Involvement of thermo-sensitive TRPM8 channels in visceral hypersensitivity in irritable bowel syndrome animal models

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AIM: We prepared two models, namely butyrate-induced irritable bowel syndrome (IBS) model rats and a post-inflammatory IBS (PI-IBS) model mice using dextran sulfate sodium (DSS). In the present study, we investigated the involvement of TRPM8 in visceral hyperalgesia in the two IBS models. **METHODS:** Butyrate-induced IBS model was prepared by intracolonic treatment with butyrate for 4 days in SD male rats. PI-IBS model was prepared by drinking freely 3% DSS until Day 4, then changing 3% DSS to tap water during recovery period. Visceral pain was induced by intracolonic treatment with the TRPM8-selective agonist WS-12, and the observed visceral pain-like behaviors ware mesured. TRPM8 immunoreactivities were detected using immunohistochemical techniques. **RESULTS:** In rectal histology of butyrate-induced IBS model rats, the number of TRPM8-expressing nerve fibers in the mucosal layer ware significantly increased compared with normal rats. In behavioral observation, WS-12-induced visceral pain-like behaviors were increased. In PI-IBS model mice, an increase in visceral pain-like behaviors was observed on Day 4, and the pain symptom observed until Day 14. **CONCLUSIONS:** These results suggest that the increase of TRPM8-expressing nerve fibers in lower gastrointestinal tracts is involved in hyperalgesia in IBS model animals.