Modulating The Type I Interferon Response During Intracellular Bacterial Infection

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Abstract
In our previous work, we have found that glutathione deficiency in Type 2 diabetic individuals predispose them to developing severe melioidosis because they have a defective IL-12 response to intracellular bacterial infection. In our search for ways to overcome the IL-12 defect, we discover a failed cancer drug that could enhance IL-12 production during bacterial infection through its stimulatory action on Type I Interferon. In this talk, I will discuss our progress on delineating the novel mechanisms of Type I IFN induction by the drug, as well as by the bacterium, Burkholderia pseudomallei.

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