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Poster Sessions

The mechanism of serotonin-induced constriction in 2K1C hypertensive rat mesenteric arteries

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Inactivation of myosin light chain phosphatase thorough myosin phosphatase targeting subunit 1 phosphorylation by Rho kinase is important for serotonin (5-HT)-induced constriction. However, the detailed mechanism of Rho kinase activation by 5-HT is unknown. We investigated whether 5-HT-induced constriction in 2-kidney, 1-clip (2K1C) hypertensive rat mesenteric arteries is mediated by Rho kinase activation through Src, epidermal growth factor receptor (EGFR), extracellular signal-regulated kinase 1 and 2 (Erk1/2), and Janus kinase (JAK).

2K1C hypertensive rats were produced by clipping of right renal arteries. After 7 weeks, mesenteric arteries were excised and performed organ chamber experiments.

Cumulative application of 5-HT induced a concentration-dependent constriction in the mesenteric arterial rings of both sham-operated and 2K1C hypertensive rats. The 5-HT-induced constriction increased to a greater extent in the 2K1C hypertensive rats. Contractile response to 5-HT significantly attenuated by inhibitors of Rho kinase, Erk1/2, Src, and EGFR in sham-treated and 2K1C hypertensive rats. However, a JAK inhibitor had no effect on 5-HT-induced constriction. These data suggest that 5-HT-induced constriction is mediated by Rho kinase activation via Src, EGFR, and Erk1/2 in 2K1C hypertensive rat mesenteric arteries.