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LETTERS

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Adaptive potential of a Pacific salmon challenged by climate change

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Pacific salmon provide critical sustenance for millions of people worldwide and have far-reaching impacts on the productivity of ecosystems. Rising temperatures now threaten the persistence of these important fishes[1,2], yet it remains unknown whether populations can adapt. Here, we provide the first evidence that a Pacific salmon has both physiological and genetic capacities to increase its thermal tolerance in response to rising temperatures. In juvenile chinook salmon (*Oncorhynchus tshawytscha*), a 4 °C increase in developmental temperature was associated with a 2 °C increase in key measures of the thermal performance of cardiac function[3,4]. Moreover, additive genetic effects significantly influenced several measures of cardiac capacity, indicative of heritable variation on which selection can act. However, a lack of both plasticity and genetic variation was found for the arrhythmic temperature of the heart, constraining this upper thermal limit to a maximum of 24.5 ± 2.2 °C. Linking this constraint on thermal tolerance with present-day river temperatures and projected warming scenarios[5], we predict a 17% chance of catastrophic loss in the population by 2100 based on the average warming projection, with this chance increasing to 98% in the maximum warming scenario. Climate change mitigation is thus necessary to ensure the future viability of Pacific salmon populations.

Rapid increases in temperature due to anthropogenic climate change are projected to have extensive impacts on natural systems due to the profound effects of temperature on organisms[6]. Indeed, species are adapted to their thermal environment at scales both local and global, such that breadths of thermal tolerance and thermal optima for performance often correspond to the thermal conditions in which species evolved[7]. The capacities for adaptation and acclimation are key determinants of how populations can cope with climate change, as they allow phenotypes to 'track' a changing environment[8–10]. In fishes, most phenotypic responses to climate change that have been documented are attributable to phenotypic plasticity[9], although genetic responses within populations are also expected to occur due to changes in selection pressures[10]. The degree to which both these processes can act on functional traits that set thermal tolerance will greatly influence the ability of populations to inhabit more extreme environments.

In many aquatic ectotherms such as fish, the limits of thermal tolerance are thought to be set by a mismatch between oxygen supply and demand[11]. Aerobic scope—the difference between an organism's minimum and maximum oxygen consumption rate—peaks at an optimum temperature (T_{opt}) and subsequently declines with further warming owing to capacity limitations of the cardiorespiratory system[12]. The limits of thermal tolerance are reached when insufficient scope is available for key aerobic activities such as swimming, growth or reproduction. Although key for active species such as the Pacific salmon (*Oncorhynchus* spp.)[1], this loss of aerobic scope may be less important for some species, such as benthic ambush predators that have minimal aerobic demands[13,14]. Yet, increased mortality in both Pacific salmon[1] and benthic eelpout (*Zoarces viviparus*)[11] populations has been linked with a loss of aerobic scope during anomalously high temperatures. Such loss of aerobic scope is largely driven by limitations on maximum heart rate (f_{Hmax}), as increased heart rate is the primary mechanism that supports increased tissue oxygen demand at higher temperature[15]. Indeed, differences in the thermal performance of cardiac function explain patterns of biogeography in marine intertidal invertebrates[16], reef fish[17] and pelagic predatory fish[18], whereas the correspondence between T_{opt} and local thermal conditions among Pacific salmon populations corresponds with differences in cardiac capacity[19]. Although these differences suggest a potential for thermal tolerance to track environmental temperatures, the potential for both adaptation and acclimation of cardiac capacity remains largely unknown. Studies of the heritability and plasticity of oxygen-limited thermal tolerance are thus needed to understand how populations might cope with rapid climate change.

