# **PERSPECTIVE**

# Stress resiliency and social interaction

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#### ABSTRACT

Our physical and mental healths are challenged by modern lifestyles and difficulties such as the COVID-19 pandemic. As a result, identifying ways through which humans might build stress resistance and quickly respond to adversity is critical. While a variety of factors contribute to stress resilience, social activity, particularly in the form of social touch, is very important. The somatosensory

system plays a significant role in transforming the socio-emotional information of social touch into active coping with stress, according to this study. Examining whether stress resilience can be controlled in humans through the activation of low-threshold C-fiber mechanoreceptors, as well as applying this technology to avoid stress-related neuropsychiatric illnesses like major depressive disorder, are important future directions.

Key Words: Stress; Resilience; Somatosensory system; Social interaction

#### INTRODUCTION

igh levels of stress are associated with modern lifestyles, particularly in the workplace. There are also social issues confronting humanity now, such as the mental health catastrophe caused by the COVID-19 epidemic. SARS-CoV-2 and its dangerous variations have significantly elevated human stress levels, owing to increased perceived threat and social isolation. As a result, mental problems such major depressive disorder and anxiety disorders are becoming increasingly common. It is necessary to first understand the neurobiological underpinnings of the stress response in order to grasp the link between resistance to stress and social touch [1]. Hans Selve created the term "stress" in the twentieth century. This idea highlights how our bodies' physiology is constantly trying to conform to and adapt to external demands. These environmental demands or stressors are perceived by our sensory systems. In the sensory organs, sensory information is transformed into action potentials, which are then sent to the brain, where the risk levels of stressors are assessed [2] . When the organism's integrity is endangered, brain regions like the amygdala and hypothalamus trigger physical changes and influence the stress response. The sympathetic nervous system is activated as part of this response, which results in the release of adrenaline from the medulla of the adrenal glands into the bloodstream, among other things. Adrenaline speeds up the heart rate and circulates the blood, preparing the body to deal with stress [3-4]. The Hypothalamic-Pituitary-Adrenal (HPA) axis, which is part of the delayed stress response, is the second system to be activated. This neuroendocrine axis converts stress-related neural activity into a hormonal message for the adrenal glands to process. The primary stress hormone (cortisol in humans and corticosterone

in rats) is released into the bloodstream from the cortex of these glands. These hormones raise blood sugar levels and create the energy needed to keep the body moving in difficult situations. The locus coeruleus and the endocannabinoid system, for example, are two extra-hypothalamic systems that play a crucial role in the development and regulation of stress reactions. Sensory systems are important in stress because they carry information from the environment to the amygdala, which is responsible for activating and deactivating stress reactions. Without that information, the amygdala is unable to assess the risk of threat and adjust stress responses accordingly. Despite the fact that sensory systems play a critical role in both producing and suppressing stress, their therapeutic potential has remained largely untapped. The somatosensory system and, more particularly, the skin's cutaneous mechanoreceptors (also known as low-threshold mechanoreceptors) are one possible therapeutic use (LTMs) CLTM afferents in non-human animals and C-Tactile (CT) afferents in humans are a type of unmyelinated C fibres found in these LTMs. These fibres are thought to transmit affective touch between conspecifics by preferring soft, dynamically moving stimuli at velocities of 1 cm/s -10 cm/s. Affective touch transmits socioemotional information to the posterior insula, a part of the brain. This area is a multisensory hub that is well connected to the anterior insula, and it connects to other sections of the brain such as the prefrontal cortex, anterior cingulate cortex, orbitofrontal cortex, and amygdala through this pathway. As a result, affective touch may be a critical role in improving stress adaptation and promoting stress resilience [5]. Touch directed at other LTMs, such as A-beta fibres and their projections to the primary and secondary somatosensory cortex, may, nevertheless, be relevant for stress.

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The brain circuits that govern stress responses are remodelled by our early life experiences and accompanying epigenetic alterations, such as glucocorticoid receptor expression. The biological mediators that regulate the reaction to stress enhance their performance each time we are exposed to stresses and adapt to them, much as a runner improves their performance via daily training. Norman Garmezy was the first to pay attention to how humans deal with stress in the early 1970s. He saw that certain youngsters had an active coping mechanism for stress and were able to adapt more quickly [6].

When we are exposed to stress and adapt to it, we develop stress resilience, which is a biological-behavioral process that occurs over time. Simply said, resilience is the result of the lessons we've learned from our stressful life situations[7].

Bruce McEween's concept of allostasis can assist clarify this concept: stress pulls us away from the homeostasis or equilibrium we have with the environment in which we live. Allostasis is the process of returning to homeostasis by generating the physiological characteristics required for the mediators of the stress response to function outside of homeostasis. The performance of the sympathetic nervous system and the HPA axis in this circumstance necessitates an energy cost or allostatic load that allows adaptation to the environment.

