

Anion-Sensitive Fluorophore Identifies the *Drosophila* Swell-Activated Chloride Channel in a Genome-Wide RNA Interference Screen

Supporting Information

Figure S1.

The late activating component of S2R+ $I_{Clswell}$ remains despite overexpression of dBest1

W94C-gfp. (A) The late activating component of S2R+ $I_{Clswell}$ is isolated after dominant negative elimination of I_{dBest1} . Inset: Step protocol. **(B)** The late activating component of S2R+ $I_{Clswell}$ is sharply rectifying (ramp protocol; inset). Red trace is 320 mOSM solution, blue trace is 80 s after the 200 mOSM solution change. The late activating $I_{Clswell}$ develops after 36 s in 200 mOSM solution.

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(TIF)

Table S1.

Secondary screening identifies Best1 as the *Drosophila* Cl_{swell} channel. Candidates with transmembrane domains and human homologs were further studied to determine if they formed the Cl_{swell} channel. ✓ indicates a positive secondary screening result; X indicates a negative result.

☆ indicates that several qPCR primer sets consistently had more than 1 melting point peak suggesting nonspecific primer binding. The effectiveness of RNAi knockdown, therefore, could not be determined by qPCR. ?? indicates that two cells overexpressing SLC1A2 had substantial I_{SCN-} currents but small $I_{Clswell}$. Thus, SLC1A2 overexpression may upregulate endogenous HEK cell $I_{Clswell}$ in the majority of the population but does not form the channel itself. HeLa cells treated with SLC1A3 siRNA (which reduced SLC1A2 and SLC1A3 mRNA by 90% and 92% respectively) had unaltered $I_{Clswell}$ (data not shown).

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