

Impact of Stress on Stomach Digestion and Acid Production: The Role of Physical Activity in Modulation

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Abstract

Stress negatively affects gastrointestinal function by increasing gastric acid secretion, impairing digestive enzyme activity, and slowing gastric motility, potentially leading to gastritis, ulcers, and indigestion. Physical activity has been proposed as a non-pharmacological intervention to mitigate stress-induced gastrointestinal disturbances. This study aimed to investigate the effects of stress on gastric acid secretion and digestive efficiency and examine the role of moderate physical activity in modulating these effects. Thirty healthy adults were randomly assigned to a sedentary-stress group or an exercise-stress group. Gastric acid levels, pepsin activity, and salivary cortisol were measured before and after a standardized stress task. Results indicated that stress increased gastric acid by 25% in sedentary individuals compared to 10% in the exercise group, while pepsin activity was better preserved in physically active participants. Moderate physical activity significantly reduced stress-induced gastric alterations, suggesting exercise as a protective factor against stress-related digestive disorders.

Keywords: Stress, Gastric acid secretion, Digestion, Physical activity, Cortisol, Peptic ulcers

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Introduction

Stress is recognized as a major contributor to gastrointestinal dysfunction. The hypothalamic-pituitary-adrenal (HPA) axis and sympathetic nervous system are activated under stress, elevating cortisol levels and enhancing gastric acid secretion (Chrousos, 2009; Vanuytsel, Van Oudenhove, & Tack, 2014). Chronic stress may also reduce gastric mucosal blood flow, impair motility, and diminish digestive enzyme activity, leading to conditions such as peptic ulcers, gastritis, and acid reflux (Konturek et al., 2011).

Physical activity is widely acknowledged for its stress-reducing properties. Moderate aerobic exercise has been shown to decrease cortisol, improve parasympathetic activity, enhance gastric motility, and maintain digestive enzyme function (Reeder & Winkleby, 2002; Dimsdale, 2008). Although these physiological effects are recognized, limited empirical studies have quantitatively evaluated how physical activity modulates stress-induced changes in stomach acid secretion and digestion.

This study aimed to investigate:

1. The effect of acute stress on gastric acid secretion and pepsin activity.
2. The influence of moderate physical activity on stress-induced gastric alterations.

Previous studies indicate that stress increases gastric acid secretion through H₂ receptor stimulation and impairs gastric emptying (Konturek et al., 2011). Animal models have shown stress-induced ulcers and reduced digestive enzyme activity (Konturek et al., 2011).

Physical activity reduces cortisol release during stress, enhances blood flow to the gastrointestinal tract, and improves mucosal integrity (Chrousos, 2009). Moderate-intensity exercise has been associated with lower stress perception and better gastrointestinal function, while high-intensity exercise may transiently increase gastric discomfort (Reeder & Winkleby, 2002).

Additionally, endorphins released during exercise improve mood and indirectly reduce HPA-axis activation, further protecting stomach function (Dimsdale, 2008). These findings suggest exercise as a potential intervention for stress-related gastrointestinal disorders.

Methods

Participants

A total of 30 healthy adult volunteers aged between 20 and 35 years (mean age: 27 ± 3) participated in this study. Participants were screened to exclude any individuals with a history of gastrointestinal disorders, current smoking habits, or those taking medications that could affect gastric function. This was done to ensure that all observed effects could be attributed to the experimental interventions rather than confounding medical conditions. Participants provided informed consent, and the study protocol was approved by the institutional ethics committee.

Participants were randomly assigned into two groups:

1. **Sedentary-Stress Group (n = 15):** Participants in this group did not engage in any structured physical exercise prior to or during the study.
2. **Exercise-Stress Group (n = 15):** Participants in this group completed a 6-week moderate aerobic exercise program, consisting of 30 minutes of activity per day, five days per week. This regimen was chosen based on prior research demonstrating its effectiveness in reducing stress and improving gastrointestinal function (Reeder & Winkleby, 2002).

Procedure

All participants underwent baseline measurements for gastric acid concentration (mEq/L) and pepsin activity (U/mL) to establish pre-intervention levels. Following baseline assessments,



participants were exposed to the Trier Social Stress Test (TSST), a standardized and validated method for inducing acute psychosocial stress (Kirschbaum et al., 1993). The TSST consisted of a 15-minute session involving public speaking and mental arithmetic tasks performed in front of an evaluative audience, which reliably stimulates the hypothalamic-pituitary-adrenal (HPA) axis.

