Deregulation of calcium homeostasis in Bcr-Abl-dependent chronic myeloid leukemia

SUPPLEMENTARY MATERIALS

Supplementary Figure 1: Impact of Orai1 inhibitor, GSK-7975A in calcium entries. (A) Store-operated Ca\(^{2+}\) entry in 32d-p210 cell line in control condition (dotted line) and in presence of 1 \(\mu\)M GSK-7975A (dark line). The ER depletion was obtained by perfusion of 15 \(\mu\)M CPA (SERCA inhibitor) in 0 mM Ca\(^{2+}\) solution, which allowed SOCE recording in presence of 1.8 mM Ca\(^{2+}\) prior to a 0 mM Ca\(^{2+}\) buffer incubation. (B) SOCE measurements (initial slope of intracellular Ca\(^{2+}\) rise) in p210-32d cells in control condition or after incubation of 10 \(\mu\)M YM 58483 or 1 \(\mu\)M GSK-7975A for 30 min. This experiment was done also with 10 \(\mu\)M GSK-7975A but no calcium entries could be measured showing a total SOCE inhibition in this experimental condition. (C) Quantification of half-time response of thrombin-induced Ca\(^{2+}\) transient with or without pre-incubation with 10 \(\mu\)M GSK-7975A during 30 minutes and incubated with 1 U/ml thrombin in 1.8 mM Ca\(^{2+}\) buffer in WT and 32d-p210 cells. The duration of the thrombin-induced Ca\(^{2+}\) transient is more dependent on SOCE in WT cells than in 32d-p210 cells. Bar graphs represent mean rates ± SEM. \(^*\) \(P < 0.01\); \(^{**}\) \(P < 0.005\); \(^{***}\) \(P < 0.001\).