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*Fracture model of bacterial growth*

The peptidoglycan (PG) cell wall is a key determinant of shape in "rod-shaped bacteria". However the mechanism guiding the growth of this elastic network of cross-linked PG (called sacculus) that maintains the integrity and shape of the rod-shaped cell remains a puzzle. We propose that the known anisotropic elasticity and anisotropic loading, due to the shape and turgor pressure, of the sacculus is sufficient to direct small gaps in the sacculus to elongate around the cell, and that subsequent repair leads to longitudinal growth without radial growth. We computationally show in our "Anisotropically stressed anisotropic elasticity model" small gaps can extend stably in the circumferential direction for the known elasticity of the sacculus. We suggest that patches of cytoskeletal protein, found recently, that propagate circumferentially in the cell wall are associated with these gaps and are steered with this common mechanism. We also show that small changes of elastic properties can in fact lead to bi-stable propagation of gaps, both longitudinal and circumferential, that can explain the bi-stability in patch movement observed in mutants.