High river temperatures have recently been linked with increased mortality of juvenile chinook salmon (*O. tshawytscha*)[20], raising concerns over the future viability of this ecologically and economically important species[2]. To assess the extent to which chinook salmon can adapt or acclimate to rising temperatures, we mated wild-caught adults in full-factorial crosses[21] and reared offspring from each family in present-day (+0 °C) and projected future (+4 °C) temperature conditions. We then measured the response of f_{Hmax} to warming[3] in juvenile offspring from each family and temperature treatment. We found that the Arrhenius break temperature of f_{Hmax} (T_{AB}), which corresponds to T_{opt} (refs 3,4), averaged 14.0 ± 1.1 °C in the +0 °C group and 16.1 ± 0.9 °C in the +4 °C group (Fig. 1 and Supplementary Fig. 1). The peak f_{Hmax} (f_{Hpeak}) averaged 153 ± 18 beats min^{-1} in the +0 °C group and 180 ± 17 beats min^{-1} in the +4 °C group. The average temperature at which f_{Hpeak} was reached (T_{peakfH}) was 20.8 ± 2.3 °C and 22.8 ± 1.9 °C in the +0 °C and +4 °C groups, respectively, whereas the arrhythmic temperature of f_{Hmax} (T_{arr}), which signifies the onset of cardiac failure and which corresponds well with the upper thermal limit for aerobic performance[3,4], was similar in the two groups at 24.2 ± 1.6 °C and 24.5 ± 2.2 °C, respectively. Thus, the average T_{AB} and T_{peakfH} increased ~2 °C after developmental acclimation to future temperatures, whereas T_{arr} increased only 0.3 °C. Indeed, T_{arr} was the only trait that did not significantly differ between treatment groups (Table 1), similar to previous findings that warm acclimation provides little

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Table 1 | The plastic and genetic effects contributing to cardiac performance and thermal tolerance in Quinsam River chinook salmon (*O. tshawytscha*).

	DF	SS	*F*	*P*	σ^2	% phenotypic var	
f_{Hrest}							
Treatment	1	43,629	753	**<0.001**	156	Plastic	69
Dam	4	313	1.35	0.252	1.12		
Sire	4	467	2.02	0.092	1.67		
Sire × Dam	4	441	1.90	0.110	1.57		
Treatment × Dam	4	165	0.71	0.583	0.59		
Treatment × Sire	4	284	1.23	0.300	1.01		
Residual	260	15,055			53.8		
f_{Hpeak}							
Treatment	1	30,636	119	**<0.001**	109	Plastic	22
Dam	4	938	0.91	0.457	3.35		
Sire	4	4,458	4.34	**0.002**	15.9	Additive	13
Sire × Dam	4	1,786	1.74	0.142	6.38		
Treatment × Dam	4	847	0.82	0.511	3.02		
Treatment × Sire	4	647	0.63	0.642	2.31		
Residual	260	6,6813			239		
f_{Hscope}							
Treatment	1	1,145	4.52	**0.034**	4.09	Plastic	1
Dam	4	1,732	1.71	0.148	6.19		
Sire	4	4,907	4.84	**0.001**	17.5	Additive	23
Sire × Dam	4	883	0.87	0.482	3.15		
Treatment × Dam	4	1,321	1.30	0.269	4.72		
Treatment × Sire	4	1,343	1.32	0.261	4.79		
Residual	260	6,5885			235		
T_{AB}							
Treatment	1	235	267	**<0.001**	0.84	Plastic	42
Dam	4	3.04	0.87	0.485	0.01		
Sire	4	12.3	3.49	**0.009**	0.04	Additive	9
Sire × Dam	4	3.99	1.14	0.340	0.01		
Treatment × Dam	4	7.26	2.07	0.086	0.03		
Treatment × Sire	4	8.19	2.33	0.057	0.03		
Residual	260	228			0.82		
T_{peakfH}							
Treatment	1	101.6	26.9	**<0.001**	0.36	Plastic	7
Dam	4	30.0	1.98	0.098	0.11		
Sire	4	23.4	1.54	0.191	0.08		
Sire × Dam	4	9.37	0.62	0.650	0.03		
Treatment × Dam	4	7.24	0.48	0.752	0.03		
Treatment × Sire	4	5.83	0.38	0.819	0.02		
Residual	260	985			3.52		
T_{arr}							
Treatment	1	2.30	0.68	0.411	0.01		
Dam	4	35.2	2.59	**0.037**	0.13	Maternal	4
Sire	4	24.1	1.77	0.134	0.09		
Sire × Dam	4	6.70	0.49	0.740	0.02		
Treatment × Dam	4	1.47	0.11	0.980	0.01		
Treatment × Sire	4	9.66	0.71	0.585	0.03		
Residual	260	882			3.15		

The results of the analysis of variance are summarized for resting heart rate (f_{Hrest}), peak f_H (f_{Hpeak}), scope for f_H (f_{Hscope}), Arrhenius break temperature (T_{AB}), temperature at which f_{Hpeak} occurs (T_{peakfH}) and arrhythmic temperature (T_{arr}). Shown are the degrees of freedom (DF), sum of squares (SS), *F* statistic, *P* value, variance component (σ^2) and the percentage of total phenotypic variance (% phenotypic var) explained by plastic, maternal or additive genetic effects. Significant values ($P < 0.05$) are given in bold.

