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【論文の内容の要旨】

Desiccation resistance is a strategy for organism could survive under low humidity environment. The physiological adaptations which affect the resistance to desiccation have been reported for many years, but the molecular basis involved in it remains elusive. Metabolism, especially respiration regulation, is considered to be important to desiccation resistance. In this study, I used *Drosophila* as a model organism, to reveal the close relationship between the metabolism and desiccation resistance. I have been analyzing phenotypic effects of mutations in genes encoding enzymes related to energy metabolism in *Drosophila*, and noticed that desiccation resistance was associated with the function of TCA cycle, which is a major pathway to produce energy. In particular, I found that desiccation resistance was significantly increased in flies with mutations in SCSα (Succinyl CoA synthetase alpha subunit), which catalyzes the succinyl coA into succinate in TCA cycle. Consistent with this result, fatbody-specific knockdown the expression level of *Scsα* also showed the desiccation resistance. And reduced metabolites in the downstream of succinyl coA and respiration rates suggested the mutation in *Scsα* indeed affected the metabolism. How is the desiccation resistance

regulated in *Scsα*? Addressing this question, physiological adaptions and metabolite analysis were initiated to learn about the mechanism. I found neither water contents nor water loss was responsible for the desiccation resistance in *Scsα*. Interestingly, *Scsα* mutants showed resistance to hydrogen peroxide (H_2O_2) which causes oxidative stress. Notably, decreased cyclic AMP (cAMP) concentration in *Scsα* mutant flies was detected using liquid chromatography–mass spectrometry (LC−MS/MS). *rutabaga*, which has defection in Ca2+/calmodulin-sensitive adenylate cyclase in cAMP signaling pathway, also exhibited the resistance to desiccation. To further confirm the mutation in *Scsα* is responsible for desiccation resistance, I employed the new technique-CRISPR/Cas9 system to generate the *Scsα* knockout mutant. Taken together, I demonstrated in *Drosophila* that reduction in SCSα function confers desiccation resistance, and decreased cAMP may responsible for the stress tolerance.