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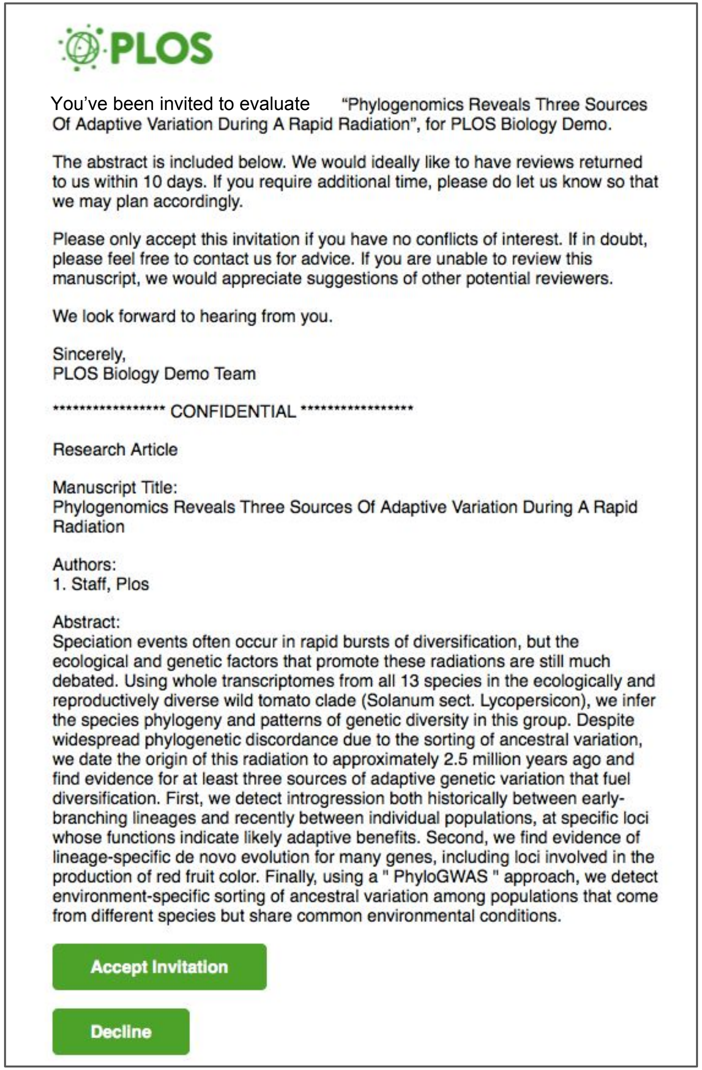

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PLOS Biology Demo Team

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Research Article

Manuscript Title:
Phylogenomics Reveals Three Sources Of Adaptive Variation During A Rapid Radiation

Authors:
1. Staff, Plos

Abstract:
Speciation events often occur in rapid bursts of diversification, but the ecological and genetic factors that promote these radiations are still much debated. Using whole transcriptomes from all 13 species in the ecologically and reproductively diverse wild tomato clade (Solanum sect. Lycopersicon), we infer the species phylogeny and patterns of genetic diversity in this group. Despite widespread phylogenetic discordance due to the sorting of ancestral variation, we date the origin of this radiation to approximately 2.5 million years ago and find evidence for at least three sources of adaptive genetic variation that fuel diversification. First, we detect introgression both historically between early-branching lineages and recently between individual populations, at specific loci whose functions indicate likely adaptive benefits. Second, we find evidence of lineage-specific de novo evolution for many genes, including loci involved in the production of red fruit color. Finally, using a " PhyloGWAS " approach, we detect environment-specific sorting of ancestral variation among populations that come from different species but share common environmental conditions.

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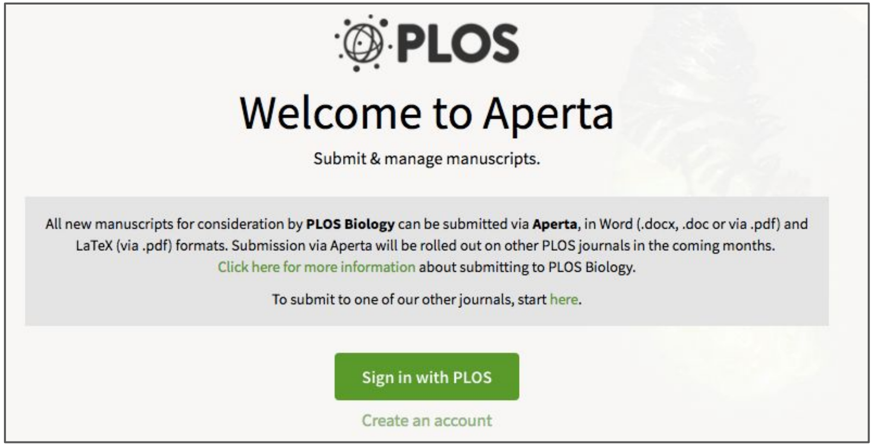

