The regulation of ionic transport in plants is essential because it establishes the key physicochemical parameters for cell function. Under abiotic stress, the intracellular levels of pH, potassium (K\textsuperscript{+}) and toxic cations, such as Na\textsuperscript{+}, change and this affect multiple cellular systems. Consequently, the knowledge of the molecular mechanism that regulates the ionic transport is fundamental and provides opportunities to use plants to our benefit [1]. The plant cells use calcium-signaling pathways to activate certain ion channels providing the correct response to a particular stress situation. As the most abundant cation in a living plant cell, K\textsuperscript{+} is an essential ion for processes of growth, development, maintenance of turgor pressure, and plasma membrane polarization [2]. Plants living under low K\textsuperscript{+} conditions often adapt their K\textsuperscript{+} uptake through the CBL-CIPK calcium-signaling pathway, that mobilizes K\textsuperscript{+} uptake in roots [3]. Under K\textsuperscript{+} deficiency, a CBL calcium sensor activates a CIPK kinase [3] that in turn phosphorilates and activate the K\textsuperscript{+} channel. When K\textsuperscript{+} levels are restored, a phosphatase dephosphorilates and inactivates the channel (Fig. 1) [5]. We have carried out structural studies with the kinase domain and its binding partner, the interacting region of the K\textsuperscript{+} channel, to understand at molecular level how K\textsuperscript{+} uptake is regulated under stress conditions.

**Figure 1.** A regulation model for the K\textsuperscript{+} channel. K\textsuperscript{+} channel is activated by interaction with CIPK-CBL. Phosphatase produces inactivation of the channel through their interaction.