Inward Rectifier Potassium Channels Inhibit *C. elegans* Egg Laying and Locomotion

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Inward-rectifier K^+ (IRK) channels function to regulate membrane excitability in neurons and neuroendocrine cells. Three *C. elegans* genes, *irk-1*, *irk-2* and *irk-3*, are predicted to encode potassium channel subunits similar to mammalian Kir1-7, which form tetrameric inward-rectifier potassium channels that are activated by diverse intracellular signals, including G proteins, ATP and phosphoinositides. To study the function of *C. elegans* inward rectifier K^+ channels, we isolated deletion alleles of *irk-1*, *irk-2* and *irk-3*. *irk-1* mutants move faster than the wild type and lay early-stage eggs. *irk-1* deletion also suppresses the egg-laying defect caused by a gain-of-function mutation in the EGL-6 G protein-coupled receptor for FMRFamides that activate inhibitory G protein signaling in the HSN egg-laying motor neurons. We did not observe gross behavioral defects of *irk-2* and *irk-3* mutants.

An *irk-1::gfp* reporter transgene was expressed in a small number of neurons, including the HSNs. Expression of ChannelRhodopsin using the *irk-1* promoter caused animals to lay eggs and accelerate in response to a blue-light stimulus, indicating that *irk-1* functions to inhibit neurons that promote egg laying and locomotion. *Xenopus laevis* oocytes expressing *irk-1* showed inward-rectifying currents in high-potassium saline. We are currently testing whether signaling pathways known to modulate IRK channels in other organisms function to modulate IRK-1 *in vitro* and *in vivo*.

Poster
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