GS03-1 **Inhibitory effect of angiotensin (1-7) on streptozotocin-induced diabetic neuropathic pain in mice**

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[Background] We have reported that expression of spinal angiotensin (Ang) converting enzyme is increased in streptozotocin (STZ)-induced type 1 diabetic mice, which in turn leads to an activation of Ang II production system. Moreover, this activation of Ang II production system causes hyperalgesia accompanied by the phosphorylation of p38 MAPK through AT1 receptors [Mol. Pharmacol., 90:205-13 (2016)]. In addition, intrathecal (i.t.) administration of Ang (1-7), an N-terminal fragment of Ang II, attenuates Ang II-induced nociceptive behavior accompanied by the inhibition of p38 MAPK phosphorylation via Mas receptors [Eur. J. Pain, 18:1471-1479 (2014)]. Here, we examined the effect of Ang (1-7) on diabetic neuropathic pain using STZ-induced type 1 diabetes mice. [Results & Conclusions] STZ-induced hyperalgesia was inhibited by i.t. administration of Ang (1-7). The inhibitory effect of Ang (1-7) was significantly reversed by co-administration of A779, a Mas receptor antagonist. Western blot analysis showed that the expressions of phospho-p38 MAPK, -ERK 1/2 and -JNK were increased in spinal cord of STZ mice. Among them, i.t. injection of Ang (1-7) inhibited only phosphorylation of p38 MAPK, which was significantly reversed by A779. Furthermore, spinal Mas receptors were expressed in neurons and microglia but absent from astrocytes. Our data show that Ang (1-7) inhibits STZ-induced hyperalgesia accompanied by the inhibition of p38 MPAK phosphorylation via Mas receptors.