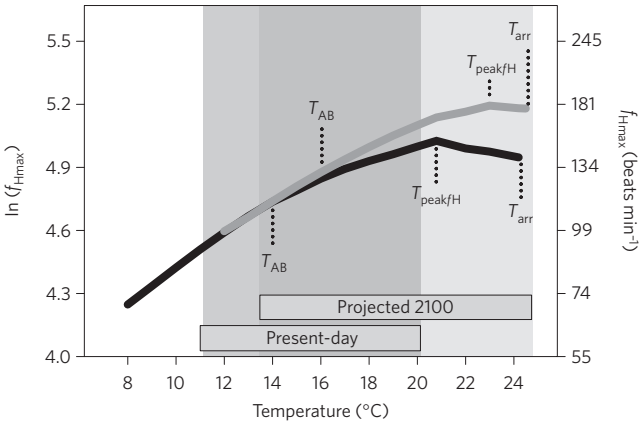

Figure 1 | Mean increase in maximum heart rate (f_{Hmax}) among all offspring from the +0 °C (black line) and +4 °C (grey line) treatment groups of Quinsam River chinook salmon (*O. tshawytscha*). Offspring were reared in two temperature treatments, reflecting current and future conditions, and the response of their f_{Hmax} to warming was measured from their acclimation temperature. Shown for each treatment group are the Arrhenius break temperature of f_{Hmax} (T_{AB}), the temperature at which the peak f_{Hmax} occurred (T_{peakfH}) and the temperature at which f_{Hmax} became arrhythmic (T_{arr}). Also shown are the present-day and projected 2100 stream temperatures during the juvenile residency of this population, from the mean spring temperature to the maximum spring temperature (present-day temperatures collected from 2000 to 2011 by the Department of Fisheries and Oceans Canada). Shading indicates the present-day and projected temperature ranges, with the darker shade indicating the overlapping temperatures.

benefit to upper temperature tolerance in Pacific salmon[22]. Still, our results indicate that chinook salmon can plastically increase the maximum capacity of their hearts (f_{Hpeak}) as well as T_{AB} and T_{peakfH}. This result differs from wild Atlantic salmon, which can adjust T_{arr} in addition to f_{Hpeak}, T_{AB} and T_{peakfH} in response to warm acclimation[23].

Our quantitative genetic breeding design allowed us to directly assess evolutionary potential. We found that sire effects significantly contributed to offspring T_{AB}, f_{Hpeak} and scope for f_H (f_{Hscope}), whereas dam effects significantly influenced T_{arr} (Table 1). As males provide only genes to their offspring, sire effects are indicative of additive genetic effects[21]. The heritability of a trait—and, thus, its evolutionary potential—is the proportion of phenotypic variation attributable to additive genetic variation. Thus, our results indicate that there is standing genetic variation within the population that can allow T_{AB}, f_{Hpeak} and f_{Hscope} to evolve. Moreover, because T_{AB} is mechanistically linked with T_{opt}, the additive genetic effects underlying T_{AB} suggest that the close correspondence between T_{opt} and local thermal conditions among Pacific salmon populations[19] might be an adaptation brought about by selection on these genetic effects. Conversely, the dam effects on T_{arr} represent maternal environmental effects, which are non-genetic effects that often occur as a result of egg provisioning. The lack of genetic variation in T_{arr} could be due to past selection on upper thermal tolerance that subsequently depleted variation in the population. Although maternal effects themselves cannot respond to selection, the ability of females to partition these environmental effects (for example, egg size) can show additive genetic variance[24]. Indeed, thermal tolerance has been positively associated with maternal egg size both within[25] and among[22] populations of Pacific salmon, suggesting a potential role of egg size in the adaptation to warmer temperatures.

