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► To cite this version:

Marie Rescan, Daphné Grulois, Enrique Ortega-Aboud, Luis-Miguel Chevin. Phenotypic memory drives population growth and extinction risk in a noisy environment. *Nature Ecology & Evolution*, 2020, 4 (2), pp.193-201. 10.1038/s41559-019-1089-6 . hal-02998140

HAL Id: hal-02998140

<https://hal.science/hal-02998140>

Submitted on 13 Nov 2020

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1 Phenotypic memory drives population growth and extinction
2 risk in a noisy environment

3 Marie Rescan¹, Daphné Grulois¹, Enrique Ortega-Aboud¹, Luis-Miguel Chevin^{1*}

4 ¹. CEFE, CNRS, University of Montpellier, University of Paul Valéry Montpellier 3, EPHE, IRD,
5 Montpellier, France

6 * Corresponding author, e-mail: luis-miguel.chevin@cefe.cnrs.fr

7
8 **Random environmental fluctuations pose major threats to wild populations. As patterns of**
9 **environmental noise are themselves altered by global change, there is growing need to identify**
10 **general mechanisms underlying their effects on population dynamics. This notably requires**
11 **understanding and predicting population responses to the color of environmental noise, i.e. its**
12 **temporal autocorrelation pattern. Here, we show experimentally that environmental**
13 **autocorrelation has a large influence on population dynamics and extinction rates, which can be**
14 **predicted accurately provided that a memory of past environment is accounted for. We exposed**
15 **near to 1000 lines of the microalgae *Dunaliella salina* to randomly fluctuating salinity, with**
16 **autocorrelation ranging from negative to highly positive. We found lower population growth,**
17 **and twice as many extinctions, under lower autocorrelation. These responses closely matched**
18 **predictions based on a tolerance curve with environmental memory, showing that non-genetic**
19 **inheritance can be a major driver of population dynamics in randomly fluctuating**
20 **environments.**

22 The demography and extinction risk of wild populations is largely determined by responses to random
23 fluctuations in their environment¹⁻⁷, the magnitude and predictability of which are currently altered by
24 anthropogenic activities^{8,9}. In addition to their amplitude, stochastic (i.e., random) fluctuations are
25 characterized by their temporal autocorrelation, which determines the extent to which one time step is
26 a good predictor for the next. Temporal autocorrelation, sometimes described as the color of noise in
27 reference to the spectral decomposition of random fluctuations, is a ubiquitous feature of natural
28 systems^{10,11}. Because environmental autocorrelation characterizes the pattern and ordering of
29 environmental noise rather than its magnitude, it was originally thought to be irrelevant to expected
30 population dynamics in a stochastic environment, which instead were predicted to only depend on the
31 mean and variance of environmental fluctuations in population growth³. However, more recent theory
32 has shown that environmental autocorrelation may have a dramatic impact on population growth and
33 extinction risk in a stochastic environment¹²⁻¹⁶. The reason is that positive temporal autocorrelation
34 facilitates phenotypic tracking of environmental fluctuations by phenotypic plasticity^{17,18}, evolutionary
35 responses to natural selection¹⁹, or both, thereby modifying the mean and variance of population
36 growth rates. However theoretical predictions are scarcer for negatively autocorrelated fluctuations,
37 where high and low conditions alternate rapidly but predictably. More importantly, we lack empirical
38 validation of this theory in a form that combines the realism of a continuously varying environment
39 (such as temperature, humidity, salinity) with the high replication required to properly investigate
40 responses to stochasticity. High replication is all the more important as environmental autocorrelation
41 is expected to influence the variance of population size among independent realizations of stochastic
42 population dynamics, therefore increasing extinction risk¹².

43 An increasing number of studies have inferred the level of environmental stochasticity in population
44 growth in the wild^{20,21} and the demographic consequences of environmental autocorrelation^{22,23}.
45 However, attributing population size fluctuations to measured environmental factors is extremely
46 challenging in the wild, where such factors may largely covary among each other, and the origin of
47 their variation is unknown. Furthermore, observations from natural population generally consist of a
48 single realization of a process with a limited number of time points (usually below 50), whereas

49 assessing the range of likely outcomes for any stochastic process in the future requires many
50 replicates, and/or long time spans. Experimental studies in the laboratory with short-lived organisms
51 appears as a necessary bridge between theory and nature, because they allow testing theoretical
52 predictions with ample replication and precision, potentially yielding general insights about population
53 responses to environmental stochasticity. However, the few experimental studies about demographic
54 consequences of environmental autocorrelation have had contrasting findings²⁴⁻²⁷. To achieve
55 significant progress on this question, we need to overcome the specificities of each individual system
56 by establishing robust and general mechanistic links between environments and population
57 demographic processes.

58 Such a link can be provided by environmental tolerance curves, which measure how fitness or its life
59 history components (survival and fecundity) respond to abiotic environmental variables such as
60 temperature or salinity. Tolerance curves are increasingly popular tools connecting physiology and
61 ecology in response to climate change²⁸⁻³⁰, notably because they share many commonalities across
62 taxa and environmental factors, thus allowing for generalization. They are typically hump-shaped,
63 with an optimum at some intermediate environmental value that coincides to some extent with the
64 historical environment of the species^{31,32}. This non-linear relationship between the environment and
65 absolute fitness is predicted to strongly drive effects of environmental stochasticity on population
66 dynamics and extinction risk¹², but these predictions still largely lack empirical validation.
67 Furthermore, environmental tolerance curves can potentially be influenced by the history of
68 environments experienced by an individual^{30,33-35} (phenotypic plasticity) or its ancestors³⁶ (trans-
69 generational plasticity), but it is unclear to what extent population dynamics will be affected by such
70 environmental memory effects. To answer these questions, we carried out a highly replicated
71 experiment with a eukaryotic microorganism, in order to investigate how environmental
72 autocorrelation influences population dynamics and extinction risk in a stochastic environment.

