N. B. (2018). Nutrients from salmon parents alter selection pressures on their offspring. *Ecology Letters*, *21*, 287–295. https://doi.org/10.1111/ele.12894

Bell, J. G., Tocher, D. R., Farndale, B. M., Cox, D. I., McKinney, R. W., & Sargent, J. R. (1997). The effect of dietary lipid on polyunsaturated fatty acid metabolism in Atlantic salmon (*Salmo salar*) undergoing parr-smolt transformation. *Lipids*, *32*, 515–525. https://doi.org/10.1007/s11745-997-0066-4

Böhm, M., Schultz, S., Koussoroplis, A.-M., & Kainz, M. J. (2014). Tissue-specific fatty acids response to different diets in common carp (*Cyprinus carpio* L.). *PLoS ONE*, *9*, e94759. https://doi.org/10.1371/journal.pone.0094759

Brett, J. R., & Groves, T. D. D. (1979). Physiological energetics. *Fish Physiology*, *8*, 280–352.

Brett, M. T., Bunn, S. E., Chandra, S., Galloway, A. W. E., Guo, F., Kainz, M. J., Kankaala, P., Lau, D. C. P., Moulton, T. P., Power, M. E., Rasmussen, J. B., Taipale, S. J., Thorp, J. H., & Wehr, J. D. (2017). How important are terrestrial organic carbon inputs for secondary production in freshwater ecosystems? *Freshwater Biology*, *62*, 833–853. https://doi.org/10.1111/fwb.12909

Colombo, S. M., Wacker, A., Parrish, C. C., Kainz, M. J., & Arts, M. T. (2017). A fundamental dichotomy in long-chain polyunsaturated fatty acid abundance between and within marine and terrestrial ecosystems. *Environmental Reviews*, *25*, 163–174. https://doi.org/10.1139/er-2016-0062

Dawson, N. J., Alza, L., Nandal, G., Scott, G. R., & McCracken, K. G. (2020). Convergent changes in muscle metabolism depend on duration of high-altitude ancestry across Andean waterfowl. *e-Life*, *9*, e56259. https://doi.org/10.7554/eLife.56259

Dawson, N. J., Millet, C., Selman, C., & Metcalfe, N. B. (2020). Measurement of mitochondrial respiration in permeabilized fish gills. *Journal of Experimental Biology*, *223*, jeb216762. https://doi.org/10.1242/jeb.216762

Dingemanse, N. J., & Dochtermann, N. A. (2013). Quantifying individual variation in behaviour: Mixed-effect modelling approaches. *Journal of Animal Ecology*, *82*, 39–54. https://doi.org/10.1111/1365-2656.12013

Ebm, N., Guo, F., Brett, M. T., Bunn, S. E., & Kainz, M. J. (2021). Polyunsaturated fatty acids in fish tissues more closely resemble algal than terrestrial diet sources. *Hydrobiologia*, *848*(2), 371–383. https://doi.org/10.1007/s10750-020-04445-1

Elliott, J. M. (1994). *Quantitative ecology and the brown trout*. Oxford University Press.

Elliott, J. M., & Hurley, M. A. (1997). A functional model for maximum growth of Atlantic salmon parr, *Salmo salar*, from two populations in northwest England. *Functional Ecology*, *11*(5), 592–603. https://doi.org/10.1046/j.1365-2435.1997.00130.x

Evangelista, C., Boiche, A., Lecerf, A., & Cucherousset, J. (2014). Ecological opportunities and intraspecific competition alter trophic niche specialization in an opportunistic stream predator. *Journal of Animal Ecology*, *83*, 1025–1034. https://doi.org/10.1111/1365-2656.12208

Fuiman, L. A. (2018). Egg boon fatty acids reveal effects of a climatic event on a marine food web. *Ecological Monographs*, *88*, 585–599. https://doi.org/10.1002/ecm.1324

Gallant, M. J., LeBlanc, S., MacCormack, T. J., & Currie, S. (2017). Physiological responses to a short-term, environmentally realistic, acute heat stress in Atlantic salmon, *Salmo Salar. Facets*, *2*(1), 330–341. https://doi.org/10.1139/facets-2016-0053

