Retraction

Retraction Notice to: (p)ppGpp Controls Bacterial Persistence by Stochastic Induction of Toxin-Antitoxin Activity

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In this article, we reported that wild-type E. coli K-12 becomes persistent by stochastically switching to a mode of slow growth via a signaling pathway comprising the second messenger (p)ppGpp, polyphosphate, protease Lon, and toxin-antitoxin (TA) modules. In performing a series of new experiments, we discovered widespread infection of E. coli mutant strains used in the paper with bacteriophage ϕ 80 and found several of the reported phenotypes to be artifacts of lysogenization with the bacteriophage. The contribution of polyphosphate and TA modules to bacterial persistence and the connection of the key components in a single pathway in persister formation, which are major conclusions of the paper, are unfortunately no longer supported. The following data were affected by the lysogenic bacteriophage: Figure 1 (panels A and B), Figure 2 (panels A and C), Figure S1 (panels A, D, and E), and Figure S2 (panels C, D, and E). For these figures, ϕ 80 infection was confirmed for strains Δ 10TA, Δ ppkx, and Δ relAspoT. We believe that the most appropriate course of action is to retract the paper. We offer our sincerest apologies to the scientific community for these errors and for any inconvenience they may have caused. Please see Harms et al., mBio 8: e01964-17 for more information and to obtain reconstructed strains.

