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Xi Huang

Nanjing University of Chinese Medicine, China

Depression-like behavior and hypomotility in acutely forced swimming rat improved by prokinetic meranzin hydrate from Chaihu-Shugan-San via ghrelin and neurocircuitry

Depression and Functional Dyspepsia (FD) are characterized by comorbidity, overlap depressed and nausea etc. and specially one-disease-one-drug-one-target with inefficacy. Parted accumulations of depression or FD's pathogenesis mediated by α2-adrenoreceptor or ghrelin etc. are considerable but shared little. Antidepressant (A) or prokinetic (P) agents are many but few A&P. Hippocampus or thalamus-coupled depression or FD is countless but void comorbidity. Ancient Gan-Zhu-Shu-Xie (GZSX whose representative TCM is Chaihu-Shugan-San) clued us to discover simultaneous antidepressant from prokinetic Meranzin Hydrate (MH) via shared α2-adrenoreceptor in acutely Forced Swimming (FS) rat from homogeneous comparison. Here we via Chaihu-Shugan-San as tool study causalities from monism (FS, MH and ghrelin) to dualism (gut-brain disorder, A&P and shared regulation) and fuse above via 7.0 T fMRI-BOLD signal, compared with well-known mechanism of positive control. Top 3 foci of BOLD following FS rat with depressed behavior and hypomotility (DB&H) are activities of Hippocampus-Thalamus-Basal ganglia (HTB) circuit. A&P MH similar to CSS stimulated deactivation of 4.02-fold for hippocampus and 1.45-fold for thalamus than fluoxetine. Ghrelin antagonist [D-Lys3]-GHRP-6 synchronously inhibited above A&P and BOLD HTB foci. As expected, prokinetic mosapride only acted on thalamus and basal ganglia and nothing for hippocampus. Among HTB, hippocampus is as protagonist for depression and deputy for FD, thalamus is on the contrary, often basal ganglia is as projection to HT according to percentage of HTB each from search of PubMed. This functionally novel connection of HTB following acute stress, treatment and regulation highlights (anti) prokinetic unified theory.

Biography

Xi Huang is currently working at Nanjing University of Chinese Medicine, China.

tcmhuangx59@163.com