Potassium channel AtTPK5: An essential or redundant regulator of photosynthesis in Arabidopsis?

Behrad Shiki Baluch

Supervisor: Cornelia Spetea Wiklund

Co-supervisor: Patrik Milton Karlsson

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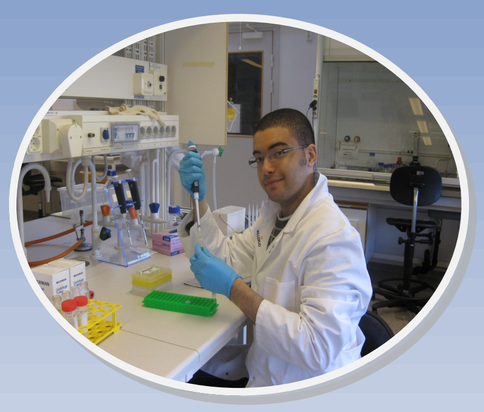


Background & aim

The plant cell contains a unique organelle known as the chloroplast which is reponsible for conducting photosynthesis. *Arabidopsis thaliana* was used for this project as a plant model to understand the functions of a specific protein known as Tandem Pore Potassium Channel 5 (AtTPK5), which is predicted to be located in the plant cell chloroplast. The aim of the present project were the following:

(1) to screen for homozygous (HM) mutants which would have fully lost or partially lost their ability to express AtTPK5.

(2) to clarify the physiological role of AtTPK5 in *Arabidopsis thaliana* by comparing plant fitness and photosynthesis in both mutant and wild-type.

Hypothesis

The experimental hypothesis for this project was that a knock-out or knock-down of AtTPK5 protein expression will negatively affect photosynthetic activity.

Methods

The *Arabidopsis thaliana* plants were grown in a hydroponic system and they consisted of two different T-DNA insertion mutants that were a knock-out (*tpk5-e*), a knock-down (*tpk5-UTR*) mutant and a wildtype. All plants belonged to the same ecotype, Columbia-0.

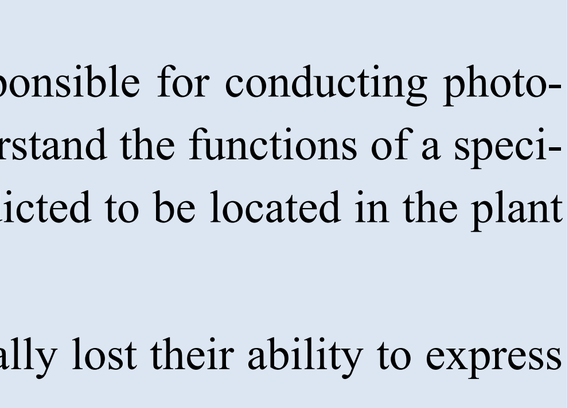
Primers were designed for the screening of HM mutants and the wildtype was used as a positive and negative control.

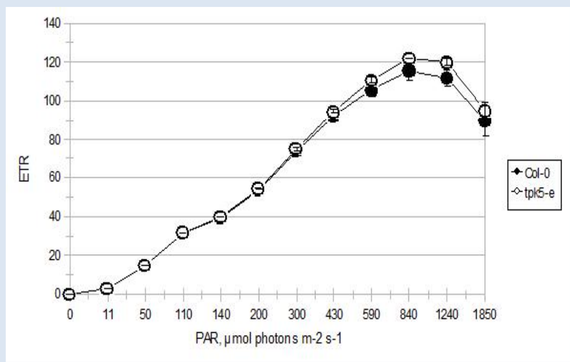
The plant photosynthetic activity was measured in 4 hour light-adapted plants as both chlorophyll fluorescence and oxygen (O_2)-rate and later calculated and presented as electron transport rate (ETR) and O_2-evolution in plants that were 54-56 and 69 days old respectively.

Arabidopsis thaliana

Results

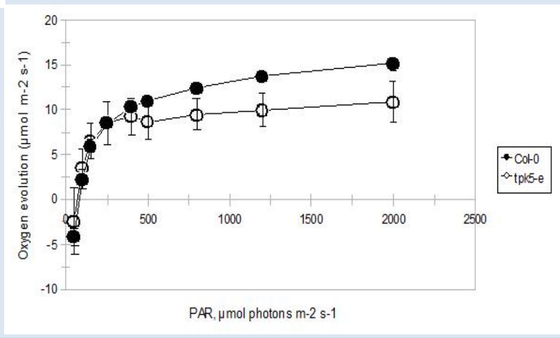
PCR screening results with the *tpk5-e* mutant and wild-type (Col-0). HM mutants were only found for the *tpk5-e* mutant during screenings.





No statistical significant difference in ETR activity when comparing the *tpk5-e* mutant with the wild-type.

O_2-evolution decreased in *tpk5-e* mutant but only during high light stress



Conclusions

The ETR-activity of the *tpk5-e* mutant plants proved to be unaffected by their AtTPK5 deficiency. The decreased O_2-evolution during high light exposure in light-adapted *tpk5-e* mutants indicate that the photosystem II (PSII) activity in the chloroplast is affected by the AtTPK5 deficiency.

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