73 **Results**

74 We exposed six ancestral lineages (hereafter genotypes) of the facultative sexual microalgae
75 *Dunaliella salina* to randomly varying salinities during 37 transfers (~100 generations). Using a liquid
76 handling robot, we transferred each line twice a week (every 3 or 4 days), diluting 15% of the
77 population of origin into fresh medium with controlled salinity, which differed across lines. More
78 precisely, our treatments consisted of 164 time series with the same mean ($\mu = 2.4\text{M NaCl}$) and
79 variance (standard deviation $\sigma = 1\text{M NaCl}$), but 4 autocorrelation levels, from negative ($\rho = -0.5$) to
80 highly positive ($\rho = 0.9$). In doing so, we modulated the pattern of salinity change between two
81 successive transfers, without modifying the overall magnitude of fluctuations (Figure 1 a. and b.,
82 upper panel). Importantly, 39 (out of 41) time series within each treatment were different, independent
83 realizations of the same stochastic process, allowing investigation of the variance introduced by
84 environmental stochasticity, which is known to be an important driver of extinction risk^{2,12}. We
85 tracked population densities and the proportion of extinct populations at each of 37 transfers (~100
86 generations, over 4 months) using optical density, fluorescence and flow cytometry. We then
87 estimated the intrinsic rate of increase between each successive transfers, assuming continuous logistic
88 growth following the 15% dilution (Figure 1c).

89 In addition to three single strains (genotypes A: CCAP 19/18, B: CCAP 19/18 and C: CCAP 19/15),
90 we used the three possible mixes of two of these strains (genotype AB, AC and BC), to test for
91 potential advantages of genetic diversity on population dynamics. One of the single genotypes (B)
92 grew too slowly to compensate for dilution at each transfer and got extinct rapidly (Extended Data 1)
93 even in constant treatments, so it was discarded from further analysis.

94 **Environmental autocorrelation strongly affects population size.** Environmental stochasticity
95 initially caused a rapid increase of variation in population size among our different lines of a same
96 stochasticity treatment (as materialized by the dashed confidence interval in Figure 2.a.), as expected
97 because population growth accumulates linearly on the log scale during a phase of exponential
98 growth^{2,12}. The stochastic variation among lines then settled to an approximately constant value,

99 characteristic of the stationary phase (days > 45, Figure 2a), where fluctuating population sizes
100 spanned several orders of magnitude, in contrast with the small variance among replicates in constant
101 environments (Extended Data 2). This is consistent with theory showing that stochastic environments
102 can cause large fluctuations in population sizes around their expected carrying capacity^{2,12,37}.

103 The distribution of log-population size was asymmetric for all treatments and genotypes, with a
104 negative skew causing an excess of very small populations undergoing high extinction risk (Figure
105 2.b. and e). Remarkably, this skewness is consistent with models of a moving optimum phenotype in a
106 stochastic environment¹² - equivalent to models of tolerance curves with an optimum
107 environment^{12,35,38} -, but not necessarily with classic phenomenological models where noise is added to
108 the population growth rate without an explicit tolerance curve^{2,3,39}. This suggests that fluctuations in
109 our experimental populations are driven to some extent by the interplay of a stochastic environment
110 with a hump-shaped tolerance curve with an optimum. However, density dependence in population
111 growth also impacts the distribution of population size, modifying the influence of the stochastic
112 environment^{2,40}. To control for this influence of density dependence, we analyzed the distribution of
113 the intrinsic growth rate r , *i.e.* the rate of exponential growth at low population density, extracted from
114 a model of continuous logistic growth (Figure 1.c). We found strong support for an asymmetrical
115 distribution of r ($\Delta\text{AIC} = -3713$ between a normal and a reverse gamma distribution), with an
116 estimated skewness between -1.7 and -0.4 (Figure 3.d).

117 Using the population sizes estimations obtained from the reverse gamma model, we found important
118 differences between the dynamics of populations exposed to different autocorrelation treatments. For
119 each of the genotype x autocorrelation treatment (from 438 to 833 data points), we computed the
120 distributions of the population size during the stationary phase, and analyzed the influence of
121 environmental autocorrelation on moments of these distributions. As environmental autocorrelation
122 increased, populations underwent fewer episodes of negative growth, and smaller fluctuations in size
123 (Figure 1). As a consequence, the mean (log) population size was larger under larger environmental
124 autocorrelation (Figure 2c, slope = 0.71 ± 0.11 , $p = 4.0 \cdot 10^{-5}$), contrary to classical population dynamics
125 predictions where the expected intrinsic growth rate only depends on the mean and variance of the

126 environment^{3,4,6}. The variance in log population size among replicate lines decreased with increasing
 127 autocorrelation (Figure 2d, slope = -3.0 ± 0.71 , $p = 1.3 \cdot 10^{-3}$). This contrasts with the common
 128 theoretical prediction that higher autocorrelation generates long batches of good/bad environments in
 129 different lines, thereby increasing population size variance among lines¹²⁻¹⁵, but is consistent with a
 130 contribution of environmental autocorrelation to phenotypic tracking of the environment (via
 131 phenotypic plasticity or genetic evolution), reducing the magnitude of population fluctuations¹⁹. We
 132 found a general trend for lower asymmetry (negative skewness closer to 0) with increasing temporal
 133 autocorrelation, but this effect highly depended on the strain (or mix) used and was not significant
 134 (Figure 3.d, slope = 0.19 ± 0.25). Temporal autocorrelation of the stationary population size was highly
 135 variable within genotype \times autocorrelation treatment (Figure 3.e). In particular, it was never found
 136 significantly different from 0, even in the highly autocorrelated treatment ($\rho = 0.9$), highlighting that
 137 temporal autocorrelation of the environmental factors translates only very weakly into autocorrelation
 138 of population sizes^{25,41-43}.

139 **Environmental memory governs population dynamics.** To reach more mechanistic insights
 140 into drivers of stochastic population dynamics in our experiment, we used the known salinity and
 141 population measurements to estimate a hump-shaped tolerance curve relating the intrinsic rate of
 142 increase $r(T)$ following transfer T to the current salinity S_T ,

$$r(T) = a_{univ} + b_{univ} \times S_T + c_{univ} \times S_T^2, \quad 1$$

143 where the subscript ‘univ’ is for univariate. This was compared to a bivariate tolerance curve that also
 144 includes an effect of memory of the previous environment (prior to the last transfer), via the salinity
 145 S_{T-1} ,

$$r(T) = a + b \times S_{T-1} + c \times S_T + d \times S_{T-1}^2 + e \times S_T^2 + f \times S_{T-1} \times S_T \quad 2$$

146 where e determines the tolerance breadth with respect to the current environment, d the breadth of the
 147 environmental memory effect, and f the interaction between past and current salinity.

