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Acute Stress Ameliorates Colitis via Central Corticotropin- Releasing Factor and Serotonin (5-HT)-3 Receptors

Mehmet Ali GÜLPINAR[1], Dilek ÖZBEYLİ[2], Serap ARBAK[2], Berrak Ç. YEĞEN[1]
[1]Department of Physiology, Faculty of Medicine, Marmara University, Haydarpaşa, İstanbul - Turkey
[2]Department of Histology & Embryology, Faculty of Medicine, Marmara University, Haydarpaşa, İstanbul - Turkey

Abstract: The central nervous system may modulate many aspects of inflammation through alterations of the autonomic nervous system and/or the hypothalamo-pituitary-adrenal (HPA) axis. The aim of present study was to examine the impact of controllable acute stress on the course of experimental colitis and to elucidate the roles of serotonin (5-HT) and corticotropin-releasing factor (CRF) in the central modulation of the stress. In the stress group, acute electric shock (AES; 0.3-0.6 mA) was applied, while in the central agonist group, rats were injected with central 5-HT agonist (400 µg/kg). In the antagonist groups, 10 min before the application of AES or central agonist, rats were injected with astressin (10 µg/kg; icv) or ramosetron (40 µg/kg; icv). Hexamethonium (15 mg/kg; ip) or RU-486 (10 mg/kg; ip) were given before and after stress or central agonist application. The severity of colonic damage was evaluated by the assessment of macroscopic score, histological analysis and tissue myeloperoxidase activity. The data are expressed as means ± SE and analyzed using Student's t-test or the Mann-Whitney U test. Both AES and central 5-HT decreased colonic damage scores seem to be mediated by central CRF and 5-HT3 receptors and by adrenal corticosteroids and sympathetic ganglia. These results show that the anti-inflammatory effect on colitis may be mediated by central mechanisms that involve the interaction of CRF and 5-HT, with the participation of the sympathetic system and HPA axis.

Key Words: Colitis, serotonin, stress, HPA axis, sympathetic nervous system

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