Several mechanisms might underlie the observed thermal plasticity and evolutionary potential of f_{Hmax} in chinook salmon. For example, exposure to high temperatures is associated with a heightened capacity of the sarcoplasmic reticulum to provide calcium ions for cardiac contraction in rainbow trout (*O. mykiss*)[26], whereas warm acclimation can increase the density of β-adrenoceptors on sockeye salmon (*O. nerka*) hearts, thereby altering their sensitivity to adrenergic stimulation[19]. These mechanisms may also be targets for selection to enhance maximum cardiac function: a heightened

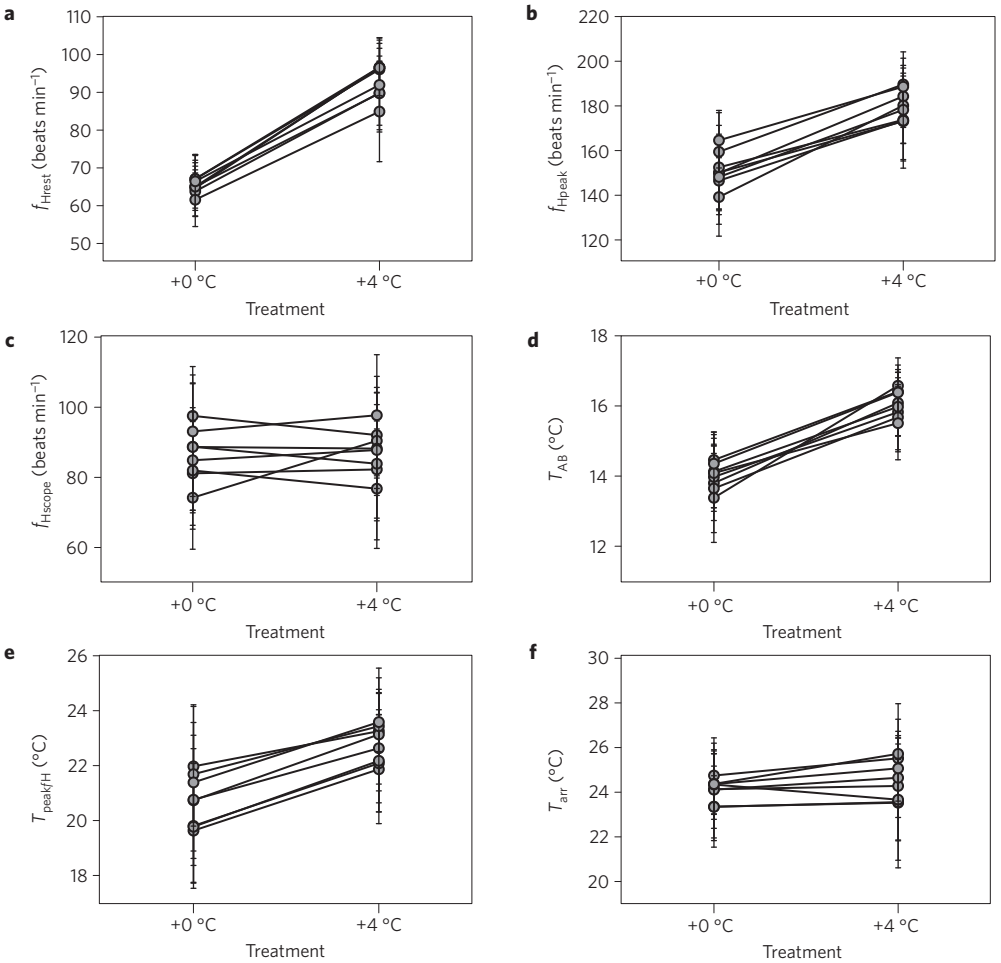

Figure 2 | Norms of reaction among paternal half-sib families of Quinsam River chinook salmon (*O. tshawytscha*). a–f, Offspring were reared in current (+0 °C) and future (+4 °C) temperature conditions and measured for their resting f_H (f_{Hrest}; **a**), peak f_H (f_{Hpeak}; **b**), scope for f_H (f_{Hscope}; **c**), Arrhenius break temperature (T_{AB}; **d**), the temperature at which f_{Hpeak} occurs (T_{peakfH}; **e**) and the arrhythmic temperature (T_{arr}; **f**). Shown are the means of each paternal family within the two treatments ±1 s.d. Connecting lines across treatments indicate the change in the mean phenotype of each family.

capacity of the sarcoplasmic reticulum has evolved in bluefin tuna (*Thunnus orientalis*), which probably contributes to their exceptionally high f_{Hpeak} (ref. 27), and more thermally tolerant populations of sockeye salmon have a greater density of ventricular β-adrenoceptors[19]. Regardless of the actual mechanism, the genetic and plastic effects detected here could act to enhance the cardiac capacity of chinook salmon in future environments.