148 We fitted parameters of the tolerance curve with (equation 2) or without memory (equation 1), under
 149 the same model of logistic growth as in the first section, assuming a Gaussian distribution of the

150 population size around its expected value (given the value at the previous time step). We found strong
151 support for the generalized bivariate tolerance curve ($\Delta AIC < -10^{-3}$ and p-values from likelihood ratio
152 test $< 10^{-9}$ for all genotypes, see Supplementary Table 1), revealing an important impact of
153 environmental memory on population growth. Growth rates were highest for populations transferred
154 from high to low salinity, while populations transferred from low to high salinities declined (Figure
155 3.a), notably because of high mortality that could be detected by flow cytometry (Extended Data 3a, b,
156 c).

157 To quantify the extent to which tolerance curves and memory effects explain the variation in growth
158 within and across autocorrelation treatments, we derived the moments of population growth rates
159 predicted by combining these tolerance curves with the realized salinity time series, and compared
160 them to their estimated counterparts that do not use information about salinity (Supplementary Table
161 1). The tolerance curve with memory correctly predicted increases in the mean (slope = 0.069 ± 0.009 ,
162 $p = 1.5 \cdot 10^{-5}$) and skewness (slope = 0.58 ± 0.09 , $p = 2.9 \cdot 10^{-5}$) of population growth rates with increasing
163 environmental autocorrelation, and a decrease in their variance (slope = -0.12 ± 0.01 , $p = 2.18 \cdot 10^{-8}$),
164 while all these effects were entirely missed by a memoryless tolerance curve (continuous vs dashed
165 lines in Figure 3b, c, d). The predicted and observed responses to environmental autocorrelation were
166 in very good quantitative agreement for the mean and variance of population growth rate (Figure 3b,
167 c), and were qualitatively consistent for the skewness of population growth rate (Figure 3.d). Temporal
168 autocorrelation of the growth rate displayed discrepancy between the fit from the data and prediction
169 from the tolerance curves (Figure 3e). Nevertheless, the tolerance curve with memory allowed for
170 negative autocorrelation of population growth rates despite positive environmental autocorrelation, as
171 observed in the data but not possible with a memoryless tolerance curve.

172 **A candidate mechanism: plasticity of glycerol content.** Although a range of physiological
173 mechanisms are probably involved in these population responses to salinity fluctuations, glycerol
174 content is a key candidate phenotypic trait. Glycerol acts as an osmoregulator, with intracellular
175 concentration responding nearly linearly to the environmental salinity⁴⁴. It is the major mechanism that
176 makes *Dunaliella* a model species for salinity tolerance⁴⁴. To investigate the possible role of this

177 metabolite in our population responses to salinity, we tracked the dynamics of intracellular glycerol
178 following a change in salinity. When going from high (3.5M NaCl) to low salinity, intracellular
179 glycerol concentration first changed almost immediately, with significant differences in glycerol
180 contents as soon as 16 minutes after transfer in 0.5, 2 or 3.5M NaCl (Supplementary Table 1),
181 probably through excretion⁴⁵, followed by a slower adjustment of glycerol level (Figure 4). In contrast,
182 the reciprocal transfer from low (0.5M NaCl) to high salinity, which requires *de novo* production of
183 glycerol⁴⁴, triggered slower change in glycerol content, with significant differences between transfer in
184 0.5 and in 2M NaCl found only after 135 minutes (Figure 4 and Supplementary Table 1). Most cells
185 even died before their glycerol content could be measured in the transfer to the highest salinity (Figure
186 4). This is consistent with our observation that transitions from low to high salinity are more
187 detrimental to the population dynamics than the reverse (Figure 3a).

188 **Extinction rates increase with decreasing autocorrelation.** Nearly half the lines went extinct
189 by the end of the experiment (Figure 5a). Environmental autocorrelation had a strong impact on
190 extinction risk. Populations from low autocorrelation treatments ($\rho = -0.5$ and $\rho = 0$) went more extinct
191 (72 and 61% extinct by the end of the experiment, median survival time: 77 days and 83 days,
192 respectively) than populations from positive autocorrelation treatments (33 and 34% extinct by the end
193 of the experiment for $\rho = 0.5$ and $\rho = 0.9$, respectively; $p < 1.10^{-4}$, log ranked test performed
194 independently for all genotypes, see Supplementary Table 1 for details). We found no evidence for an
195 effect of genetic variation on extinction risk in our experiment, as single-strain lines did not differ
196 significantly from mixed-strain lines, and there was no indication that recombination contributed to
197 population persistence in mixed-strain lines (Supplementary Information). Simulations using the
198 tolerance curve with environmental memory combined with the actual times series of environments
199 gave results qualitatively similar to our data (Figure 5b). By contrast, a memoryless tolerance curve
200 reversed the predictions, with the lowest extinction risk predicted under negative or null
201 autocorrelation. The reason for this reversal is that, without environmental memory (and neglecting
202 rapid evolution), environmental autocorrelation does not help phenotypic tracking of the
203 environmental optimum, and therefore has no direct effect on the distribution of population growth

204 rates (dashed lines in Figure 3b-d), but only influences how growth rates are integrated into the
205 population dynamics. Because all things being equal, more autocorrelated growth rates cause larger
206 variance in population size (as confirmed in simulations without memory, $p < 10^{-3}$), they are expected
207 to increase extinction risk in the absence of environmental memory^{12,15}. This effect is totally buffered
208 by environmental memory: with higher mean and lower variance in growth rates, populations
209 experiencing positively autocorrelated environments are less likely to fall below a critical threshold for
210 extinction.

