# Description of Additional Supplementary Files

**Supplementary Movie 1:** Activation mechanism and key conformational changes of dTRPγ upon camphor binding.

In dTRPγ $_{Apo}$ state, an exogenous CHS binds within the vanilloid site. When camphor binds to the same pocket, it competitively displaces CHS and induces conformational changes of the channel protein. Conformational waves extend from the binding pocket, triggering backward tilting of VSLD away from the pore axis. Then, movement of the S4-5 linker propagates these conformational changes toward the ion permeation pathway. Pore profile exhibits an expansion of the permeation pathway, underlying the structural basis of dTRPγ activation by camphor.

**Supplementary Movie 2:** Conformational changes induced by $Ca^{2+}$-binding at the inhibitory site.

In dTRPγ-0 $Ca^{2+}$ and dTRPγ-1 $Ca^{2+}$, the ICDs adopt loosely packed conformations. When $Ca^{2+}$ binds at the $CBS_{ICD}$ in dTRPγ-2 $Ca^{2+}$, it shows a tightly packed arrangement. The binding of inhibitory $Ca^{2+}$ drives a secondary structure transition in the distal N-terminus from an α-helix to a loop, accompanied by the rigid-body movements of the ARD and CCD. These conformational rearrangements remodel the subunit interface by establishing a dense network of salt bridges and polar interactions, and filling the space around the CCD. This process stabilizes the compact ICD architecture and blocks cation permeation.