# Increased Cholinergic Enteric Neuromuscular Pathways Brought on by Psychological Stress are Mediated by Glucocorticoid Receptors

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## Abstract

A common word for the processes thought to be involved in a number of mental and physical diseases is psychological stress. There is little agreement on what constitutes psychological stress, despite the fact that the concept and its effects on health and wellbeing are of great importance. The origins, evolution, and present status of three definitional and research approaches to psychological stress are examined. The proportional weight that each of the three viewpoints on psychological stress places on the environment, the organism, and how those three factors interact through time varies. The three views' conceptual, methodological, and practical consequences are discussed. Promising leads for further investigation are dealt with.

**Keywords:** Repeated acute stress

## Introduction

Gastrointestinal dysfunctions are known to be correlated with Repeated Acute Stress (RASt). The processes behind these impacts are still not completely known, though. While glucocorticoids are unmistakably stress hormones, their role in RASt-induced gastrointestinal dysfunctions as well as the function of Glucocorticoid Receptors are yet unknown (GR). Our study's objective was to assess GR's contribution to RASt-induced alterations in gut motility, especially via the Enteric Nervous System (ENS).

We identified the effects of RASt on the ENS phenotype and intestinal motility using a mouse Water Avoidance Stress (WAS) paradigm. Then, we looked at the ENS's expression of glucocorticoid receptors and how those changes in ENS phenotypic and motor response were caused by RASt.

We demonstrated that under baseline circumstances, GR were expressed in myenteric neurons in the distal colon, and that RASt facilitated their nuclear translocation. In comparison to controls, RASt raised the percentage of ChAT-immunoreactive neurons, the tissue concentration of acetylcholine, and improved cholinergic neuromuscular transmission. The rise in acetylcholine levels in colonic tissue and in vivo colonic motility were both inhibited by a GR-specific antagonist, which we demonstrated using the compound CORT108297. Our research implies that a GR-dependent increased cholinergic component in the ENS is at least partially responsible for the functional alterations in motility caused by RASt.

Repeated Acute Stress (RASt) is known to be linked to GI disorders or dysfunctions like visceral hypersensitivity and altered gastrointestinal motility, both of which result in slowed gastric emptying and sped up colonic transit time, or altered epithelial functions like increased epithelial secretion or permeability. The mediators and receptors involved, as well as the processes causing RASt-induced alterations in gastrointestinal functioning, have not yet been fully uncovered.

Each element of the stress response is activated in response to repeated acute stress. This reaction can take two different forms. On the one hand, sympathetic nervous system activation causes a quick release of neuromediators such catecholamines with adrenaline and noradrenaline, which then causes rapid synaptic effects. On the other hand, stimulation of the Hypothalamic-Pituitary-Adrenal (HPA) axis results in delayed effects and may result in longer-lasting alterations. The HPA axis is activated when the brain releases corticotropin-releasing hormone (CRH), which then triggers the pituitary gland to release Adreno-Corticotropic Hormone (ACTH), and the adrenal glands to release Glucocorticoids (GC). Among the mediators generated during HPA activation and implicated in RASt effects on GI, CRH has received the greatest research attention.

The ENS, an integrated neural network situated all throughout the gastrointestinal tract and involved in the control of gastrointestinal processes including motility and secretion, can be directly affected by corticotropin-releasing hormone in particular. The activation of the corticotropin-releasing factor receptor subtypes 1 and 2 was found to mediate CRH effects on the ENS in a region- and function-dependent way (CRF-R1 and CRF-R2). The idea that stress may affect digestive function by affecting these receptors has been investigated in several researches. In addition, CRH activates both of these receptors, but doing so results in functionally diverse reactions: although CRF-R1 activation improves colonic motor response, CRF-R2 activation decreases stomach emptying. Furthermore, CRH activates CRF-R2, which results in an increase in mucosal chloride secretion.

The involvement of GC in RASt-mediated effects affecting GI functions and its probable impact upon the ENS is still substantially unclear when compared to CRH. The Glucocorticoid Receptor (GR) and the Mineralocorticoid Receptor (MR) are the two main nuclear receptors that mediate the effects of GC. MR receptors, which are often active under baseline circumstances, are highly affine for GC. The GR receptors that are active under "inducible" circumstances, such as during HPA activation, have a decreased affinity for GC. When GC activates GR, it moves from the cytoplasm to the nucleus and binds to the GR response element, which activates a number of transcriptional processes. The corticosterone-degrading enzyme 11-Hydroxysteroid Deshydrogenase type 2 (11-HSD-2) limits the effects of corticosterone.

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