Gourtay, C., Chabot, D., Audet, C., Madec, L., Huelvan, C., Ducros, L., Claireaux, G., Mazurais, D., & Zambonino-Infante, J. L. (2020). Effect of thermal and nutritional conditions on fatty acid metabolism and oxidative stress response in juvenile European sea bass (*Dicentrarchus labrax*). *Marine Biology*, *167*, 144. https://doi.org/10.1007/s00227-020-03729-3

Guderley, H., Kraffe, E., Bureau, W., & Bureau, D. P. (2008). Dietary fatty acid composition changes mitochondrial phospholipids and oxidative capacities in rainbow trout red muscle. *Journal of Comparative Physiology B*, *178*, 385–399. https://doi.org/10.1007/s00360-007-0231-y

Hixson, S. M., & Arts, M. T. (2016). Climate warming is predicted to reduce n-3, long-chain, polyunsaturated fatty acid production in phytoplankton. *Global Change Biology*, *22*, 2744–2755. https://doi.org/10.1111/gcb.13295

IPCC. (2013). Climate change 2013 the physical science basis: Working Group I contribution to the fifth assessment report of the Intergovernmental Panel on Climate Change. In D. Jacob, A. R. Ravishankara, & K. Shine (Eds.), (p. 139.). Cambridge University Press.

Ishizaki, Y., Masuda, R., Uematsu, K., Shimizu, K., Arimoto, M., & Takeuchi, T. (2005). The effect of dietary docosahexaenoic acid on schooling behavior and brain development in larval yellowtail. *Journal of Fish Biology*, *58*, 1691–1703. https://doi.org/10.1111/j.1095-8649.2001.tb02323.x

Johnsson, J. I., Brockmark, S., & Näslund, J. (2014). Environmental effects on behavioural development consequences for fitness of captive-reared fishes in the wild. *Journal of Fish Biology*, *85*, 1946–1971. https://doi.org/10.1111/jfb.12547

Jonsson, B., Forseth, T., Jensen, A. J., & Næsje, T. F. (2001). Thermal performance of juvenile Atlantic Salmon, *Salmo salar* L. *Functional Ecology*, *15*(6), 701–711. https://doi.org/10.1046/j.0269-8463.2001.00572.x

Jonsson, B., & Jonsson, N. (2011). *Ecology of Atlantic salmon and brown trout: Habitat as a template for life histories* (Vol. 33). Springer Science & Business Media.

Kainz, M., Telmer, K., & Mazumder, A. (2006). Bioaccumulation patterns of methyl mercury and essential fatty acids in lacustrine planktonic food webs and fish. *Science of the Total Environment*, *368*(1), 271–282. https://doi.org/10.1016/j.scitotenv.2005.09.035

Kanno, Y., Vokoun, J. C., & Letcher, B. H. (2014). Paired stream–air temperature measurements reveal fine-scale thermal heterogeneity within headwater brook trout stream networks. *River Research Applications*, *30*, 745–755. https://doi.org/10.1002/rra.2677

Killen, S. S., Christensen, E., Cortese, D., Závorka, L., Cotgrove, L., Crespel, A., Munson, A., Nati, J. J. H., Norin, T., Papatheodoulou, M., & McKenzie, D. J. (2021). Guidelines for the reporting of methods for estimating metabolic rates using aquatic intermittent-closed respirometry. *EcoEvoRxiv*. https://doi.org/10.32942/osf.io/gnzh7

Kotrschal, A., Rogell, B., Bundsen, A., Svensson, B., Zajitschek, S., Brännström, I., Immler, S., Maklakov, A. A., & Kolm, N. (2013). The benefit of evolving a larger brain: Big-brained guppies perform better in a cognitive task. *Animal Behaviour*, *86*, e4–e6. https://doi.org/10.1016/j.anbehav.2013.07.011

