Characterizing a Novel *sgk-1* Gain-of-Function Mutation in *C. elegans*

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Regulation of cellular metabolism and lipid transport in *Caenorhabditis elegans* is necessary for growth, reproduction, and survival and is mediated in part by the conserved complex Rictor/mTORC2 via downstream phosphorylation of SGK-1 and Akt. Our lab previously identified a novel gain-of-function mutation in *sgk-1* that restores growth and vitellogenesis in a *rict-1* loss-of-function background. This mutation was found to demonstrate little difference in phenotype when expressed singly or in combination with a previously characterized *sgk-1* gain-of-function mutation. The novel mutation was also expressed in combination with a *pdk-1* gain-of-function mutant and in an F-box overexpression strain to further characterize its phenotypic effects. I found that expressing a gain-of-function mutation in *pdk-1* can further restore body size, but not vitellogenesis, of *sgk-1* mutants in a *rict-1* loss-of-function background. Furthermore, I found that the novel *sgk-1* mutation does not restore vitellogenesis in an F-box overexpression strain. These results suggest the need to determine if the *pdk-1* mutation acts through Akt signaling to restore body size and to identify the mechanism by which F-box suppresses vitellogenesis and growth phenotypes in *C. elegans.*