211 **Discussion**

212 Random environmental fluctuations largely contribute to natural baseline rates of extinction¹⁻⁶, which
213 are currently aggravated by trends such as global climate change. Furthermore anthropogenic
214 activities, beyond their effects on mean environments, are altering the patterns of these random
215 fluctuations, including their temporal autocorrelation^{8,9}. There is thus a pressing need to empirically
216 characterize and quantify drivers of extinction risk in an autocorrelated stochastic environment. Using
217 a highly replicated experiment with many independent environmental time series, we found that
218 environmental autocorrelation can have a strong impact on population dynamics and extinction risk. In
219 other words, extinction risk depends not only on the mean and variance of environmental fluctuations,
220 but also on the order in which environments are encountered, within and across generations. This
221 implies that the color of environmental noise, as increasingly documented in the ecological
222 literature^{10,13,15,16,24-26}, is likely to be an important driver of population dynamics. Importantly, the
223 impacts of environmental autocorrelation on population dynamics could be predicted accurately in our
224 experiment using the simple and general concept of the environmental tolerance curve, which
225 summarizes all influences of the environment with a few parameters. This validates the utility of this
226 tool as a way to predict population dynamics in a changing environment, by connecting physiology to
227 ecology²⁸. However, we only reached accurate predictions when an influence of past environment was
228 accounted for in these tolerance curves.

229 The large influence of environmental memory on population dynamics in our experiment implies that
230 the phenotype of an individual cell is partly affected by the environment experienced by its recent
231 ancestor(s), which can be described as trans-generational phenotypic plasticity, or environment-
232 dependent parental effect, a form of non-genetic inheritance³⁰. There is abundant evidence in microbes
233 that prior exposure to stress can affect later stress responses⁴⁶, and such acclimation experiments
234 generally span over several cellular generations. In our experiment, genetic evolution could in
235 principle also contribute, because some lines are highly polymorphic, but the time scale of ~3
236 generations between transfers is not consistent with large evolutionary change between transfers. In
237 addition, clones isolated from mixed BC populations displayed similar tolerance curves as the whole
238 population (Extended Data 5), confirming that (trans-generational) phenotypic plasticity is the major
239 mechanism responsible for this short-term environmental memory.

240 Our experiment included elements of realism that are uncommon in the laboratory, such as
241 continuously distributed fluctuations of an environmental variable, and a large number of independent
242 realizations of the same stochastic process. This represents a large step towards bridging the gap
243 between theory and natural populations on quantitative rather than qualitative grounds, an important
244 goal for population ecology. Note that in our batch culture setting, the environment changed every 3 or
245 4 days, *i.e.* every 2 to 5 generations (depending on salinity) for our model species *D. salina*, while
246 salinity remained constant during the interval between transfers. This means that the autocorrelation
247 we report is not a value per generation or per unit time as in theoretical models, but across transfers.
248 This limitation is common to any experiment that uses organisms with continuous overlapping
249 generations in batch culture, since changing the environment continuously over time in a stochastic
250 way is essentially impossible. Such discretization does not hamper our conclusions about the influence
251 of environmental correlation on population growth and extinction risk in our experiment, but it should
252 be kept in mind when comparing quantitatively the effect of environmental autocorrelation in our
253 experiment versus in wild populations of higher organisms, where censuses and environmental
254 measurements are made at regular intervals (typically a year²).

263 **Methods**

264 **Dunaliella strains**

265 We used two closely related strains of the facultative sexual microalgae *Dunaliella salina*,
266 CCAP 19/12 (A) and CCAP 19/15 (C), and one more distant strain CCAP 19/18 (B). The lines were
267 not axenic nor clonal when received from Culture Collection of Algae and Protozoan (Glasgow). We
268 exposed them to increasing salinities (up to 4.8M) during several weeks to eliminate putative non-
269 *Dunaliella* species of eukaryotes and finally isolated them by cell sorting (BD FACS Aria IIu, up to
270 10^5 cells). We then initiated 1074 lines from six ancestral backgrounds, with either low or high genetic
271 diversity. Single strain lines (A, B and C) were used for the low genetic diversity treatment, and high
272 genetic diversity lines were initiated by a 50% mix of two strains (AB, BC and AC).

273 **Experimental design**

274 Each ancestral genetic background was exposed to three constant salinities (with 5 replicate lines per
275 salinity), and 156 fluctuating salinity times series. The latter consisted of 39 independent time series
276 (with the first one replicated 3 times) for each of four autocorrelation treatments: $\rho = -0.5$ (blue noise),
277 $\rho = 0$ (white noise), $\rho = 0.5$ and $\rho = 0.9$ (red noise).

278 At each transfer, a 15% aliquot of each population was replicated in fresh medium with controlled
279 salinity. The same dilution rate ($d = 15\%$) was used in all treatments, and set so as to compensate for
280 the intrinsic growth rate across variable environments for the slowest growing strain (strain B).
281 However, growth rates were overestimated in our preliminary measurements, so that the dilution rate
282 that we used caused the loss of most B lines before the end of the experiment.

283 Constant treatments were low ($[\text{NaCl}] = 0.8\text{M}$), medium (2.4M), and high (3.2M) salinities. The
284 stochastic treatments involved continuously ranging, (near) normally distributed salinities, with same
285 mean 2.4M and variance 1 among treatments, but temporal autocorrelation set by the treatment. We
286 generated fluctuating salinity time series as follows:

- 287 - Generate a first-order autoregressive (AR1) process of length 1000000, mean 2.4, variance 1
288 and autocorrelation set by the treatment. An AR1 process is a stationary Gaussian process, in
289 which the value at a given time step is a linear combination of the value at the previous time
290 step plus an independent normally distributed noise.
- 291 - Truncate this theoretical process so as to keep only reachable salinity values. This involved
292 removing salinities below 0 and above $[\text{NaCl}]_{\text{max}} = 4.8\text{M}$, as well as salinities that made the
293 transition from the previous time point impossible because of the constraints imposed by the
294 15% dilution upon transfer.
- 295 - Estimate the (reduced) variance of this truncated process
- 296 - Increase input variance (step += 0.05) and repeat all previous steps, until the reduced variance
297 $V = 1 \pm 0.01$
- 298 - Generate 39 times series of length 37 time steps for each autocorrelation treatment, using as
299 input variance the value from the iterative search described above. The mean, variance, and
300 temporal autocorrelation of these times series were checked to conform to their setpoint
301 values. The distribution of salinities in all time series for the different autocorrelation
302 treatments did not deviate from the expected Gaussian (Extended Data 4).

