Title:	Human Fibrinogen Inhibits Amyloid Assembly of Biofilm-Forming CsgA
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Abstract:	Curli is a biofilm-forming amyloid that is expressed on the surface of Gram-negative enteric bacteria such as Escherichia coli and Salmonella spp. Curli is primarily composed of the major structural subunit, CsgA, and interacts with a wide range of human proteins that contribute to bacterial virulence. The adsorption of curli onto the contact-phase proteins and fibrinogen results in a hypocoagulatory state. Using an array of biochemical and biophysical tools, we elucidated the molecular mechanism of interaction between human fibrinogen and CsgA. Our results revealed that a substoichiometric concentration of fibrinogen delays the onset of CsgA aggregation by inhibiting the early events of CsgA assembly. The presence of fibrinogen prevents the maturation of CsgA into fibrils and maintains the soluble state of CsgA. We also demonstrate that fibrinogen interacts more effectively with the disordered conformational state of CsgA than with the ordered β-rich state. Our study suggested that fibrinogen is an anti-curli protein and that the interplay of CsgA and fibrinogen might be a host defense mechanism against curli biogenesis, biofilm formation, bacterial colonization, and infection.
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