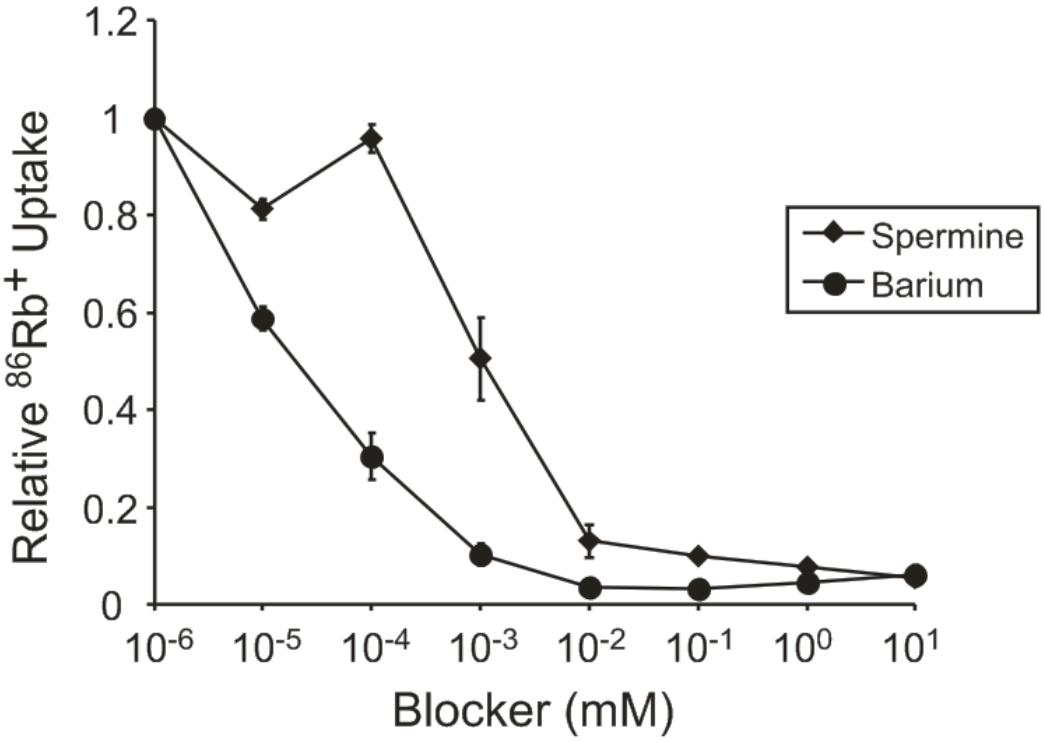
Supporting Material

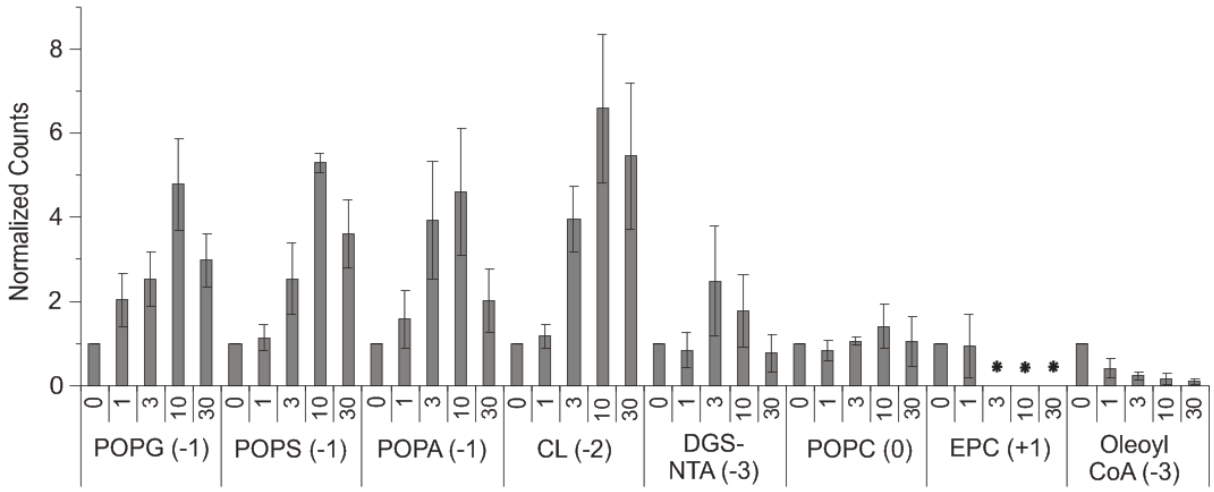
Dual-Mode Phospholipid Regulation of Human Inward Rectifying Potassium Channels