303 **Transfers and growth**

304 Experimental lines were cultured in 96 deep-well plates with artificial saline water + 2% Guillard's
305 F/2 marine water enrichment solution (Sigma; G0154-500ml). Lines were distributed in 18 plates,
306 such that each combination of ancestral genotype x environmental treatment was equally represented
307 in all plates. Population positions in each plate were then randomized across the 60 central wells
308 (external wells were not used because they experience larger evaporation). A liquid handling robot
309 (Biomek NX from Beckman) was used to serially transfer these lines twice per week (alternating
310 three-day and four-day cycles) into fresh medium. The salinity in this new medium was set
311 independently in each well by adjusting the volumes of a hyposaline ($[\text{NaCl}]_{\text{min}} = 0\text{M}$) and a
312 hypersaline ($[\text{NaCl}]_{\text{max}} = 4.8\text{M}$) solution, following

$$V_{\rho,i,T}^{Hyper} = V_{tot} \frac{[NaCl]_{\rho,i,T} - d [NaCl]_{\rho,i,T-1}}{[NaCl]_{max}} \quad 3$$

$$V_{\rho,i,T}^{Hypo} = V_{tot} - V_{\rho,i,T}^{Hyper}$$

313 where $[NaCl]_{\rho,i,T}$ and $[NaCl]_{\rho,i,T-1}$ are the setpoint salinities at transfers T and $T - 1$ (respectively) for
 314 time series i of autocorrelation treatment ρ , $V_{tot} = 800 \mu\text{L}$ is the total volume of culture, and $d = 15\%$ is
 315 the dilution rate as defined above.

316 Plates were covered with plastic lids, sealed with Parafilm and placed at a fixed position in a growth
 317 chamber, with temperature set at 24°C , and light at $200 \mu\text{mol.m}^{-2}.\text{s}^{-1}$ for 12:12h LD cycles.

318 **Population density measures**

319 After each transfer, cell density before dilution was measured using 3 complementary methods. We
 320 measured optical density (absorbance at 680nm) and fluorescence (excitation at 390-80nm, emission at
 321 685-40 nm) in a 200 μL sample of the each population using a BMG ClarioStar spectrophotometer.
 322 Cells from the same sample were then counted by flow cytometry (Guava EasyCyte HT). *Dunaliella*
 323 cells were isolated from debris using forward scatter (FSC), side scatter (SSC), red (695/50 nm) and
 324 yellow (583/26 nm) fluorescence emissions (excitation 488 nm, See Extended Data 3a., b. and c. for
 325 details). Cytometer performance was checked using a standardized fluorescent bead reagent
 326 (EasyCheck Kit, Merck Millipore) before each measure. Because of time limitation, only half of the
 327 plates went into the flow cytometer after each transfer, while the remaining plates were read in the
 328 next transfer, such that each plate was passed through the flow cytometer once per week.

329 To calibrate among our different measures of population density, we first regressed the fluorescence
 330 and OD over the population density as estimated from cytometer counts (see Extended Data 3 d. and
 331 e.), excluding the data points where spectrometer measures were too low and exhibited high
 332 uncertainty (fluorescence < 400 and absorbance < 0.01). We obtained:

$$\begin{aligned} [Duna]_{OD} &= 5273448 (OD - OD_{blank}) \\ [Duna]_{OD} &= 11.543428 (Fluo - Fluo_{blank}) \end{aligned} \quad 4$$

333 where OD_{blank} and $Fluo_{blank}$ are the fluorescence and absorbance in wells known to be empty (control
334 wells or extinct populations) within each given plate.

335 **Statistical analysis**

336 We analyzed population dynamics using an ad hoc state-space model. As typical in state-space
337 models⁴⁸, our model comprised two main components: (i) an underlying, unobserved process
338 describing the true stochastic dynamics of the populations size; (ii) and an observation model, which
339 describes how this process translates into measurements, with errors that are independent after
340 conditioning by the underlying process. We wrote an explicit likelihood function in C++ and
341 optimized it in R (version 3.5.2), using the TMB package⁴⁹. R and C++ codes are available from the
342 Dryad digital repository⁵⁵.

343 Observation model

344 In a preliminary experiment, we determined the error distributions of cytometer counts, absorbance
345 and fluorescence error. We made replicated (x3) measured of population sizes in serial dilutions of a
346 *Dunaliella* culture, with concentration ranging from 30 to 10^6 cells.mL⁻¹, and fitted generalized linear
347 regression between measurements and the known relative population sizes, expressed as a ratio of the
348 maximal population size. We compared AIC from normal, lognormal, poisson and negative binomial
349 models. Absorbance and fluorescence error were respectively normally and log-normally distributed,
350 while cytometer measures followed a negative binomial distribution, with mean the actual population
351 size. We thus used as the observation model for cytometer counts C , fluorescence F and absorbance
352 OD ,

$$\begin{aligned} C &\sim \mathcal{NB}(N, \theta_C) \\ \log(F) &\sim \mathcal{N}(\log(N), \theta_F) \\ OD &\sim \mathcal{N}(N, \theta_{OD}) \end{aligned} \tag{5}$$

353 where \mathcal{NB} denotes a negative binomial, N is the true population density (governed by the unobserved
354 process), and the θ are parameters that control the error variances (or overdispersion for cytometer
355 counts) of these measurements.

356 Process model

357 We assumed that population density followed continuous logistic growth:

$$\frac{dN(t)_{g,\rho,i}}{dt} = r(t)_{g,\rho,i} \left(1 - \frac{N(t)_{g,\rho,i}}{K_g} \right) \quad 6$$

358 where $N(t)_{g,\rho,i}$ is the population density for the population from strain g , environmental treatment ρ ,
359 and time serie i at time t , K_g the carrying capacity of genotype g and $r(t)_{g,\rho,i}$ its intrinsic growth rate in
360 the environment experienced at time t . In the constant treatment analysis, K_g was also allowed to vary
361 between salinity treatments ($K_{g,S}$).

