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# Precedence for the Structural Role of Flagella in Biofilms

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Continuing the trend of impressive results using microscopy to decipher how biofilms form, Serra et al. recently discovered that flagella in biofilms serve a structural role (1). In *Escherichia coli* colonies on a relatively cold agar surface (28°C), Serra et al. found in some regions that flagella wrapped around cells and anchored them; hence, the flagella play a role in cementing cells together like polysaccharides, DNA, and protein. However, this new role for flagella is not unprecedented, since my colleagues and I showed in 2007 that 20 *E. coli* flagellum genes are regulated throughout biofilm formation in flow cells (2). Our manuscript was the first report of the importance of flagella in mature biofilms, and we deduced that flagella were “important throughout all stages of biofilm development from early attachment as well as in developed biofilms.”

It may be argued that Serra et al. studied the spatial organization of cells in a colony, whereas we used gene expression profiling in a temporal manner, or that there is a difference in the biofilms that were studied (flow cells at 37°C versus agar plates at 28°C), but the point remains the same: in mature biofilms, flagella were shown years ago to likely play an important role beyond that of motility to a surface to initiate biofilm formation since the genes for flagella were turned on in nonmotile biofilm cells. Hence, it is satisfying to see that one of those roles has now been discerned.

On a less important note about attribution in the same *mBio* paper, we used gene expression profiling to determine that a previously unstudied protein, YmgB, was related to biofilm formation and to acid resistance influenced by indole; we also determined how it functions at the atomic scale by determining its structure and renamed it AriR for regulator of acid resistance influenced by indole (3). Determination of its structure was important for determining that it acts as a regulator, since although it is only 5% identical to Hha (a protein that interacts with histone-like nucleoid structuring protein H-NS to regulate transcription), the structures of AriR and Hha are closely related, so the function of AriR as a regulator was not clear until a structure was determined (3). In Fig. 1 of the same *mBio* paper of Serra et al., the authors

continue to use the older name YmgB for this protein, which ignores the original contribution about its regulatory function, even though all of their follow-up studies confirm the role that AriR plays in acid resistance (and for which it was named). Also, recently Tschowri et al. authored a work related to AriR but did not mention our original work with this protein (4). Of course, follow-up papers add new mechanistic insight into what was originally discerned; for example, it is now more clear that AriR works through the Rcs system to regulate acid resistance. Therefore, although it is perhaps quixotic, one hopes eternally as a scientist that, as details emerge on important systems (arguably, acid resistance is one of the most important phenotypes of *E. coli*), the original research may be cited. Citing the original and transformative work does not detract from the current work and provides perspective (as well as sometimes a means of livelihood for the original authors).

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