The effects of stress are translated into changes in neuronal plasticity in brain areas that influence the response to stress, such as the hippocampus and the medial prefrontal cortex, by glucocorticoids such as cortisol and corticosterone activating glucocorticoid receptors [8-9]. When allostatic mediators, such as the sympathetic nervous system and the HPA axis, are exposed to stressors again, stress hormones improve their performance. As a result, the stress coping response improves, allowing for faster adaptability to stresses. When the body fails to adjust to stress, an allostatic overload occurs, resulting in an imbalance in the release of glucocorticoids as well as the ratio of glucocorticoid receptors to mineralocorticoid receptors. When compared to glucocorticoid receptors, mineralocorticoid receptors have a 10 fold greater affinity for corticosterone. Mineralocorticoid receptors bind glucocorticoids in the cytoplasm of neurons during the start of the stress response, while glucocorticoid receptors are active towards the end of the stress response and contribute in adaptation and recovery. At a pathological level, an imbalance in the ratio of glucocorticoid receptors to mineralocorticoid receptors neuro-inflammatory processes hippocampus, amygdala, and medial prefrontal cortex, impairing the ability to cope with stress and increasing susceptibility to stress-related neuropsychiatric diseases like mood disorders and neurodegenerative diseases like Alzheimer's disease. It is vital to understand what behavioural elements can promote stress resilience in order to prevent certain mental diseases. The activity of the HPA axis is ideal for creating active coping with stress and attaining adaptation, which is the key biological hallmark of stress resilience. Lower or higher levels of HPA axis activity cause allostatic overload and produce the stress-prone behavioural phenotype. Slow stroking, which may engage the CT system, may aid to maximise HPA activity, according to research. Before being subjected to a chronic unpredictable mild stress paradigm, rats' skin was stroked at a slow (5 cm/s) or fast (30 cm/s) velocity. Slow stroking, in contrast to fast stroking, tended to attenuate the HPA axis; plasma corticosterone levels in the slow stroking condition were comparable to those in non-stressed rats. Slow stroking had an anxiolytic impact and increased active coping in anxious animals, as evidenced by an increase in climbing and a decrease in floating behaviour in the forced swim test.

These findings show that the somatosensory system in rats can be used to modify stress resilience. Furthermore, they support a previous study that found moderate skin stimulation reduces the development of depressive-like behaviours and improves episodic memory in rats exposed to a chronic unpredictable mild stress regimen. In social interaction tests, resilient mice demonstrate active coping with social defeat stress, developing social behaviour similar to non-defeated rodents. Through two pathways that are engaged by social touch, social activity can modify stress responses. The oxytocin system and the HPA axis also communicate with one another. When rodents are stressed, oxytocin can inhibit the HPA axis, resulting in a drop in plasma corticosterone levels. In several mammals, this brain circuit plays a crucial role in the incentive to engage in social activity and acts as a natural reinforcer. The VTA-NAc-DA circuit has a high density of oxytocin receptors, which promotes its functioning during stress [10].

### CONCLUSION

Social buffering, which allows the development of active coping and stress resilience, is one of the most fundamental evolutionary benefits of social behaviour. C-LTM stimulation promotes stress resilience in rats in this situation. CTs will need to be studied in people in the future to establish a comparable causative route and to assess their therapeutic potential in stress-related disorders such major depressive disorder and anxiety disorder. As difficulties like as the COVID-19 epidemic raise the degree of stress in our life and its influence on our mental health, this will become increasingly important.

## REFERENCES

- Hassard J, Teoh K, Cox T, et al. Calculating the Costs of Work-Related Stress and Psychosocial Risks - A Literature Review. European Agency for Safety and Health at Work. 2014.
- Quittkat HL, Düsing R, Holtmann FJ, et al. Perceived impact of covid-19 across different mental disorders: a study on disorderspecific symptoms, psychosocial stress and behavior. Front Psychol. 2020; 3256.
- Sapolsky RM. Glucocorticoids, the evolution of the stressresponse, and the primate predicament. Neurobiol Stress. 2021; 14: 100320
- Pérez-Valenzuela C, Terreros G, Dagnino-Subiabre A. Effects of stress on the auditory system: an approach to study a common origin for mood disorders and dementia. Rev Neurosci. 2019; 30(3): 317-24
- 5. McEwen BS, Akil H. Revisiting the stress concept: implications for affective disorders. J Neurosci. 2020; 40 (1): 12-21
- Herman JP, Nawreen N, Smail MA, et al. Brain mechanisms of HPA axis regulation: neurocircuitry and feedback in context Richard Kvetnansky lecture. Stress. 2020; 23 (6): 617-32.
- 7. R Wyrofsky R, Reyes BA, Van Bockstaele EJ. Co-localization of the cannabinoid type 1 receptor with corticotropin-releasing factor-containing afferents in the noradrenergic nucleus locus coeruleus: implications for the cognitive limb of the stress response. Brain Struct Funct. 222 (7): 3007-23
- 8. Fast CD, McGann JP. Amygdalar gating of early sensory processing through interactions with locus coeruleus. J Neurosci. 2017; 37 (11): 3085-01
- 9. Marshall AG, McGlone FP. Affective touch: the enigmatic spinal pathway of the C-tactile afferent. Neurosci Insights. 2020; 15: 2633105520925072
- 10. Cascio CJ, Moore D, McGlone F. Social touch and human development. Dev Cogn Neurosci, 2019; 35: 5-11.