362 Given that $r(t)_{g,\rho,i}$ is constant over the time interval between two successive transfer, population size at
363 transfer $T + 1$ can be implemented from population size at transfer T by integrating equation 6
364 ($\Delta t_{T,T+1} = 3$ or 4 days), following a 15% dilution rate (d):

$$N(T + 1)_{g,\rho,i} = \frac{d N(T)_{g,\rho,i} e^{\Delta t_{T,T+1} r(T)_{g,\rho,i}} \times K_g}{K_g - d N(T)_{g,\rho,i} (e^{\Delta t_{T,T+1} r(T)_{g,\rho,i}} - 1)} \quad 7$$

365 A convenient property of state-space models is that observation errors are independent conditional on
366 the process⁵⁰ (and independent of the process). Therefore, the overall log-likelihood in our model was
367 simply the sum of the log likelihood of the process (equation 7, 8, 9, 10) and that of all observations
368 (equation 5), producing an explicit formula amenable to numerical optimization.

369 *Density dependence analysis using constant treatments*

370 As our model was unable to estimate density dependence directly from the time series of fluctuating
371 salinities, we first analyzed the population dynamics from constant time series in an independent
372 model for this purpose. We considered that both the intrinsic growth rate $r_{g,S}$ and the carrying capacity
373 $K_{g,S}$ were constant across replicates and transfers for one strain g in one salinity S . The probability
374 distribution of population size at transfer $T + 1$, for strain g and salinity treatment S is then

$$N(T + 1)_{g,S,i} \sim \text{logNormal}(\text{LogisticGrowth}(N(T)_{g,S,i}), \beta) \quad 8$$

375 where $\text{LogisticGrowth}(d N_{g,S,i,T})$ is the continuous logistic function given by equation 8 and β is the
376 process error. For each genotype, likelihood ratio test between a model where $K_{g,S}$ was constant across

377 salinity and a model where $K_{g,S}$ varies with salinity was performed. We found that $K_{g,S}$ differed
 378 significantly but slightly between salinity treatments (Extended Data 2 d), so we used a constant K_g
 379 for each genotype in further analysis.

380 *Fluctuating growth analysis*

381 Environmental fluctuations caused variation in the intrinsic rate of increase r in all our stochastic lines.
 382 The distribution of r results from the interaction between moments of the stochastic environment
 383 (mean, variance and autocorrelation) and the growth response of each genotype to the environment.
 384 The distribution of r may be characterized by its mean, variance, but also skewness¹² and potentially
 385 by the autocorrelation of r across transfers. We estimated the parameters of this distribution for each
 386 autocorrelation treatment x genotype, within the state space model described by equations 5 and 8. We
 387 fitted either an autocorrelated normal (equation 9) or a reverse gamma distribution (equation 10) for
 388 $r(T)_{g,\rho,i}$,

$$r(T)_{g,\rho,i} \sim \mathcal{N} \left((1 - \rho_r) \bar{r}_{g,\rho} + \rho_{r_{g,\rho}} r(T-1)_{g,\rho,i}, (1 - \rho_{r_{g,\rho}}^2) \sigma_{r_{g,\rho}}^2 \right) \quad 9$$

or

$$r_{max_{g,\rho}} - r(T)_{g,\rho,i} \sim \Gamma(\alpha_{g,\rho}, \beta_{g,\rho}) \quad 10$$

389 where r_{max} , $\alpha_{g,\rho}$ and $\beta_{g,\rho}$ are the maximal growth rate, the shape and the scale parameter for the
 390 gamma distribution for strain g in the ρ environment. Mean $\bar{r}_{g,\rho}$, standard deviation $\sigma_{g,\rho}$ and skewness
 391 $skew(r_{g,\rho})$ were simultaneously estimated (estimation and standard error) from $r_{max_{g,\rho}}$, $\alpha_{g,\rho}$ and $\beta_{g,\rho}$
 392 using the ADREPORT procedure of the *TMB* package.

393 Population densities were estimated as random variables with a residual component of variation, and
 394 were hence only very weakly affected by the assumed shape of the growth rate distribution ($r^2 > 0.99$
 395 between the normal and the reverse gamma estimates, see Extended Data 6). This allowed us to
 396 visualize the goodness of fit for the reverse gamma and the autocorrelated normal. Using the
 397 population size estimates, we computed the realized intrinsic growth rate, corrected for density
 398 dependence:

$$\widehat{r(T)}_{g,\rho,i} = \frac{\text{Ln} \left[\frac{K_g/d \widehat{N(T)}_{g,\rho,i} + 1}{K_g/N(T+1)_{g,\rho,i} + 1} \right]}{\Delta t_{T,T+1}} \quad 11$$

399 and plotted them against the predicted distribution fitted by the normal and reverse gamma model
 400 (Extended Data 1).

401 To assess the extent to which population fluctuations were driven by responses to fluctuating salinity,
 402 we then used salinity as an environmental covariate. We explicitly expressed the growth rate $r(T)_{g,\rho,i}$
 403 as a function of both the current ($S_{g,\rho,i,T}$) and the previous salinity ($S_{g,\rho,i,T-1}$). We modeled a bivariate
 404 tolerance curve using a 2nd order polynomial, with parameters depending on the strain g ,

$$r(T)_{g,\rho,i}^S = a_g + b_g \times S_{\rho,i,T-1} + c_g \times S_{\rho,i,T} + d_g \times S_{\rho,i,T-1}^2 + e_g \times S_{\rho,i,T}^2 + f_g \times S_{\rho,i,T-1} \times S_{\rho,i,T} \quad 12$$

405 leading to equation 2 in the main text.

406 Population size at $T + 1$ then followed a logistic growth (equation 7) with growth rate determined by
 407 current and previous salinity following equation 10, and we assumed a constant Gaussian distribution
 408 of the residuals generated by the process error, with standard deviation β :

$$\log(N(T + 1)_{g,S,i}) \sim \mathcal{N} \left(\log \left(\text{LogisticGrowth}(N(T)_{g,S,i,T}, r(T)_{g,\rho,i}^S) \right), \beta \right) \quad 13$$

409 Similar analysis was performed using only the salinity of growth ($S_{g,\rho,i,T}$) in order to fit a tolerance
 410 curve without environmental memory (equation 1 in the main text):

$$r(T)_{g,\rho,i}^S = a_{univ,g} + b_{univ,g} \times S_{\rho,i,T} + c_{univ,g} \times S_{\rho,i,T}^2 \quad 14$$

411 Moments of population density and intrinsic growth rates.

412 We used the population density outputs of the gamma model (equation 10) to estimate the mean,
 413 variance, skewness and autocorrelation of log population density of non-extinct lines during the
 414 stationary phase (day > 40), for each autocorrelation treatment \times genotype combination (39
 415 independent lines over up to 26 transfers – replicates 2 and 3 of series 1 were excluded from this
 416 analysis). Confidence intervals for these estimates were obtained by bootstrap (1000 simulations),
 417 where population densities were sampled for each simulation from a normal distribution with mean

418 and standard deviation given by their corresponding estimate and standard error. We then performed
419 mixed linear regression between these computed moments and the environmental autocorrelation, with
420 ancestral genotype as a random factor, using the R package *lme4*⁵¹. To include the uncertainty in the
421 estimation of population size moments, we performed 1000 generalized linear regression with
422 population moment sampled from a normal distribution with parameters their estimate and error, and
423 corrected for multiple imputation using the R package *MICE*⁵².

