

Digest: Diet links cryptic genetic variation to growth-aging trade-offs

Luna Canaj 

Department of Biology, Western University, London, ON, Canada

Corresponding author: Department of Biology, Western University, 1151 Richmond Street, London, Ontario, Canada. Email: lcana@uwo.ca

This article corresponds to Shephard, A. M., Rohner, P. T., Ledón-Rettig, C. C. (2026). Cryptic genetic variation revealed by diet uncovers a trade-off between growth and telomere length. *Evolution*, 80 (6), 1357–1364. <https://doi.org/10.1093/evolut/qpag042>.

Abstract

Antagonistic trade-offs between early-life fitness and somatic maintenance are predicted to maintain variation in longevity, yet empirical support is scarce. Shephard et al. (2026) experimentally tested whether such trade-offs are concealed under typical developmental conditions using Mexican spadefoot tadpoles (*Spea multiplicata*) reared on typical or atypical diets. The atypical diet revealed cryptic genetic variation in larval growth and a significant negative genetic correlation with telomere length, indicating a diet-dependent trade-off in somatic maintenance.

Heritable variation in lifespan stubbornly persists despite evolutionary theory suggesting it should be eliminated. How is it that individuals in the same population age at different rates? A leading explanation is that variation in lifespan is maintained by antagonistic trade-offs: Genetic variants that enhance early-life traits such as growth or fecundity come at a cost to somatic maintenance processes (Shephard et al., 2026; Williams, 1957).

Empirical validation of these trade-offs has proven elusive, given the infrequent observations of discernable negative genetic correlations between growth and lifespan in natural populations (Shephard et al., 2026). An under-researched explanation concerns cryptic genetic variation, which refers to genetic differences that have minimal or no observable phenotypic effect under typical environmental conditions but manifest when organisms are exposed to novel or stressful environments (Shephard et al., 2026). Nevertheless, experimental data directly linking cryptic genetic variation to trade-offs with somatic maintenance remain scarce.

Telomeres serve as a valuable indicator of somatic maintenance and genomic integrity and are an established correlate of longevity. Early life telomere length predicts future survival in many vertebrate species, with their shortening over successive cell divisions being associated with impaired cellular and tissue performance (Shephard et al., 2026). Rapid growth is expected to accelerate telomere shortening either through demanding more cell divisions or because metabolic by-products of accelerated growth induce oxidative stress (Shephard et al., 2026). The inconsistent detection of the expected negative relationship suggests that trade-offs might only be apparent under specific environmental conditions.

To examine this further, Shephard et al. (2026) exploited the ecology of North American spadefoot toads. The Mexican spadefoot toad (*Spea multiplicata*) typically feeds on detritus (non-living particulate organic matter) during larval development. Their ephemeral desert ponds also contain live fairy shrimp, a protein-rich food source capable of accelerating larval growth (Shephard et al., 2026). In sympatric populations, *S. multiplicata* is competitively excluded from shrimp by the plains spadefoot (*S. bombifrons*), making shrimp an ecologically atypical diet. Prior work confirms stress under the shrimp diet, causing delayed metamorphosis that reduces larval survival (Shephard & Ledón-Rettig, 2025).

To test their hypothesis, full-sibling families of *S. multiplicata* were reared on either detritus or shrimp using a split-brood design. Six larvae per family were reared on each diet, with three randomly selected juveniles per condition subsequently euthanized for telomere analysis. Telomere length was measured by quantitative polymerase chain reaction (qPCR) from hindlimb muscle tissue ($n = 60$). Mixed models estimated diet-specific heritability and genetic correlations between traits were estimated using best linear unbiased predictors (BLUPs).

Under the detritus diet, heritability for larval growth rate was low ($H^2 = 0.11$), but leapt to $H^2 = 0.40$ on the shrimp diet (Figure 1). This is characteristic of cryptic genetic variation, as family lineage became far more predictive of growth rate only under the atypical diet. Telomere length heritability remained stable across diets ($H^2 = 0.21$). A significant negative genetic correlation emerged between growth rate and telomere length on the shrimp diet ($r = -0.88$, $p < 0.001$), compared to a negligible correlation on the detritus diet ($r = 0.22$, $p = 0.55$). This reveals a trade-off be-

Received March 19, 2026; accepted April 22, 2026

Associate Editor: Kati Moore; Handling Editor: Jason Wolf

© The Author(s) 2026. Published by Oxford University Press on behalf of The Society for the Study of Evolution (SSE).

