Poster Number

11 **Molecular basis of bacteriolysin-induced degradation of SUMO E2 Ubc9**

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SUMOylation is a reversible protein post-translational modification indispensable for viability in all eukaryotes, which is a sequential catalytic cascade involves E1, E2 and E3 enzymes. Modulation of SUMOylation has emerged as a strategy exploited by many pathogens during infection. Cholesterol-dependent cytolysin (CDC) is a large family of pore-forming toxins that commonly associated with bacterial pathogenesis. Pore formation on cell membrane is thought to be the major functions of CDCs, while increasing number of studies showed that CDCs can also modulate diverse host post-translational modifications during infection, including SUMOylation. Three CDCs, LLO, PLY and PFO were found to trigger a proteasome-independent degradation of SUMO E2 conjugating enzyme Ubc9, leading to a global decrease of SUMOylation, which partially linked to a yet-to–be identified aspartyl protease. However, the underlying mechanism of the LLO-induced Ubc9 degradation is unknown.
Here we show that recombinant LLO, PLY and two other CDCs, SLO from S. pyogenes and SLY from S. Suis, all possessed inhibitory effect on both Ubc9 stability and SUMOylation when applied to both HeLa cells and THP-1 cells. Results from cell fractionation and immunostaining suggested that the degradation event was mainly occurred within nuclei. By using different Ubc9 mutants defective in covalent or non-covalent SUMO binding, we demonstrated that interaction between SUMO and Ubc9 is not critical for CDC-induced Ubc9 degradation. On the other hand, we found that phosphorylation of Ubc9 was involved in this degradation process. CDC-induced Ubc9 degradation was enhanced when pre-incubating cells with phosphatase inhibitors. Furthermore, degradation of Ubc9S71A mutant was suppressed upon LLO treatments. These results suggested that CDCs share similar strategies during bacterial infection and phosphorylation of Ubc9 may be one of the key steps during CDC-induced degradation.

12 **Mcp1p tracks microtubule plus ends to destabilize microtubules at cell tips**

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Microtubule plus ends are dynamically regulated by a wide variety of proteins for performing diverse cellular functions. Here, we show that the fission yeast Schizosaccharomyces pombe uncharacterized protein mcp1p is a microtubule plus-end tracking protein which depends on the kinesin-8 klp6p for transporting along microtubules towards microtubule plus ends. In the absence of mcp1p, microtubule catastrophe and rescue frequencies decrease, leading to an increased dwell time of microtubule plus ends at cell tips. Thus, these findings suggest that mcp1p may synergize with klp6p at microtubule plus-ends to destabilize microtubules.