Palladin Compensates for Branching Complex in Promoting Actin-Based Motility in *Listeria*

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Palladin is an actin binding protein which has important functions in cell motility both in normal cells during development or in wound healing and in metastatic cancers. The Beck lab has recently shown that palladin promotes actin polymerization, while also stabilizing and crosslinking actin filaments. In this work we take advantage of the fact that the intracellular pathogen *Listeria monocytogenes* hijacks host cell actin for motility by forming tails of actively polymerizing actin. These actin comet tails are a useful tool to study the regulation of actin dynamics. We first demonstrated that palladin localized to bacterial cell entry and motility. When palladin expression was reduced, the comet tails of *Listeria* were shortened and motility was slowed. Previous work has suggested that palladin may be able to form new branches of actin during polymerization, so we next sought to determine whether palladin can replace the functions of Arp2/3, the only protein complex currently known to create new branches on actin. We found that motility was maintained and comet tails were restored in cells treated with an Arp2/3 inhibitor when palladin was overexpressed. Finally, we used purified proteins to clearly demonstrate that the actin comet tails can be maintained by palladin in the absence of Arp2/3. Collectively, our results reveal that palladin is needed for the structural integrity of comet tails as its depletion causes dramatic changes to comet tail organization during bacterial actin based-motility. Notably this is the first study to identify a protein that can functionally replace the Arp2/3 complex.