The HrpB2 - HrcU interaction in the context of Type III secretion and pathogenesis in *Xanthomonas axonopodis* pathovar citri

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Xanthomonas axonopodis pv. citri (*Xac*), the causal agent of the citrus canker, uses a type III secretion system (TTSS) to inject virulence factors into host cells. We have previously shown that HrcU, a conserved inner membrane T3SS subunit, interacts with HrpB2. Here, we demonstrate that a fragment corresponding to the cytosollic C-terminal domain of HrcU ($HrcU_{207-357}$) suffers cleavage in a conserved N264-P265-T266-H267 sequence that corresponds to the autolytic site of the paralog FlhB of the bacterial flagellum. We show that a fragment in which this sequence is mutated ($HrcU_{207-357AAAH}$) is not cleaved and retains the ability to interact with HrpB2 and that the interaction site is localized to within residues 277-357 of HrcU. We show that HrpB2 is secreted and that *Xac* strains with non-polar HrpB2 and HrcU deletions lost the ability to elicit canker disease. We are currently testing the hypothesis that the cleavage of the C-terminal domain of HrcU protein may be an important step in the orderly assembly of the TTSS and/or a prerequisite for the subsequent secretion of TTSS pilus subunits and effector molecules.