Wayland W. L. Cheng,[†‡] Nazzareno D'Avanzo,[†‡] Declan A. Doyle,[§] and Colin G. Nichols[†‡]*

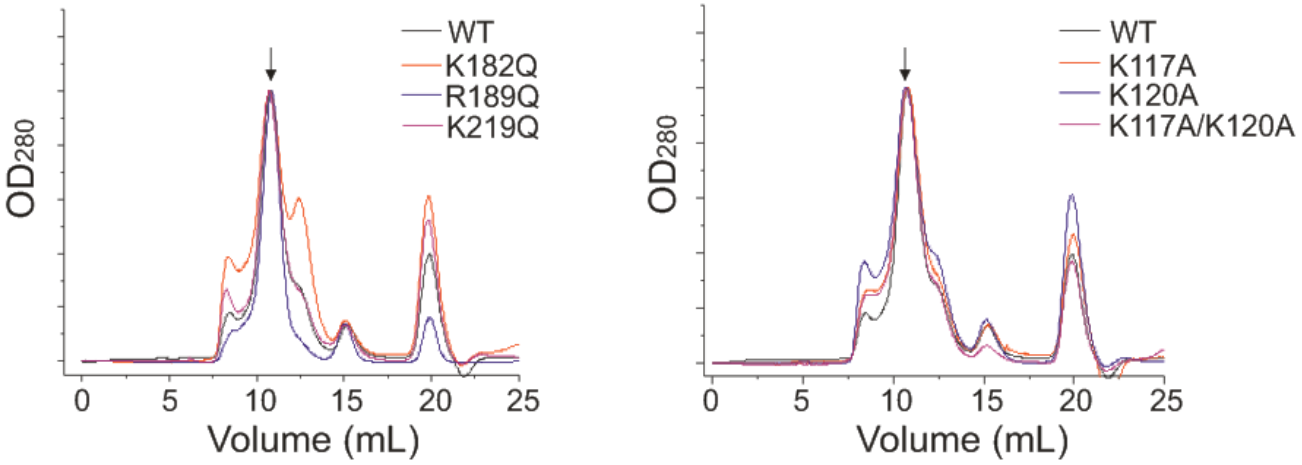
[†]Department of Cell Biology and Physiology, and [‡]Center for Investigation of Membrane Excitability Diseases, Washington University School of Medicine, St. Louis, Missouri; and [§]School of Biochemistry and Immunology, Trinity College Dublin, Dublin, Ireland



SUPPLEMENTARY FIG. 1 $^{86}Rb^{+}$ uptake counts of Kir2.1 with addition of extra-liposomal Ba^{2+} or spermine. Valinomycin-normalized counts are plotted relative to counts with 10^{-6} mM of blocker (n=3, +/- s.e.m.).



SUPPLEMENTARY FIGURE 2 $^{86}Rb^{+}$ uptake counts of Kir2.2 showing anionic phospholipid dependence on a 1% PIP_2 background as in Fig. 2A. Counts are taken at 20 min (n=3, +/- s.e.m.).



SUPPLEMENTARY FIGURE 3 Gel Filtration profiles of WT and mutant Kir2.1 channels. Gel filtration profiles of Kir2.1 and all mutants tested were similar with the protein peak eluting at the same volume for all the mutants. Three 0.5 mL fractions were collected from 10-11.5 mL and concentrated for functional experiments.