Many questions and few answers about the role of ion channels and transporters in plastid structure (and function)

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Plastids harbor many key metabolic processes including photosynthesis which represents the basis for the carbon, nitrogen and sulphur autotrophy of plants, and finally also provides food and oxygen for other organisms. Several crucial plastid-located molecules (e.g., chlorophylls, heme prostethic groups), proteins (transcription factors, enzymes, etc.) contain metals as co-factors or require metal ions for their activity [1]. Similarly, the driving force of photosynthetic ATP production is a chemi-osmotic gradient (also termed proton-motive force) produced by protons that are accumulating in the lumen of the thylakoid membranes. At the same time, transportation of other ions is important to counterbalance unidirectional ion fluxes/accumulation and thus to maintain the ion homeostasis within the plastids and their compartments (like the thylakoid lumen). In spite of increasing knowledge about the molecular background of photosynthesis, data about channels and transporters located in the plastid envelope membrane and in the thylakoids are still rather scarce [2]. A better understanding of the latter and their roles in the stress adaptation of plastids and thus plants is especially important for potential breeding of plants with improved ion homeostasis and productivity under adverse environmental conditions.

After a general overview, this presentation will discuss in detail the role of two recently characterized chloride ion channels located in the thylakoid membrane [3,4]. Analyses of plastid structure in mutants indicated that (i) the lack of the CLCe channel resulted in a bow-like arrangement of the thylakoid system and in the appearance of a large thylakoid-free stromal region in the dark-adapted samples [3], while (ii) the lack of a voltage-dependent channel (VCCN1) resulted in bended granum shape structure in the light and slight alterations in granum diameter and repeat distance values, the latter revealed by small-angle neutron scattering measurements [4]. Ultrastructural data about the *clcexvccn1* double mutants lacking both channels will be also presented.

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