Functional cooperation between the IP3 receptor and phospholipase C secures the high sensitivity to light of Drosophila photoreceptors in vivo.

By elsec_admin
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By elsec_admin April 22, 2015


Abstract:

Drosophila phototransduction is a model system for the ubiquitous phosphoinositide signaling. In complete darkness, spontaneous unitary current events (dark bumps) are produced by spontaneous single Gq? activation, while single-photon responses (quantum bumps) arise from synchronous activation of several Gq? molecules. We have recently shown that most of the spontaneous single Gq? activations do not produce dark bumps, because of a critical phospholipase C? (PLC?) activity level required for bump generation. Surpassing the threshold of channel activation depends on both PLC? activity and cellular [Ca(2+)], which participates in light excitation via a still unclear mechanism. We show here that in IP3 receptor (IP3R)-deficient photoreceptors, both light-activated Ca(2+) release from internal stores and light sensitivity were strongly attenuated. This was further verified by Ca(2+) store depletion, linking Ca(2+) release to light excitation. In IP3R-deficient photoreceptors, dark bumps were virtually absent and the quantum-bump rate was reduced, indicating that Ca(2+) release from internal stores is necessary to reach the critical level of PLC? catalytic activity and the cellular [Ca(2+)]) required for excitation. Combination of IP3R knockdown with reduced PLC? catalytic activity resulted in highly suppressed light responses that were partially rescued by cellular Ca(2+) elevation, showing a functional cooperation between IP3R and PLC? via released Ca(2+). These findings suggest that in contrast to the current dogma that Ca(2+) release via IP3R does not participate in light excitation, we show that released Ca(2+) plays a critical role in light excitation. The positive feedback between PLC? and IP3R found here may represent a common feature of the inositol-lipid signaling.

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