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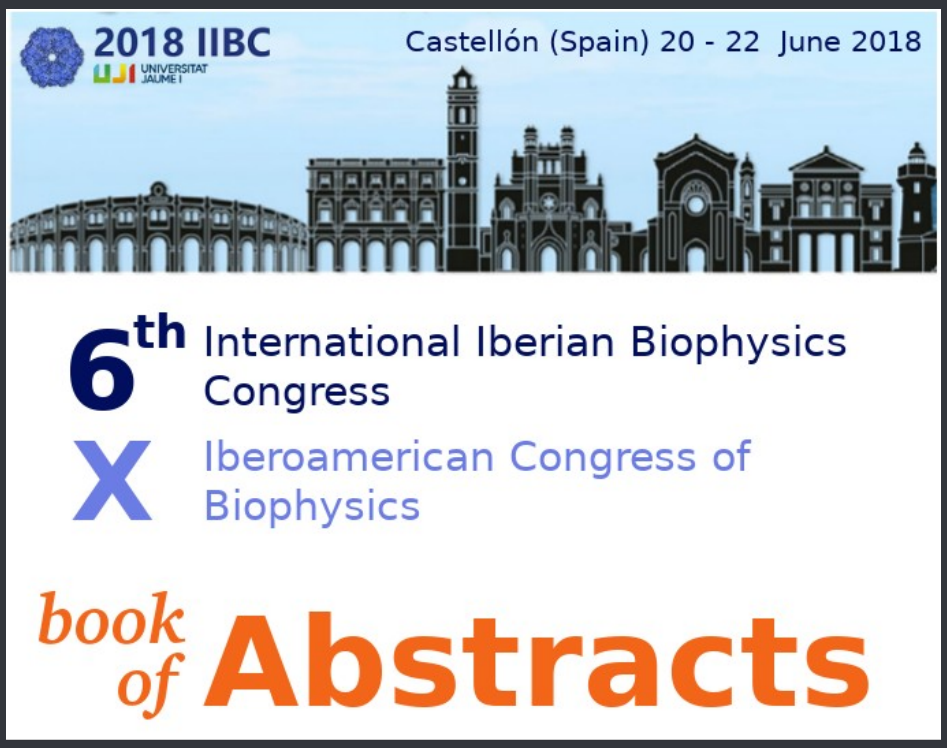

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P3.10 The antiparasitic bephenium is a potent agonist of caenorhabditis elegans levamisole-sensitive nicotinic receptors

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Nicotinic acetylcholine receptors (nAChRs) are ligand-gated ion channels involved in neuromuscular transmission. In nematodes, muscle nAChRs are main targets of antiparasitic drugs. Nematode parasites contain three pharmacological classes of muscle nAChRs, which are activated by levamisole (L-type), nicotine (N-type) and bephenium (B-type). The free-living nematode *Caenorhabditis elegans* is a model of parasitic nematodes, useful for drug discovery. Because in *C. elegans* muscle only the N-AChR and L-AChR classes have been described, we explored the behavioral (by paralysis assays) and molecular actions (by patch clamp recordings) of the antiparasitic bephenium. As in parasites, bephenium produced spastic paralysis. A mutant strain lacking the L-AChR showed full resistance to bephenium, indicating that this receptor is the drug target. Bephenium activated L-AChRs from isolated larvae muscle cells, eliciting channel activity as that elicited by levamisole. The analysis revealed that it is a potent agonist of the L-AChR and an open-channel blocker at higher concentrations. In contrast, we demonstrated that it is a very low efficacious agonist of the mammalian muscle nAChR. Molecular docking studies proposed that bephenium can form key interactions required for activation in mammalian and nematode nAChRs, revealed differences with ACh binding, and provided explanations for the experimental results.

P3.11 Origin of desensitization in the light-gated ion channel channelrhodopsin

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Channelrhodopsins (ChRs) are light-gated cation channels containing an all-trans retinal as a chromophore. In spite their wide use to activate neurons with light, the photocurrents of ChRs rapidly decay in intensity under both continuous illumination and fast trains of light pulses, broadly referred to as desensitization. This undesirable phenomenon has been explained by two interconnected photocycles, each of them containing a non-conductive dark state (D1 and D2) and a conductive state (O1 and O2). While the D1 and O1 states correspond to the dark-state and P3(520) intermediate of the primary all-trans photocycle of ChR2, the molecular identity of D2 and O2 remains unclear. By performing comprehensive time-resolved UV/vis experiments on dark-adapted and pre-illuminated sample we show that the P4(480) state, the last intermediate of the all-trans photocycle, is photoactive. Its photocycle contains a red-shifted intermediate, I3(530), whose decay matches the decay of O2 to D2. This and other results indicate that the D2 and O2 states correspond to the P4(480) and I3(530) intermediates, connecting desensitization of ChR2 with the photochemical properties of the P4(480) intermediate. This finding has important practical consequences in strategies aiming to reduce the desensitization tendency of ChR2 for optogenetic applications.