We did not detect any significant treatment × sire effects for any of the analysed traits, although this effect on T_{AB} approached statistical significance ($P = 0.057$; Table 1 and Fig. 2). This result suggests a possible influence of a genotype-by-environment interaction effect on T_{AB}, which occurs when certain genotypes are more phenotypically plastic than others. Such genetic variation in plasticity could allow plasticity itself to evolve as an adaptation to environmental variation. Indeed, anadromous populations of brown trout (*Salmo trutta*) have a greater capacity for thermal plasticity in early life-history traits and global gene expression levels than do resident populations that experience a lower degree of environmental heterogeneity, suggesting that their thermal reaction norms are locally adapted[28,29]. The evolution of plasticity could thus be another important adaptation for populations faced with warmer and more variable environments.

The maximum present-day temperature during the juvenile stream residency of this population is 20.1 °C (Fig. 1). This maximum temperature lies just below the average T_{peakfH} of the +0 °C group measured here, indicating that the maintenance of maximum cardiac function corresponds well with maximum environmental temperatures. We also found that the average T_{arr} of the +4 group is only 4.4 °C higher than the present-day maximum temperature of the river, and that this upper thermal limit of cardiac function is constrained by a lack of plasticity and genetic variation. To evaluate how this constraint on thermal tolerance could limit this population in a warmer climate, we modelled the potential for future temperatures to exceed individuals' T_{arr}—thereby causing death—based on the most recent warming projections made by the Intergovernmental Panel on Climate Change[5]. We calculated the probability of a future 'catastrophic loss' in the population by estimating the likelihood of a day in which the mean stream temperature is greater than the T_{arr} of 50% of the individuals in the population, based on the variation in T_{arr} among all individuals measured here ($n = 280$). Using the average projection for western North America, we predict a 5% chance of catastrophic loss in the population by 2075 and a 17% chance by 2100. Under the maximum warming scenario, we predict a 55% chance of catastrophic loss by 2075 and a 98% chance by 2100. Although the pace of warming will differ among stream habitats that differ in water source and landscape, these findings indicate that projected increases in temperature will probably be detrimental to salmon populations.

In conclusion, the results presented here comprise the first quantitative assessment of both the heritability and the plasticity of oxygen-limited thermal tolerance. Our finding of a constraint on the upper limit of thermal tolerance suggests a susceptibility of Pacific

salmon populations to the projected increases in temperature. Nevertheless, the potential for adaptation and acclimation of T_{AB}, $T_{peak/H}$ and f_{Hpeak} indicates a degree of resilience to warming conditions. Moreover, the plasticity of T_{arr} might increase if salmon can undergo transgenerational acclimation of thermal tolerance, which has recently been identified as another important mechanism by which fish can avoid the loss of aerobic scope in high temperatures[30]. Although such adaptive mechanisms comprise a natural resilience to climate change, it is clear that the potential magnitude of change could preclude sufficient adaptive responses. Mitigation of climate change is thus needed to ensure the future viability of Pacific salmon populations, as well as the ecosystems and economies that they support.

Methods

Using diversion channels located at the Fisheries and Oceans Canada Quinsam River Hatchery, we caught eight males and eight females completing their spawning migration. Only unmarked, non-hatchery raised fish were used in the study. Gametes were taken from each spawner and transported to Yellow Island Aquaculture Ltd on Quadra Island. There, gametes were crossed in four 2×2 full-factorial crosses[21] to produce 16 different full-sib families. Each cross was replicated four times, with two replicates from each family being reared in one of two temperature treatments: present-day (+0 °C) or projected future (+4 °C). After entry into the exogenous feeding stage, offspring were given family- and replicate-specific tags and transported to the University of British Columbia in Vancouver. There, the +0 °C and +4 °C groups were kept for the remainder of the experiment at 8.0±0.8 °C and 12.4±0.3 °C, respectively.

We measured the response of f_{Hmax} to warming[3] in offspring from each family and temperature treatment. These measurements provide transition temperatures—T_{AB}, $T_{peak/H}$ and T_{arr}—that provide functional estimates of corresponding limitations in aerobic scope. As the heart is the primary mechanism supporting oxygen delivery, limitations on f_{Hmax} ultimately limit aerobic scope[15]. Indeed, in all of the studies performed so far, T_{AB} and T_{arr} have been found to be within 1–2 °C of the T_{opt} and the upper critical temperature of aerobic scope, respectively[3,4].

