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Investigation on post-translational regulation of HAK5 as a response to potassium recovery after starvation in *A.thaliana*

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1. はじめに

Potassium is an essential macronutrient for plants. Therefore, plants have developed different means to take up potassium from the soil. In *Arabidopsis thaliana*, 2 transporters are known: AKT1 being a channel protein and HAK5 a proton symporter. The expression of both proteins responds to low potassium concentrations but HAK5 is the main functional transporter below 10 µM. HAK5 is transcribed when plants suffer stress in low potassium conditions and is indicated to be post-translationally regulated when potassium concentration recovers.

2. 方法

In order to confirm the post-translational regulation of HAK5 in *A.thaliana* recovering from potassium deficiency, sGFP-fused HAK5 was introduced into *hak5-2* and fluorescence observation was carried. The observation was carried at time course of 2, 30, 60, 120 minutes. Translation inhibitor, cycloheximide (CHX) was applied to narrow the cause of any response to changes in potassium concentration.

3. 結果

As a result, it was observed that the fluorescence at the plasma membrane of sGFP-fused HAK5 decreased when shifted from low K (3 µM) to high K (1 mM). We have observed the same result with proAt5g43040 driven sGFP-fused HAK5 which takes into account the possibility of transcriptional or translational regulation, we have observed the same result with proAt5g43040 driven sGFP-fused HAK5. During which punctate structures and vacuole-like fluorescent structures were also observed. CHX treatment was carried giving similar trend in response to potassium concentration which ascertained that this decrease in fluorescence was not caused due to translational regulation.

4. 結論

Finally, this thesis shows that HAK5 undergoes post-translational regulation in response to potassium concentration when *A.thaliana* is shifted from a potassium deficient state to a potassium supplied state. Also, it suggests that this is a post-translational down-regulation and that HAK5 is directed to vacuolar degradation.