424 Similar regressions were performed on 1000 simulations of the growth rate moments, sampled from a
425 normal distribution with parameters the estimate and standard error obtained from the gamma model.
426 Mean, variance, skewness estimates, and their associated errors, were directly fitted in the gamma
427 model. Autocorrelation of the intrinsic rate of increase was computed using the realized population
428 sizes estimated in the gamma model, with similar bootstrap and sampling as used for the population
429 log density analysis.

430 Fitted moments of the intrinsic rate of increase were compared to analytical predictions based on the
431 tolerance curves, with or without memory. Subsequent salinities in our time series closely follow a
432 binormal distribution (except for a weak truncation caused by our experimental design, see Extended
433 Data 4), with mean $\mu = 2.4M$ and variance $\sigma^2 = 1$ at both steps, and correlation given by the
434 environmental autocorrelation ρ . Combining this with the tolerance curve with memory leads to
435 explicit formulas in the Mathematica 11 notebook available from the Dryad repository⁵⁵. In particular,
436 we found that the mean growth rate \bar{r} increases linearly with the environmental autocorrelation $\rho(E)$,
437 while the variance $\text{Var}(r)$ is a quadratic function of $\rho(E)$. Analytic formula for the skewness and the
438 variance are more complex, but show notably that environmental memory allows for negative
439 autocorrelation of the growth rate $\rho(r)$, even when the environment is positively autocorrelated, a
440 pattern that we found in our data but that could not be achieved without a memory effect.

441 Without memory (equation 14), the moments of the distribution of growth rates take a simpler form,
442 summarized in Supplementary Table 2 (details in Mathematica 11 notebook in Dryad digital
443 repository⁵⁵). In the absence of environmental memory, the mean, variance and skewness of the
444 intrinsic rate of increase do not change with environmental autocorrelation ρ . Skewness is always

445 negative for humped-shaped tolerance curves ($c_{univ} < 0$). The effect of the environmental
446 autocorrelation $\rho(E)$ on the autocorrelation of the intrinsic rate of increase $\rho(r)$ depends on the distance
447 between the salinity optimum and the mean environment. When the phenotypic optimum corresponds
448 to the mean environment, $\rho(r) = \rho^2(E)$, while $\rho(r)$ tends rapidly towards $\rho(E)$ as the mismatch between
449 environmental mean and salinity optimum increases.

450 **Survival analysis**

451 We determined the extinction time for each population in the stochastic environment. Because OD,
452 fluorescence and cytometry measures were not precise enough to discriminate between population
453 sizes below 1000 cells.mL⁻¹, we considered that a population was extinct from the time it fell below an
454 estimated 1000 cells.mL⁻¹ (corresponding to a small number of cytometer counts, and a negligible
455 density compared with the carrying capacity close to 10⁶) and remained under this threshold for at
456 least 5 transfers. After this delay, observed growth events that occurred only in 8 populations were due
457 to contamination.

458 The survival responses in each treatment were then assessed by plotting the Kaplan-Meier survival
459 curves (non-parametric model for the ratio of non-extinct populations through time), and analyzing
460 differences in survival by log rank tests (*ggsurvplot* R package⁵³). Independent analyses were
461 performed for each genotype, and average extinction rates and autocorrelation effects were found
462 similar in all genotypes except B. To illustrate these effects, we plotted the Kaplan-Meier survival
463 curves and median survival time (time by which 50% of the population were extinct) for the joined
464 genotypes in each autocorrelation treatment (Figure 5).

465 **Simulations**

466 Tolerance curves fitted with (equation 12) and without (equation 14) memory were used to simulate
467 population dynamics under our fluctuating salinity treatments. Process noise and observation errors
468 were not simulated. Population size variance was analyzed using the same bootstrap method as used
469 for the real dynamics, except that here the true simulated population sizes were known and not

470 sampled from their estimation distribution. Simulations did not include demographic stochasticity, so
471 simulated lines were considered extinct after population densities were found below 1000 cells.mL⁻¹.

472 **Intracellular glycerol dosage**

473 A background culture of genotype C was used to measure variation in intracellular glycerol
474 concentration in response to salinity changes. The ancestral population was cultivated in 2.4M for 10
475 days until it reached the late exponential phase. It was then split and acclimated for 7 days in 50mL
476 flasks at low (0.5M) and high (3.5M NaCl) salinities. Each of these flasks was then split again and
477 transferred to 0.5M, 2M and 3.5M NaCl in 50mL flasks, and sampled after 16, 45, 72 minutes, 2h15,
478 4h15 and 1 day for glycerol dosage.

479 Intracellular glycerol concentration was estimated from the difference between total and extra-cellular
480 (after cell filtration) glycerol concentrations. 800μL Free Glycerol Reagent (Sigma Aldrich) was
481 mixed to 200μL culture (with or without cells), and we measured optical density at 540nm after 5
482 minutes incubation at 37°C. Glycerol concentration was then interpolated from a (linear) standard
483 curve. *Dunaliella* densities were also estimated by flow cytometry, and we made two independent
484 replicates of all measures. Intracellular glycerol concentration per cell was computed as

$$485 \quad [\text{glycerol}]_{\text{intra}} = \frac{[\text{glycerol}]_{\text{total}} - [\text{glycerol}]_{\text{extra}}}{[\text{Dunaliella}]},$$

486 where [] denotes a density/concentration. We computed the first-order standard errors of the intra-
487 cellular glycerol concentration using the delta method, accounting for the (independent) error of the
488 cytometer, the total and the extracellular glycerol measures (Figure 4). We then performed Wald test
489 to assess whether different glycerol levels were found under different treatments (Supplementary
490 Table 1).

