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TRACKING THE LOCALIZATION OF CpsA, AN ESSENTIAL VIRULENCE FACTOR OF *MYCOBACTERIUM TUBERCULOSIS*

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Tuberculosis, an infectious disease that affects one third of the world's population, is caused by the pathogenic bacterial species *Mycobacterium tuberculosis* (*Mtb*), which grows within macrophages. Recent studies have identified that the membrane protein CpsA plays an essential role in *Mtb*'s ability to evade degradation by the host cell. Successful clearance of *Mtb* from infected cells requires triggering of the lysosomal trafficking pathway, LAP, that depends on reactive oxygen species generated by NADPH oxidase. CpsA, however, blocks this response by inhibiting NADPH Oxidase and thus LAP, enabling *Mtb* to evade the immune response. Currently, however, the localization of CpsA within the cell is unknown.

To clarify the mechanism of action of CpsA, we must pinpoint where it is located within the bacterial cell. To accomplish this, we will engineer plasmid-derived CpsA-GFP and CpsA-mCherry fusion proteins, introduce them into competent *Mycobacterium smegmatis* cells, and localize the encoded proteins *in vivo*. This approach will uncover the precise intracellular location of CpsA, clarifying whether it localizes uniformly throughout the membrane, or is localized to a specific region of the cell. Furthermore, we will be able to determine whether the protein is present within the host cytosol during infection.

Because CpsA alters host cellular trafficking in order to evade the immune response, advancements in our knowledge about the functional properties of this protein should enhance our understanding of host innate immunity.