At the acclimation temperature of each fish (8 °C for the +0 °C group, 12 °C for the +4 °C group), individuals were anaesthetized and measured for their resting f_H (f_{Hrest}). f_{Hmax} was then pharmacologically induced and measured at every +1 °C temperature increment until cardiac arrhythmia was observed. An analysis of variance model was used to evaluate the genetic and plastic effects on offspring f_{Hrest}, f_{Hpeak}, f_{Hscope} ($=f_{Hpeak}-f_{Hrest}$), T_{AB}, $T_{peak/H}$ and T_{arr} (see Supplementary Methods for more details).

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Author contributions

All authors designed the experiment; N.J.M. conducted the experiment and data analyses; J.W.H. contributed materials and logistical support during the experiment; N.J.M., A.P.F. and B.D.N. wrote the paper. All authors provided intellectual input, and read and approved the manuscript.

Additional information

Supplementary information is available in the online version of the paper. Reprints and permissions information is available online at www.nature.com/reprints. Correspondence and requests for materials should be addressed to B.D.N.

Competing financial interests

The authors declare no competing financial interests.

EVOLUTIONARY ECOLOGY

Survival of the fittest

Evolutionary adaptation will help some animals cope with future climate change, but for juvenile salmon there may be limits to how far the thermal tolerance of cardiac function can adapt.

Philip L. Munday

Which animal populations will persist in a future warmer world depends, in part, on their ability to adapt to higher temperatures. As the world warms, populations can move to track preferred temperatures, they may acclimate to warmer temperatures through phenotypic plasticity, or they may become better suited to the new environment through evolutionary adaptation[1]. There are many examples of how projected future temperatures could dramatically affect the performance of a wide range of different plant and animal species, but whether these species will adapt over the longer term is largely unknown, especially in aquatic environments[2,3]. Writing in *Nature Climate Change*, Muñoz *et al.*[4] show that physiological traits important to the survival of juvenile chinook salmon have both plasticity and genetic potential to adapt to warmer temperatures. However, the news is not all good, because one key trait — the upper thermal limit for heart function — appears to lack plasticity or significant genetic variation. As a result chinook populations face an increasing risk of catastrophic population loss if river temperatures continue to warm.

A powerful way to examine evolutionary potential is to use quantitative genetic breeding designs, where a number of different males are each cross-bred with a number of different females and the phenotypic variation of the offspring is compared within and among family lines[1,2]. Using this technique it is possible to partition phenotypic variation in the offspring to that due to fathers, mothers, the interaction between mothers and fathers, and non-genetic environmental sources. Mothers contribute both their genes and nutritional provision to their babies in the egg. Fathers, however, just contribute their genes. By estimating the phenotypic variation due to fathers, and comparing it to the total phenotypic variation, one can estimate if there is heritable genetic variation in a trait of interest. This is exactly what Muñoz *et al.* did with chinook salmon, to test for heritable variation in thermal tolerance. They also went one step further: by rearing the baby salmon under two different temperature regimes, one matching current day river temperatures, and one matching projected future temperatures, they were also able to test for developmental plasticity in thermal tolerance.

In fish and other aquatic animals, the capacity to supply sufficient oxygen to the tissues may set the upper limits for thermal tolerance[5]. The heart is the primary organ that sends oxygenated blood to the tissues and it beats faster at higher temperatures to supply more oxygen. Muñoz *et al.* tested for plasticity and genetic variation in key aspects of cardiac function in the juvenile salmon at higher temperatures, including the maximum heart rate achieved, the optimum temperature for heart rate, and arrhythmic temperature (the point of cardiac failure)[6]. First, they found plasticity in maximum heart rate and the optimum and maximum temperatures for heart rate. Juvenile salmon reared at projected future temperatures had a higher maximum heart rate (180 versus 153 beats per minute), a higher optimum temperature for heart rate, and a higher temperature at which maximum heart rate was achieved. In other words, the heart performed better at higher temperatures in the fish that had grown up at the higher temperatures — a process known as developmental acclimation[7]. However, not all cardiac traits exhibited this plasticity. There was no difference in the arrhythmic temperature between the two temperature groups. This means that the temperature at which the heart fails did not respond to developmental acclimation in the same way as the other cardiac traits.

