Regulation of the motile-to-sessile switch in *Agrobacterium tumefaciens*

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As with many flagellated bacteria, *Agrobacterium tumefaciens* transitions between an actively motile phase and an immobilized, or sessile existence. This lifestyle switch underlies formation of *A. tumefaciens* biofilms and interactions with plant hosts, including pathogenesis. *A. tumefaciens* forms complex biofilms on plant tissues, and similar structures can be recapitulated on abiotic surfaces. On a wide variety of surfaces, bacteria attach via a single pole, and this requires production and localization of a polar adhesin called the unipolar polysaccharide (UPP). Flagellar propulsion must also be modulated during the transition to surface attachment. These and other processes are influenced by a complex network of regulatory factors and environmental conditions. Integrated at the center of this network is the bacterial second messenger cyclic diguanosine monophosphate (c-di-GMP). *A. tumefaciens* encodes >30 annotated putative diguanylate cyclases (DGCs) that can drive c-di-GMP synthesis, a portion of which also have phosphodiesterase (PDE) domains that can degrade it. Only a fraction of DGC/PDE proteins have a significant impact on UPP production and other attachment processes. Among these is DcpA, an enzyme with distinct DGC and PDE activities that has a marked influence on these processes. DcpA activity is tightly regulated, requiring unusual metabolites called monapterins, in conjunction with several regulatory proteins. The monapterin(s) is required to maintain the bias of DcpA towards PDE activity, thereby preventing inappropriate deployment of the UPP until cells productively interact with surfaces. Another level of regulatory control is imparted through the acid-responsive ChvG-ChvI two component system, and its periplasmic regulator ExoR. ExoR-ChvG-ChvI are a global regulatory pathway that paradoxically down-regulates motility and biofilm formation. ChvI-dependent motility inhibition is imparted through a novel gene called *mirA*.