Lazzarotto, V., Corraze, G., Leprevost, A., Quillet, E., Dupont-Nivet, M., & Médale, F. (2015). Three-year breeding cycle of rainbow trout (*Oncorhynchus mykiss*) fed a plant-based diet, totally free of marine resources: Consequences for reproduction, fatty acid composition and progeny survival. *PLoS ONE*, *10*, e0117609. https://doi.org/10.1371/journal.pone.0117609

Lund, I., Höglund, E., Ebbesson, L. O., & Skov, P. V. (2014). Dietary LC-PUFA deficiency early in ontogeny induces behavioural changes in pike perch (*Sander lucioperca*) larvae and fry. *Aquaculture*, *432*, 453–461. https://doi.org/10.1016/j.aquaculture.2014.05.039

Marhounová, L., Kotrschal, A., Kverková, K., Kolm, N., & Němec, P. (2019). Artificial selection on brain size leads to matching changes in overall number of neurons. *Evolution*, *73*, 2003–2012. https://doi.org/10.1111/evo.13805

Martin, N., Bureau, D. P., Marty, Y., Kraffe, E., & Guderley, H. (2013). Dietary lipid quality and mitochondrial membrane composition in trout: Responses of membrane enzymes and oxidative capacities. *Journal of Comparative Physiology B*, *183*, 393–408. https://doi.org/10.1007/s00360-012-0712-5

Morgan, I. J., & Metcalfe, N. B. (2001). Deferred costs of compensatory growth after autumnal food shortage in juvenile salmon. *Proceedings of Royal Society B: Biological Science*, *268*, 295–301. https://doi.org/10.1098/rspb.2000.1365

Murray, D. S., Hager, H., Tocher, D. R., & Kainz, M. J. (2014). Effect of partial replacement of dietary fish meal and oil by pumpkin kernel cake and rapeseed oil on fatty acid composition and metabolism in Arctic charr (*Salvelinus alpinus*). *Aquaculture*, *431*, 85–91. https://doi.org/10.1016/j.aquaculture.2014.03.039

Nakagawa, S., & Schielzeth, H. (2010). Repeatability for Gaussian and non-Gaussian data: A practical guide for biologists. *Biological Reviews*, *85*, 935–956. https://doi.org/10.1111/j.1469-185X.2010.00141.x

Näslund, J., Aldvén, D., & Závorka, L. (2015). Eggs from anadromous adults provide marine-derived nutrients to Atlantic salmon and brown trout parr in late autumn–observations from a Swedish coastal stream. *Environmental Biology of Fishes*, *98*(12), 2305–2313. https://doi.org/10.1007/s10641-015-0436-y

Norin, T., & Malte, H. (2012). Intraspecific variation in aerobic metabolic rate of fish: Relations with organ size and enzyme activity in brown trout. *Physiological and Biochemical Zoology*, *85*, 645–656. https://doi.org/10.1086/665982

Oberg, E. W., & Fuiman, L. A. (2015). Linking fatty acids in the diet and tissues to quality of larval southern flounder (*Paralichthys lethostigma*). *Journal of Experimental Marine Biology and Ecology*, *467*, 7–15. https://doi.org/10.1016/j.jembe.2015.02.021

O'Briain, R. (2019). Climate change and European rivers: An eco-hydromorphological perspective. *Ecohydrology*, *12*(5), e2099. https://doi.org/10.1002/eco.2099

Pilecky, M., Závorka, L., Arts, M. T., & Kainz, M. J. (2021). Omega-3 PUFA profoundly affect neural, physiological, and behavioural

competences – Implications for systemic changes in trophic interactions. *Biological Reviews*. https://doi.org/10.1111/brv.12747

Pinsky, M. L., Eikeset, A. M., McCauley, D. J., Payne, J. L., & Sunday, J. M. (2019). Greater vulnerability to warming of marine versus terrestrial ectotherms. *Nature*, *569*, 108–111. https://doi.org/10.1038/s41586-019-1132-4