Importantly, Muñoz *et al.* also found significant additive genetic variation in maximum heart rate and the optimum temperature for heart rate. This means there is significant potential for evolutionary adaptation of these traits as river temperatures increase. Salmon in the future may inherit a higher peak heart rate and higher optimum temperature for heart rate that will help them cope with warmer water temperatures. However, the arrhythmic temperature did not exhibit significant genetic variation. This is bad news for the salmon because there was neither plasticity

DP RM / ALAMY

Chinook salmon, Pacific Coast from California to Alaska.

nor substantial genetic variation in this important physiological trait. Juvenile salmon may literally have a heart attack if river temperatures in the future exceed the arrhythmic temperature (24.5 °C).

To test the vulnerability of future chinook populations to climate change, Muñoz *et al.* estimated the likelihood that future river temperatures would reach the arrhythmic temperature, the point at which the heart fails causing death. They concluded that there was at least a 5% chance of catastrophic population loss by 2075 and up to a 98% chance by 2100. Not great odds for the salmon if global warming continues unchecked.

One positive, however, is that the quantitative genetic breeding design revealed significant variation in the arrhythmic temperature of juveniles that could be attributed to mothers. This suggests that the way mothers allot resources to their eggs can influence the cardiac function of their offspring. For example, larger eggs may produce fitter offspring with stronger hearts. This provides some hope that plasticity and heritability of maternal provisioning could potentially help juvenile salmon adapt to higher temperatures in the future. It may be mothers, not fathers, that hold the key to chinook population survival.

Most studies investigating the effects of climate change on aquatic species still focus on testing the acute effects of high temperatures and extrapolating these results to populations in the future. However, there is an increasing realization of the need to incorporate an evolutionary perspective if we are to reliably predict the success of future populations[2,3]. Key questions remain about the scope for evolutionary adaptation to climate change, and the pace of adaptation compared with the pace of environmental change. Also, how important is phenotypic plasticity compared with genetic evolution in responding to climate change? And what are the limits to adaptive responses? An important message from this study is the need to consider a range of phenotypic traits when examining evolutionary potential. While the salmon exhibited considerable plasticity and heritable genetic variation in most of the traits examined, this variation was absent in one key physiological trait. It may be that one trait, which has been honed by natural selection in the past, that determines the fate of chinook salmon in the future. ❐

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WARMING TRENDS

Adapting to nonlinear change

As atmospheric carbon dioxide concentrations rise, some regions are expected to warm more than others. Now research suggests that whether warming will intensify or slow down over time also depends on location.

Alexandra K. Jonko

When we need to wrap our head around a very complex problem, it is helpful to simplify and make approximations. Many of the methods we currently employ to understand climate change, arguably one of the most complex problems around, use approximations of linearity and aggregation of regional effects to global averages. In reality, all natural systems are nonlinear, and none of us live in a global average world. Deviations from these assumptions are particularly important when we are concerned with climate change adaptation strategies. However, integrated assessment models[1] — the tools developed to inform adaptation decisions — are often based on linear approximations of climate change. As they report in *Nature Climate Change*, Peter Good and colleagues[2] investigate sources of regional nonlinearities in climate model projections of future warming.

Integrated assessment models are important decision tools for policy makers. They represent the complex relationships between the earth system and social and economic realms[3]. Because they include so many different processes, their representation of the earth system is necessarily very simple, often consisting of only a few equations. Many of these models assume linearity in the response of climate to an external forcing.

In a linear system, doubling a perturbation doubles the response. In the context of global warming, the perturbation might be an increase in carbon dioxide concentrations. The resulting increase in surface temperatures is the system response. In a linear climate, the temperature response to a doubling of carbon dioxide levels would be exactly the same as the temperature response to a subsequent doubling. Making this approximation proves powerful when we are interested in the general behaviour of the climate system. However, when making decisions about adaptation and strategies, projections based on linear global assumptions are of limited use, and we need to take a closer look at how well they hold up in different locations and for different climate change scenarios. This is what Good *et al.*[2] have done using a framework, developed in previous work[4], that allows them to separate the climate's response to an external forcing (such as a doubling or quadrupling of atmospheric carbon dioxide) into its linear and nonlinear components.

Nonlinearities in climate have previously been studied both in observational warming trends[5] and in future model projections[6]. What distinguishes the work of Good *et al.*[2] from previous studies is their focus on regional patterns of nonlinearity. The metric they use to quantify nonlinearity is a spatially varying 'doubling difference' — the difference between the temperature change caused by the first and that caused by the second doubling of carbon dioxide. Positive doubling differences imply that the second doubling of carbon dioxide leads to a stronger warming than the first