A Preliminary Screening on Cytosolic Free Calcium and *Orai1* Expression in Stromal Interaction Molecule 1 Silencing Acute Myeloid Leukemia Cells

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Stromal interaction molecule (*STIM1*) is the main regulator for basal cytoplasmic calcium levels via store-operated calcium entry (SOCE). SOCE dysregulation has been reported to be involved in numerous cancers development and metastasis. However, the association of *STIM1* and SOCE in acute myeloid leukemia (AML) pathogenesis remains unclear. The present work investigates the SOCE activities via *Orai1* expression and cytosolic free calcium level assessment in *STIM1* silencing AML cells such as THP-1 and Kasumi-1 cells. *STIM1* and *Orai1* expressions were profiled through real-time reverse transcription PCR (qRT-PCR) platform. The changes in the cytosolic free calcium levels were quantified using the intracellular calcium indicator fura-2 acetoxymethyl ester (Fura-2AM). This study revealed that *STIM1* and *Orai1* were expressed in both AML cell lines. *STIM1* expression was observed more than 2.5-fold higher in THP-1 cells compared to Kasumi-1 cells, which could be related to the aggressive phenotype of THP-1 cells with M5 subtype rather than Kasumi-1 cells with M2 subtype. *STIM1* knockdown has shown a regularity effect on SOCE activities by suppression of *Orai1* and decreases the calcium influx profile, especially in THP-1 cells. This observation supports the importance of *STIM1* in the regulation of calcium homeostasis in AML through SOCE. Findings from this study suggest that *STIM1* may serve as a potential therapeutic target for AML, especially for aggressive FAB subtypes. However, further comprehensive work is needed to support these findings.

Keywords: *STIM1*, *Orai1*, calcium, SOCE, AML.

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