**Brief Idea**

**Title:** Effect of PH on Copper hypertolarence: How does the Copper exporters of *Staphylococcus aureus* are maintaining its functionality within low PH of Phagosome?

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We know that, when any bacterium entered into our body as per the innate immunity phagocytic cells majorly macrophages activated and use copper into phagosome to kill the phagocytized bacterium [1]. Unfortunately most of the bacterium is capable to escaping this due to presence of some genes which code for copper exporters on cell membrane, similarly for *Staphylococcus aureus* where it code for P1B-1-type ATPase and P1B-3-type ATPase which helped to lower the cytoplasmic copper concentration and enhanced survival [1]. But my question is that how do the ion channels (copper exporters) are functioning well inside of phagosome? The pH of early and late phagosome and phagolysosome are respectively 6.1–6.5; 5.5–6.0; and 5–5.5 [2] and we know that different pH can alter protein shape, so what is the scenario for those exporters and how does its functioning well? The peptidoglycan barrier can be degraded by Lysozyme [2], so it is possible that the low pH can hamper the bacterium copper efflux efficiency or it is may also possible that they might have any protective mechanism. It is not measured yet so in different pH it can be done.

**References:**