All rights reserved. For commercial re-use, please contact reprints@oup.com for reprints and translation rights for reprints. All other permissions can be obtained through our RightsLink service via the Permissions link on the article page on our site-for further information please contact journals.permissions@oup.com

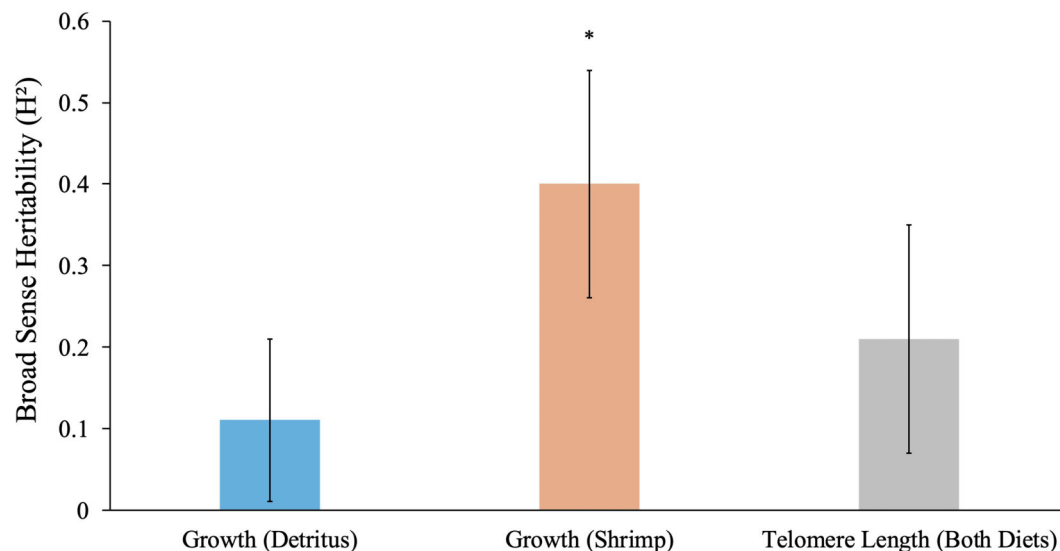


Figure 1. Broad-sense heritability (H^2) for larval growth rate on detritus and shrimp diets, and for juvenile telomere length across both diets, in *Spea multiplicata*. Genetic variance for larval growth rate was statistically significant only on the shrimp diet ($p < 0.001$, denoted by *), while variance on the detritus diet did not differ significantly from zero ($p = 0.195$). Heritability for juvenile telomere length remained stable across diets. Larval growth rate heritability reflects diet-specific estimates, whereas telomere length heritability reflects a single pooled estimate, as the best-fitting model did not support diet-specific genetic variance for this trait. Error bars represent ± 1 SE. Values derived from Shephard et al. (2026).

tween rapid growth and somatic maintenance that is absent under normal dietary conditions. The proximate mechanisms may involve heightened metabolic costs of rapid growth on an atypical diet, or toxic nitrogenous by-products of high-protein consumption (Moatt et al., 2020). The authors note, however, that because the correlation analyses relied on BLUPs and a full-sibling design that cannot separate parental effects, caution should be taken when interpreting results.

The authors suggest a potential application of these findings for human health research, where shifts toward evolutionarily novel diets, such as processed foods, may reveal similar trade-offs and susceptibility to age-related disease. Norwitz et al. (2021) illustrate this parallel with the ApoE4 allele, where carriers face elevated Alzheimer's risk in Western dietary contexts, yet the risk appears attenuated in populations following traditional Mediterranean diets (Norwitz et al., 2021). The authors describe how Italian ApoE4 carriers living in Italy survived into old age at normal rates, whereas those who had emigrated to the United States did not (Norwitz et al., 2021).

In summary, heritability is a property of a trait within a given environment: The conditions under which organisms are studied shapes which genetic variants become phenotypically visible. In their study, Shephard et al. (2026) provide compelling evidence that sus-

ceptibility to aging and disease is a heritable trade-off where expression is contingent on the organism's environment.

Conflict of interest

The author declares no conflict of interest.

References

- Moatt, J. P., Savola, E., Regan, J. C., Nussey, D. H., & Walling, C. A. (2020). Lifespan extension via dietary restriction: Time to reconsider the evolutionary mechanisms? *BioEssays*, 42(8), 1900241. <https://doi.org/10.1002/bies.201900241>
- Norwitz, N. G., Saif, N., Ariza, I. E., & Isaacson, R. S. (2021). Precision nutrition for Alzheimer's prevention in ApoE4 carriers. *Nutrients*, 13(4), 1362. <https://doi.org/10.3390/nu13041362>
- Shephard, A. M., & Ledón-Rettig, C. C. (2025). A novel carnivorous diet reduces brain telomere length. *Biology Letters*, 21(2), 20240593. <https://doi.org/10.1098/rsbl.2024.0593>
- Shephard, A. M., Rohner, P. T., & Ledón-Rettig, C. C. (2026). Cryptic genetic variation revealed by diet uncovers a trade-off between growth and telomere length. *Evolution*, 80(6), 1357–1364. <https://doi.org/10.1093/evolut/qpag042>
- Williams, G. C. (1957). Pleiotropy, natural selection, and the evolution of senescence. *Evolution*, 11(4), 398–411. <https://doi.org/10.1111/j.1558-5646.1957.tb02911.x>

Received March 19, 2026; accepted April 22, 2026

Associate Editor: Kati Moore; Handling Editor: Jason Wolf

© The Author(s) 2026. Published by Oxford University Press on behalf of The Society for the Study of Evolution (SSE).

All rights reserved. For commercial re-use, please contact reprints@oup.com for reprints and translation rights for reprints. All other permissions can be obtained through our RightsLink service via the Permissions link on the article page on our site-for further information please contact journals.permissions@oup.com