The Journal of General Physiology

Specificity of cholesterol and analogs to modulate BK channels points to direct sterol–channel protein interactions

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Volume 137, No. 1, January 3, 2011. Pages 93–110.

Please note that in the original Fig. S1 B, the y axis was mislabeled. The y axis of the plot should have been Po/Po_{Max} rather than Po. The correct figure and legend appear below.

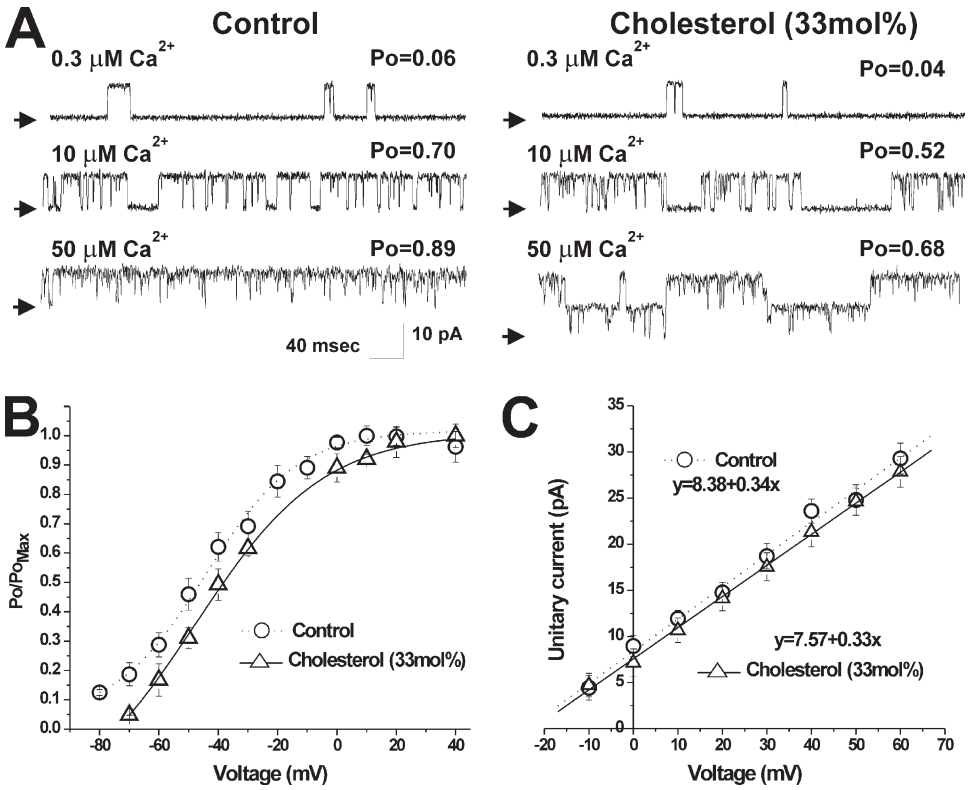

Figure S1. Basic properties of BK channels in POPE/POPS 3:1 (wt/wt) bilayers. (A) Original records of cbv1 channel activity obtained at 0.3, 10, and 50 μM Ca^{2+} at the cytosolic side of the control sterol-free POPE/POPS 3:1 (wt/wt) and 33 mol% cholesterol-containing lipid bilayer. Records show a progressive increase in Po as $[Ca^{2+}]_i$ is increased. Channel openings are shown as upward deflections; arrows indicate the baseline. The membrane potential for each recording was set to 0 mV. (B) A voltage (V)–Po plot obtained after the incorporation of cbv1 protein into control ($n = 5$) versus cholesterol-containing ($n = 7$) bilayers underscores that cholesterol presence does not alter the voltage dependence of cbv1 channel gating. Po values at each voltage in cholesterol-containing versus cholesterol-free (control) bilayers were normalized to their corresponding maximum (Po_{Max} in cholesterol-containing bilayer, ≈0.7; Po_{Max} in control bilayer, ≈0.9). (C) Cbv1 channel unitary current amplitude (i)–voltage (V) relationships from records obtained in 300/30 mM K^+ render unitary (slope) conductances of ≈340 and ≈330 pS for control ($n = 5$) and cholesterol-containing ($n = 7$) bilayers; these values are characteristic of BK channels. For B and C, data were obtained at $[Ca^{2+}]_i = 10$ μM. Curve fitting was performed using Origin7 (OriginLab).