Title:	Revisiting long-chain fatty acid metabolism in Escherichia coli: integration with stress responses
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Abstract:	Long-chain fatty acids (LCFAs) are a tremendous source of metabolic energy, an essential component of membranes, and important effector molecules that regulate a myriad of cellular processes. As an energy-rich nutrient source, the role of LCFAs in promoting bacterial survival and infectivity is well appreciated. LCFA degradation generates a large number of reduced cofactors that may confer redox stress; therefore, it is imperative to understand how bacteria deal with this paradoxical situation. Although the LCFA utilization pathway has been studied in great detail, especially in Escherichia coli, where the earliest studies date back to the 1960s, the interconnection of LCFA degradation with bacterial stress responses remained largely unexplored. Recent work in E. coli shows that LCFA degradation induces oxidative stress and also impedes oxidative protein folding. Importantly, both issues arise due to the insufficiency of ubiquinone, a lipid-soluble electron carrier in the electron transport chain. However, to maintain redox homeostasis, bacteria induce sophisticated cellular responses. Here, we review these findings in light of our current knowledge of the LCFA metabolic pathway, metabolism-induced oxidative stress, the process of oxidative protein folding, and stress combat mechanisms. We discuss probable mechanisms for the activation of defense players during LCFA metabolism and the likely feedback imparted by them. We suggest that besides defending against intrinsic stresses, LCFA-mediated upregulation of stress response pathways primes bacteria to adapt to harsh external environments. Collectively, the interplay between LCFA metabolism and stress responses is likely an important factor that underlies the success of LCFA-utilizing bacteria in the host.
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