

## Structure Prediction and validation of mutant kir6.2 genes

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### ABSTRACT

Diabetes is one of the leading causes of morbidity and mortality, consuming a significant proportion of public health spending. Several receptors (insulin-like growth factor receptor, glucose transporter, and kir6.2 and their associated signaling pathways have been elucidated and are involved in glucose regulation and diabetes. Kir6.2, a major subunit of the ATP-sensitive K<sup>+</sup> channel, an inward-rectifying potassium ion channel, is an integral membrane protein that allows K<sup>+</sup> to flow from the outside of the cell to the inside, which is controlled by G-proteins associated with sulfonylurea receptor (SUR), to constitute the ATP-sensitive K<sup>+</sup> channel. Ten possible mutations affecting the regular mechanism of kir6.2 have been identified as probable causes of type 1 diabetes. Due to the unavailability of the crystal structure of kir6.2 protein, an attempt was made here to predict both the secondary and tertiary structures using *in silico* approach.