Title:	Electrostatic lipid–protein interactions sequester the curli amyloid fold on the lipopolysaccharide membrane surface
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Abstract:	Curli is a functional amyloid protein in the extracellular matrix of enteric Gram-negative bacteria. Curli is assembled at the cell surface and consists of CsgA, the major subunit of curli, and a membrane-associated nucleator protein, CsgB. Oligomeric intermediates that accumulate during the lag phase of amyloidogenesis are generally toxic, but the underlying mechanism by which bacterial cells overcome this toxicity during curli assembly at the surface remains elusive. Here, we elucidated the mechanism of curli amyloidogenesis and provide molecular insights into the strategy by which bacteria can potentially bypass the detrimental consequences of toxic amyloid intermediates. Using a diverse range of biochemical and biophysical tools involving circular dichroism, fluorescence, Raman spectroscopy, and atomic force microscopy imaging, we characterized the molecular basis of the interaction of CsgB with a membrane-mimetic anionic surfactant as well as with lipopolysaccharide (LPS) constituting the outer leaflet of Gram-negative bacteria. Aggregation studies revealed that the electrostatic interaction of the positively charged C-terminal region of the protein with a negatively charged head group of surfactant/LPS promotes a protein–protein interaction that results in facile amyloid formation without a detectable lag phase. We also show that CsgB, in the presence of surfactant/LPS, accelerates the fibrillation rate of CsgA by circumventing the lag phase during nucleation. Our findings suggest that the electrostatic interactions between lipid and protein molecules play a pivotal role in efficiently sequestering the amyloid fold of curli on the membrane surface without significant accumulation of toxic oligomeric intermediates.
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