Store-operated Ca\(^{2+}\) entry (SOCE) is activated by endoplasmic reticulum Ca\(^{2+}\) store depletion and is a major Ca\(^{2+}\) influx pathway in non-excitable cells. It is widely accepted that Orai1 protein in the plasma membrane (PM) is the channel that mediates SOCE in many cell types. Orai1 is activated when STIM1, an ER transmembrane protein, senses a decrease in the ER Ca\(^{2+}\) concentration and redistributes to ER-PM junctions, where it recruits Orai1 in coincident puncta. We have recently shown that at rest Orai1 actively recycles between an endosomal compartment and the cell membrane in Xenopus laevis oocytes and that ER Ca\(^{2+}\) store depletion translocates most of the Orai1 to the PM. We have also shown that during oocyte maturation Orai1 is internalized resulting in the absence of SOCE in the Xenopus egg. In mammalian cells, mitosis is the only known physiological situation where SOCE is inhibited but the underlying mechanism is not fully understood. In this study we investigated the trafficking of Orai1 in CHO cells that express a tagged Orai1. We also looked at the distribution of Orai1 during mitosis. We show that at steady state about 40% of the total Orai1 pool is at the PM whilst the remaining 60% localizes intracellularly, suggesting that Orai1 constitutively recycles between the two compartments. After the depletion of the ER Ca\(^{2+}\) stores the distribution of Orai1 shifts drastically with now most of the protein localized at the PM. Moreover, we show that the rate of Orai1 exocytosis is 3 fold faster after the depletion of the ER Ca\(^{2+}\) stores. During mitosis the total expression of Orai1 is reduced and the fraction of plasma membrane Orai1 tends to be less than in interphase cells. A fraction of the mitotic cells show no Orai1 expression at all. Interestingly, the reduction of Orai1 expression is reversed by inhibiting the proteasome complex, suggesting that Orai1 is degraded during mitosis.