491 **Data availability statement**

492 The population dynamics and glycerol acquisition data that support the findings of this study are
493 available from the Dryad digital repository⁵⁴.

494 **Code availability statement**

495 R, C++ and Mathematica codes are available from the Dryad digital repository⁵⁵.

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613

614 **Acknowledgements**

615 This work was funded by the European Research Council (ERC) under the European Union's Horizon
616 2020 research and innovation programme (grant agreement STG-678140-FluctEvol). We thank the
617 MRI-IGMM platform for access to the cell sorter, the GenSeq platform (LaBEX CEMEB,
618 Montpellier) for sequencing, and O. Cotto, C.A Klausmeier, R. Lande, and J. Tufto, and three
619 anonymous reviewers for helpful feedback.

620 **Author Contribution**

621 L-M and M.R designed the experiment. D.G and M.R conducted the population dynamics experiment.
622 L.M and M.R performed the statistical analysis. D.G. and M.R extracted and amplified barcode genes
623 of all lines and D.G, E.O-A and M.R analyzed the bioinformatics data.

624 **Competing interests**

625 The authors declare no competing interests.

626 **Figures legends**

627 **Figure 1: Salinity, growth rates and population dynamics for two time series with different**
628 **environmental autocorrelations.** a. Times series number 19 for ancestral genotype AC and
629 autocorrelation 0. b. Times series number 22 for ancestral genotype AC and autocorrelation 0.9. For
630 the population growth rate, colors indicate whether growth rate compensates (blue, $r > 0.542$) or not
631 (red) the bi-weekly dilution. Population size estimates and standard errors from the reverse gamma fit
632 (solid line and ribbon) are represented together with the three raw measurements at each transfer
633 (dots): cytometer counts (black), fluorescence (dark gray) and optical density (light gray). c. Estimated
634 logistic population dynamics during transfers 14 to 19 (days 49 to 76), accounting for the biweekly
635 dilution (arrows down). Red points and error bars correspond to the estimates and standard error for
636 population size fitted in the model. Note that even when the population reaches high densities (here
637 $> 2 \cdot 10^5$, while carrying capacity is estimated around 10^6), the biweekly 15% dilution leads to nearly
638 exponential growth after each transfer. The colored straight lines materialize the exponential phase,
639 over which the population grows at rate r , the intrinsic rate of increase, colored in the same way as in
640 the times series of r above.

641 **Figure 2. Population dynamics in a stochastic environment.** Colors correspond to the temporal
642 autocorrelation of salinity time series: -0.5: blue, 0: green, 0.5: orange, 0.9: red. a. Time series of
643 population sizes under the 156 stochastic salinity series are plotted for genotype A (shaded lines),
644 together with the exponential of the mean log population size (solid line) \pm standard deviation (dashed
645 lines). Population densities larger than $1000 \text{ cells.mL}^{-1}$ are plotted on the log scale, on which mean and
646 standard deviation were also estimated, excluding extinct lines. Population sizes under 1000 are
647 plotted on the linear scale, thus allowing tracking extinctions. b. Histograms of log-population size
648 during the stationary phase for genotype A (day > 40). Panels c-f: Moments of the distribution of log
649 population size during the stationary phase (computed on 22497 points). Different symbols represent
650 different genotypes: A (■), C (+), AB (▲), AC (×) and BC (●), and bootstrapped 95% confidence
651 interval are represented. The solid line represents the generalized linear regression on environmental
652 autocorrelation.

653 **Figure 3. Population growth rate in response to environmental autocorrelation.** a. Tolerance
654 curve with environmental memory relating growth rate r (intrinsic rate of increase) to the current (S_T)
655 and previous salinities (S_{T-1}), for genotype C (gray surface, black to light gray: $r = -2$ to $r = 1$),
656 compared with the 3119 growth rates fitted under the reverse gamma model, under each
657 autocorrelation treatment (points – same colors as Figure 2). The thick black line corresponds to the
658 growth rate that allows overcoming the biweekly dilution ($r = 0.542$) and the thin black line shows
659 $r = 0$. Ellipsoids at the bottom materialize the joint distributions of pre- and post-transfer salinities,
660 under our four autocorrelation treatments. Panels b-e: Moments of the intrinsic rate of increase of lines
661 A (■), C (+), AB (▲), AC (×) and BC (●) in the four autocorrelation treatments. Mean (b.), variance
662 (c.), skewness (d.) and autocorrelation (e.) of r are plotted against environmental autocorrelation for all
663 genotypes, with their standard errors. Predictions from the tolerance curve with (solid line) or without
664 (dotted line) environmental memory are also plotted for genotype C.

665
666 **Figure 4: Intracellular glycerol concentration in genotype C after transfer to a new salinity.** The
667 transfers were at time = 0, from 0.5M (dashed lines) or 3.5M (solid lines), to 0.5M (blue), 2M (purple)
668 and 3.5M (red) NaCl. Transfer from 0.5 to 3.5M caused high mortality, and thus could not be
669 analyzed. Each measurement was replicated twice, and first-order standard errors (bars) were
670 computed using the delta method, accounting for the independent errors of the extra-cellular, total
671 glycerol and population size (cytometer) estimations. The first time point corresponds to 16 min post-
672 transfer.

673 **Figure 5: Population extinctions across environmental autocorrelations.** a. Survival curves
674 (Kaplan-Meier) for the pooled genotypes A, AB, AC, BC and C, under the four autocorrelation
675 treatments (same colors as in Figure 2). For each autocorrelation treatment, the solid lines show the
676 estimated proportion of non-extinct populations, among the initial 195 populations (39 independent
677 time series x 5 genotypes), and the dashed lines indicate 95% confidence bands. Stars and numbers
678 indicate median survival times (times at which 50% of the populations has gone extinct) in the
679 corresponding survival curve. b-c: Survival curves estimated from simulations parametrized with (b)
680 or without (c) environmental memory, using the same salinity time series as in the experiment.