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Differences in gastrointestinal motility between mouse lines with high and low swim-stress-induced analgesia is dependent on opioid receptor activity in the central nervous system

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It is well known that the activation of Gastrointestinal (GI) opioid receptors by endogenous and exogenous opioids results in GI Transit (GIT) inhibition. However, a direct linkage between the activity of the opioid system and GIT remains largely unknown. The aim of this study was to characterize the activity of central and GI opioid receptors that presumably influence GIT in two mouse lines divergently bred for high (HA line) and low (LA line) Swim Stress-Induced Analgesia (SSIA). The contribution of opioid receptors to the regulation of peristalsis in HA and LA mice was investigated by means of morphine-stimulated [35S]GTPγS assay in the CNS and in the small and large intestine. HA mice compared to their LA counterparts showed enhanced G-protein activation in the thalamus (152±2.6 vs. 136±2.4, $p<0.001$), cortex (130±1.9 vs. 117±1.7, $p<0.001$) and the spinal cord (135±1.4 vs. 120±2.6, $p<0.001$). No stimulation of [35S]GTPγS binding was detected in the small or large intestine. In conclusion, differences in gastrointestinal transit between HA and LA mice could be reflected by alterations in opioid-related G - protein activation in the CNS rather than the gastrointestinal tract.

Biography

Mariusz Sacharczuk is currently working in the Department of Pharmacodynamics/Department of Internal Medicine and Hypertension at Medical University of Warsaw, Poland.

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