Rodriguez, F., Duran, E., Vargas, J. P., Torres, B., & Salas, C. (1994). Performance of goldfish trained in allocentric and egocentric maze procedures suggests the presence of a cognitive mapping system in fishes. *Animal Learning and Behaviour*, *22*, 409–420. https://doi.org/10.3758/BF03209160

Salin, K., Auer, S. K., Anderson, G. J., Selman, C., & Metcalfe, N. B. (2016). Inadequate food intake at high temperatures is related to depressed mitochondrial respiratory capacity. *Journal of Experimental Biology*, *219*, 1356–1362. https://doi.org/10.1242/jeb.133025

Salin, K., Auer, S. K., Rey, B., Selman, C., & Metcalfe, N. B. (2015). Variation in the link between oxygen consumption and ATP production, and its relevance for animal performance. *Proceedings of Royal Society B: Biological Science*, *282*, 20151028. https://doi.org/10.1098/rspb.2015.1028

Salin, K., Mathieu-Resuge, M., Graziano, N., Dubillot, E., Le Grand, F., Soudant, P., & Vagner, M. (2021). The relationship between membrane fatty acid content and mitochondrial efficiency differs within-and between-omega-3 dietary treatments. *Marine Environmental Research*, *163*, 105205. https://doi.org/10.1016/j.marenvres.2020.105205

Syrjänen, J., Korsu, K., Louhi, P., Paavola, R., & Muotka, T. (2011). Stream salmonids as opportunistic foragers: The importance of terrestrial invertebrates along a stream-size gradient. *Canadian Journal of Fisheries and Aquatic Sciences*, *68*(12), 2146–2156. https://doi.org/10.1139/f2011-118

Taipale, S. J., Kahilainen, K. K., Holtgrieve, G. W., & Peltomaa, E. T. (2018). Simulated eutrophication and browning alters zooplankton nutritional quality and determines juvenile fish growth and survival. *Ecology and Evolution*, *8*, 2671–2687. https://doi.org/10.1002/ece3.3832

Tiwari, T., Buffam, I., Sponseller, R. A., & Laudon, H. (2017). Inferring scale-dependent processes influencing stream water biogeochemistry from headwater to sea. *Limnology and Oceanography*, *62*, S58–S70. https://doi.org/10.1002/lno.10738

Tocher, D. R. (2010). Fatty acid requirements in ontogeny of marine and freshwater fish. *Aquatic Research*, *41*, 717–732. https://doi.org/10.1111/j.1365-2109.2008.02150.x

Tocher, D. R. (2015). Omega-3 long-chain polyunsaturated fatty acids and aquaculture in perspective. *Aquaculture*, *449*, 94–107. https://doi.org/10.1016/j.aquaculture.2015.01.010

Twining, C. W., Brenna, J. T., Lawrence, P., Winkler, D. W., Flecker, A. S., & Hairston Jr., N. G. (2019). Aquatic and terrestrial resources are not nutritionally reciprocal for consumers. *Functional Ecology*, *33*(10), 2042–2052. https://doi.org/10.1111/1365-2435.13401

Woodward, G., Bonada, N., Feeley, H. B., & Giller, P. S. (2015). Resilience of a stream community to extreme climatic events and long-term recovery from a catastrophic flood. *Freshwater Biology*, *60*, 2497–2510. https://doi.org/10.1111/fwb.12592

Závorka, L., Crespel, A., Dawson, J. N., Killen, S. S., & Kainz, M. J. (2021). Data from: Climate change induced deprivation of dietary essential fatty acids can reduce growth and mitochondrial efficiency of wild juvenile salmon. *Figshare*, https://doi.org/10.6084/m9.figshare.13061684.v2

SUPPORTING INFORMATION

Additional supporting information may be found online in the Supporting Information section.

How to cite this article: Závorka, L., Crespel, A., Dawson, N. J., Papatheodoulou, M., Killen, S. S., & Kainz, M. J. (2021). Climate change-induced deprivation of dietary essential fatty acids can reduce growth and mitochondrial efficiency of wild juvenile salmon. *Functional Ecology*, 35, 1960–1971. https://doi.org/10.1111/1365-2435.13860