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MRS2: A Prospective Drug Target in Gastric Cancer:

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Mitochondrial-RNA-splicing-protein-2 (MRS2) is a magnesium (Mg^{2+}) protein channel localized within mitochondria. In gastric cancer (GC), the over-expression of MRS2 in malignant cells, and the consequential increase in Mg^{2+} uptake into the mitochondria, is believed to offer immunity against cancer therapeutics, thereby rendering many existing GC treatments less efficacious, or, entirely obsolete. According to the International Agency for Research, GC is the fifth most common cancer worldwide and the third deadliest, however, despite the prospective link between MRS2 and GC, the molecular mechanisms which underlie MRS2 function remain unknown. My research aims to reveal the fundamental basis for MRS2 action as the first step towards understanding its structural and functional mechanisms within GC. To date, I have (a) discovered a novel mechanism for MRS2 auto-regulation and (b) created a mutant within MRS2, which abolishes protein channel structure and stability—both ultimately exhibiting applicability to future pharmacological therapeutic development for GC.