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Protein Kinase Modulates the $GABA_C$ Currents in Cone-horizontal Cell Axon-terminals Isolated from Catfish Retina

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Protein kinase modulation of gamma-aminobutyric acid C ($GABA_C$) currents in freshly dissociated catfish retinal cone-horizontal cell axon-terminals was studied under voltage clamp with the use of the whole cell patch-clamp technique. Responses to pulses of GABA were monitored in intracellular application of adenosin 3′,5′-cycle monophosphate (cAMP)-dependent protein kinase (PKA) and protein kinase C (PKC) activators, and their inhibitors or inactive analogues. Intracellular application of forskolin, an adenylate cyclase activator, reduced the GABA activated current, while H8 dihydrochloride, a cAMP inhibitor, increased the GABA response. 1-oleoyl-2-*sn*-glycerol (OAG), a PKC activator, increased the GABA activated current. GF-109203X, a PKC inhibitor, reduced the GABA response like PKA analogues. The GABA activated Cl^- currents were increased by PKC and suppressed by PKA. Furthermore, GABA responses with intracellular application of OAG were reduced by after pretreatment of forskolin. These results suggest that retinal $GABA_C$ responses in cone-horizontal cell axon-terminals are modulated by both protein kinase A and C on different phosphorylating sites, respectively.