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Dysregulated stress signal sensitivity and inflammatory disinhibition as a pathophysiological mechanism of stress-related chronic fatigue

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Graphical abstractAnnotation: *Stress signal sensitivity as a pathophysiological mechanism in chronic fatigue*. We propose that sensitivity of immune cells to stress signals (glucocorticoids, catecholamines) may be the missing link in elucidating how stress turns into chronic fatigue. Initial findings point towards a reduction of basal stress signal sensitivity under chronic stress ("Basal state"). A decrease of stress signal sensitivity after a challenge has been shown in a chronically stressed population ("Acute stress condition"). In the long run, resistance of immune cells to stress signals under conditions of chronic stress, further reinforced under acute stress, might translate into self-maintaining inflammation and inflammatory disinhibition under acute stress, respectively, which in turn lead to chronic fatigue.

Abstract

Chronic stress and its subsequent effects on biological stress systems have long been recognized as predisposing and perpetuating factors in chronic fatigue, although the exact mechanisms are far from being completely understood. In this review, we propose that sensitivity of immune cells to glucocorticoids (GCs) and catecholamines (CATs) may be the missing link in elucidating how stress turns into chronic fatigue. We searched for in vitro