Prostaglandin E2 (PGE2) is a crucial mediator of inflammatory pain sensitization. Harvey et al.demonstrated that inhibition of a specific glycine receptor subtype (GlyR alpha3) by PGE2-induced receptor phosphorylation underlies central inflammatory pain sensitization.

They showed that GlyR alpha3 is distinctly expressed in superficial layers of the spinal cord dorsal horn. Mice deficient in GlyR alpha3 not only lack the inhibition of glycinergic neurotransmission by PGE2 seen in wild-type mice but also show a reduction in pain sensitization induced by spinal PGE2 injection or peripheral inflammation. Thus, GlyR alpha3 may provide a previously unrecognized molecular target in pain therapy.

They showed that punctate GlyRα3 IR is distinctly expressed in lamina II of the spinal cord dorsal horn, and GlyRα3 subunit IR puncta were found to colocalize with gephyrin [1)].

1)
Harvey RJ, Depner UB, Wässle H, Ahmadi S, Heindl C, Reinold H, Smart TG, Harvey K, Schütz B, Abo-Salem OM, Zimmer A, Poisbeau P, Welzl H, Wolfer DP, Betz H, Zeilhofer HU, Müller U. GlyR alpha3: an essential target for spinal PGE2-mediated inflammatory pain sensitization. Science. 2004 May 7;304(5672):884-7. PubMed PMID: 15131310.