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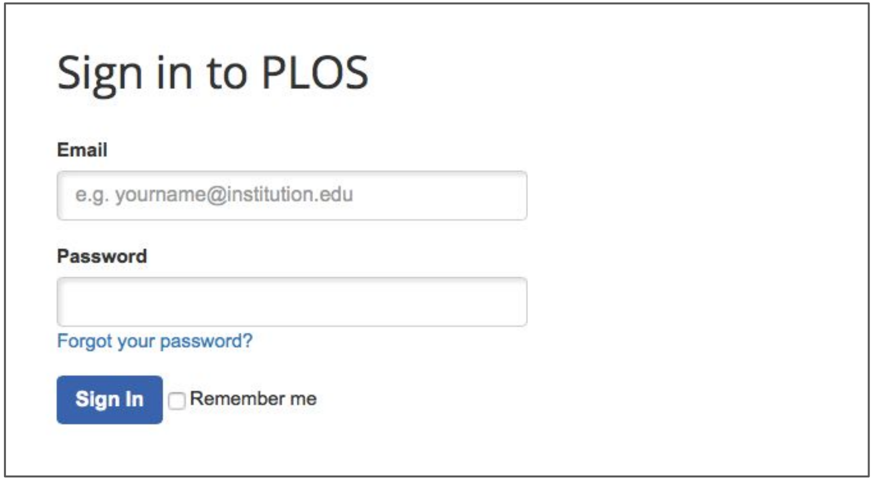

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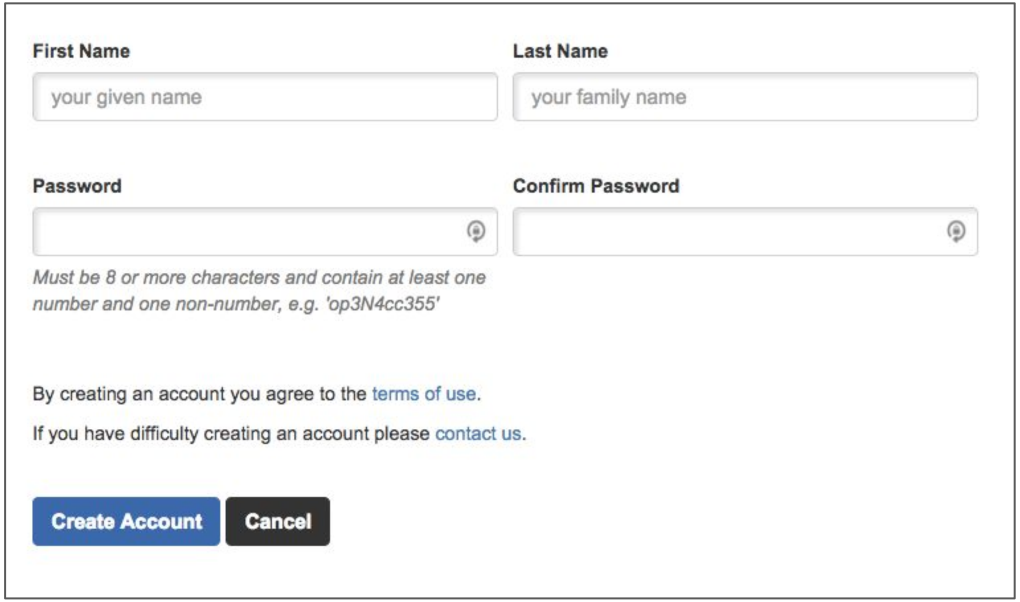

