Proceedings of the Joint Meeting AGI-SIBV-SIGA
Assisi, Italy – 19/22 September, 2011
ISBN **978-88-904570-2-9**

Oral Communication Abstract – 2B.01

UNFOLDED PLASTID PRECURSORS IN THE CYTOSOL: TARGETING VS DEGRADATION BY QUALITY CONTROL

HWANG I.

Division of Integrative Biosciences and Biotechnology, POSTECH, Pohang, 790-784 (Korea)

A large number of nuclear encoded proteins are targeted to plastids posttranslationally as unfolded precursors. Thus, navigation of them through the cytoplasm is intrinsically dangerous to the cell viability because their accumulation in cytosol due to faulty import process can result in formation of cytotoxic and life threatening non-specific aggregates. Therefore cells must have a mechanism to monitor targeting of precursors to plastids and prevent cytosolic accumulation of precusors. Recently, we demonstrated Arabidopsis Hsp70-4, a member of heat shock protein 70 and its interacting E3 ligase AtCHIP play critical role in this process. Hsp70-4 and AtCHIP causes degradation of unimported or import defective precursors. The degradation was mediated by the ubiquitination/proteasome system. Hsp70-4 specifically recongizes and binds to sequence motifs present in the transit peptide of RbcS in vitro and in vivo and induces ubiquitination of unimported precursors. In *ppi2* mutants with a T-DNA inertion in *Toc159*, encoding the major import receptor, transcription of Hsp70-4 and AtCHIP was elevated. In ppi2 plants, endogenous RbcS and Cab precursors were degraded by the Hsp70-4 and AtCHIP-mediated UPS. In addition, in this pathway, BAG isoforms also are involved in positively or negatively depending on individual isoforms.