**Reactive Intermediate Deaminase A (RidA) – A Bacterial Metabolite Detoxifying Protein from an Opportunistic Pathogen, *Streptococcus sanguinis***

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Amino acid catabolism (breakdown) is an essential metabolic process in all organisms which has important functions ranging from biosynthesis of cellular building blocks to energy production. However, breakdown of certain amino acids can lead to the formation of toxic intermediates that are detrimental to many cellular processes. In bacteria, breakdown of amino acids leads to the formation of a toxic intermediate, 2-aminoacrylate (2AA) which can inhibit essential celluar functions. In all organisms, including bacteria, an enzyme designated as reactive intermediate deaminases (Rid) catalyzes the conversion of 2AA into non-toxic pyruvate. RidA is a well-characterized member of this family of proteins, and its importance in bacterial pathogens led us to investigate RidA's role in *Streptococcus sanguinis*. *S. sanguinis* is an opportunistic pathogen and one of the leading causes of subacute infective endocarditis. Computational analysis was used to identify the presence of a RidA homolog, SSA_0809, in *S. sanguinis*, and the ability of SSA_0809 to detoxify 2AA to pyruvate was demonstrated using a well-established biochemical assay. Computational and biochemical analysis led us to hypothesize that SSA_0809, henceforth SsRidA, is a RidA homolog in *S. sanguinis* capable of deaminating toxic 2AA. Although we were able to establish that SsRidA is capable of deaminase activity, the mechanism by which SsRidA accomplishes this remains unclear. To better understand the catalytic mechanism of SsRidA, we have solved the structure of SsRidA in multiple conformations using X-ray crystallography technique. Currently, *ridA* gene deletions studies are underway to understand the physiological role of RidA in *S. sanguinis*. Understanding biochemical properties, structure, and the functional role of SsRidA, will help us in delineating the metabolic pathways important for *Streptococcus sanguinis* survival during endocardial infection.