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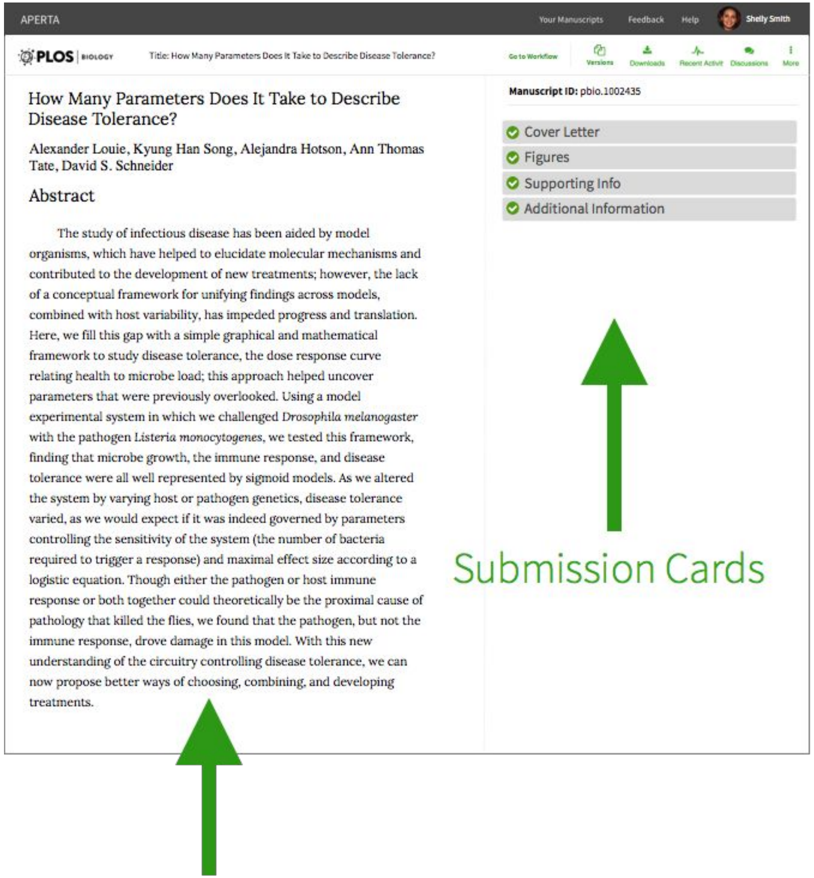

Title: How Many Parameters Does It Take to Describe Disease Tolerance?

**Manuscript ID:** pbio.1002435

- ✓ Cover Letter
- ✓ Figures
- ✓ Supporting Info
- ✓ Additional Information

How Many Parameters Does It Take to Describe Disease Tolerance?

Alexander Louie, Kyung Han Song, Alejandra Hotson, Ann Thomas Tate, David S. Schneider

Abstract

The study of infectious disease has been aided by model organisms, which have helped to elucidate molecular mechanisms and contributed to the development of new treatments; however, the lack of a conceptual framework for unifying findings across models, combined with host variability, has impeded progress and translation. Here, we fill this gap with a simple graphical and mathematical framework to study disease tolerance, the dose response curve relating health to microbe load; this approach helped uncover parameters that were previously overlooked. Using a model experimental system in which we challenged *Drosophila melanogaster* with the pathogen *Listeria monocytogenes*, we tested this framework, finding that microbe growth, the immune response, and disease tolerance were all well represented by sigmoid models. As we altered the system by varying host or pathogen genetics, disease tolerance varied, as we would expect if it was indeed governed by parameters controlling the sensitivity of the system (the number of bacteria required to trigger a response) and maximal effect size according to a logistic equation. Though either the pathogen or host immune response or both together could theoretically be the proximal cause of pathology that killed the flies, we found that the pathogen, but not the immune response, drove damage in this model. With this new understanding of the circuitry controlling disease tolerance, we can now propose better ways of choosing, combining, and developing treatments.

