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Title of the Thesis: Study on Sorcin Mediated Pathway of Multidrug Resistance in Gastric Carcinoma

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ABSTRACT

Gastric cancer (GC) remains a major global health challenge, with high mortality rates despite advancements in chemotherapy. Multidrug resistance (MDR) is a key factor contributing to treatment failure, primarily driven by drug efflux transporters like P-glycoprotein (P-gP) and other molecular mechanisms. Sorcin, a soluble calcium-binding protein, has emerged as an important regulator of MDR in various cancers, including GC. The present study focuses on the role of Sorcin as a biomarker for identifying the MDR phenotype in the patients and to explore its underpinning interplay with ERK signalling and regulation of P-gP in MDR.

A prospective study was conducted on 78 advanced GC patients to analyze the correlation of Sorcin expression, with clinicopathological features, and overall survival. Molecular experiments include qRT-PCR, IHC, WB, CO-IP, cell culture, development of resistant cell lines, MTT assay, rhodamine123 accumulation assay and siRNA transfection to assess the role of Sorcin in MDR. Findings revealed that Sorcin was significantly upregulated in GC tissues, particularly in Signet Ring Cell Carcinoma (SRCC), and was associated with aggressive tumor characteristics. Elevated Sorcin expression correlated with chemoresistance, poor prognosis and shorter survival, suggesting it as a potential predictor of MDR phenotype. Protein-protein interaction studies confirmed association of Sorcin with P-gP and ERK1/2, indicating its mechanistic interplay in chemoresistance. Notably, Sorcin silencing led to a significant downregulation of P-gP and ERK1/2, underscoring its potential as a therapeutic target to overcome MDR.

Overall, the outcomes of the study highlights Sorcin as a key biomarker for MDR in GC and underscores its potential as a novel therapeutic target. Future research focusing on Sorcin-targeted therapies may improve treatment outcomes for GC patients.

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