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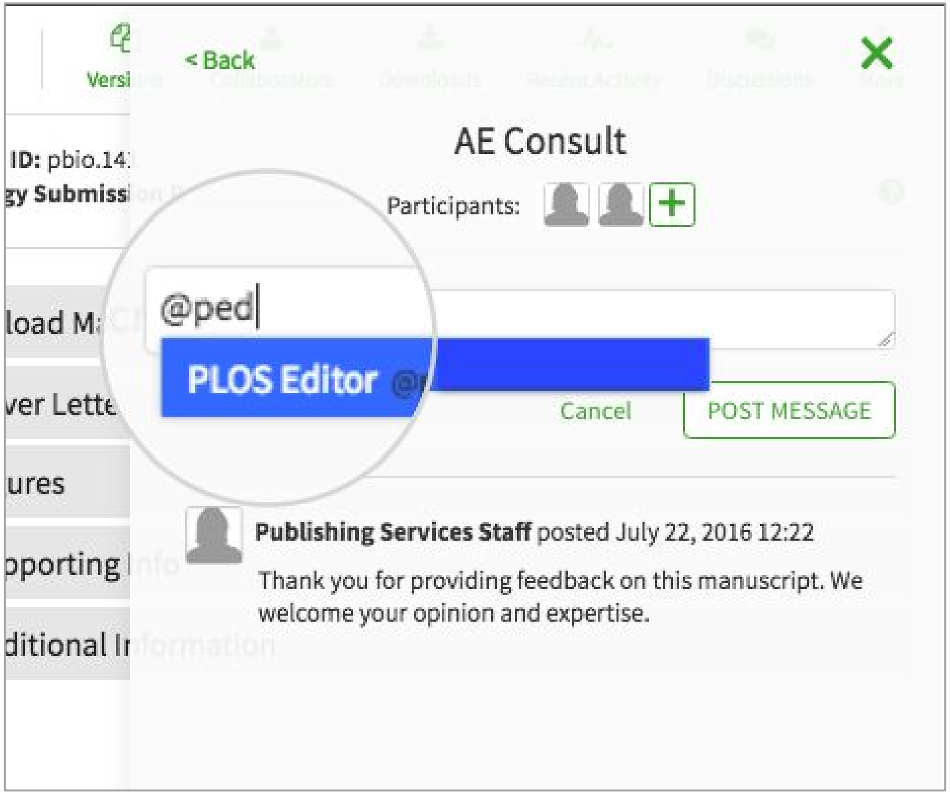

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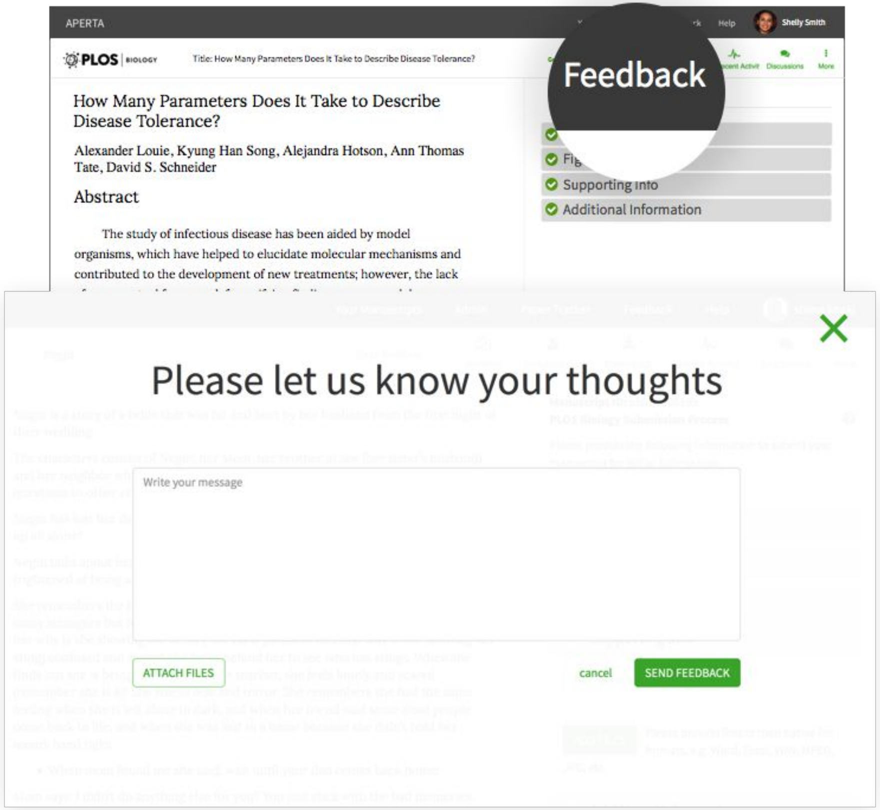

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