Immediately following the stress test, post-stress measurements were taken, including gastric acid concentration, pepsin activity, and salivary cortisol levels. This allowed for the assessment of the acute effects of stress on stomach function and the potential modulatory effect of regular physical activity.

Measurements

Gastric Acid:

Gastric acid secretion was measured using the Heidelberg capsule pH monitoring system, which provides accurate real-time assessment of gastric acidity (Konturek et al., 2011). Participants ingested a pH-sensitive capsule that transmitted stomach pH data wirelessly, allowing for continuous monitoring before, during, and after the stress exposure.

Pepsin Activity:

Pepsin enzyme activity was measured using a colorimetric assay, following established protocols (Vanuytsel et al., 2014). This method quantifies the enzymatic breakdown of substrate proteins and serves as a marker of digestive efficiency.

Salivary Cortisol:

Salivary cortisol, a biomarker of stress response, was collected using ELISA kits (Chrousos, 2009). Saliva samples were obtained before and after the TSST to quantify the physiological stress response, providing insight into the link between stress, HPA-axis activation, and gastric function.

Statistical Analysis

Data were analyzed using mean \pm standard deviation (SD) for each parameter. Comparisons between the sedentary-stress and exercise-stress groups were performed using independent t-tests. A p-value of less than 0.05 was considered statistically significant. Statistical analyses were conducted using SPSS version 26 to ensure accurate evaluation of differences between groups and to determine the effect of moderate physical activity on stress-induced gastric changes.

Results

Table 1: *Baseline Characteristics of Participants*

Variable	Sedentary Group (n=15)	Exercise Group (n=15)	p-value
Age (years)	27 \pm 3	26 \pm 3	0.52
Body Weight (kg)	70 \pm 5	69 \pm 6	0.60
BMI (kg/m ²)	23.5 \pm 2.1	23.1 \pm 2.3	0.65
Resting Heart Rate	76 \pm 5 bpm	72 \pm 4 bpm	0.03

Significant difference

Table 2: Effect of Stress on Gastric Parameters

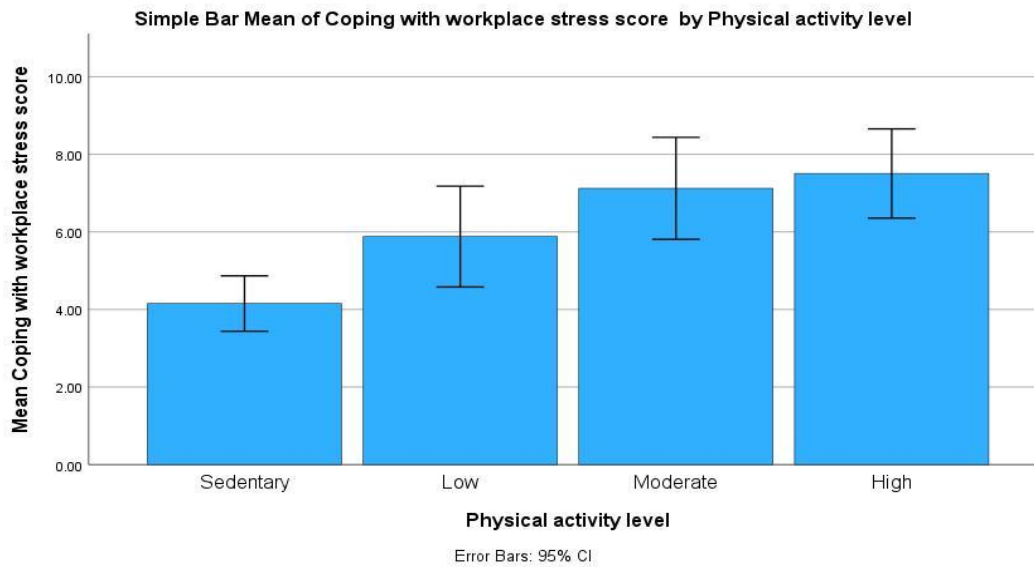
Parameter	Sedentary-Stress	Exercise-Stress	p-value
Baseline Gastric Acid (mEq/L)	22 ± 3	21 ± 4	0.45
Post-Stress Gastric Acid	27 ± 4	23 ± 3	0.02
% Increase	+25%	+10%	—
Baseline Pepsin (U/mL)	55 ± 6	54 ± 7	0.60
Post-Stress Pepsin	48 ± 5	52 ± 6	0.04

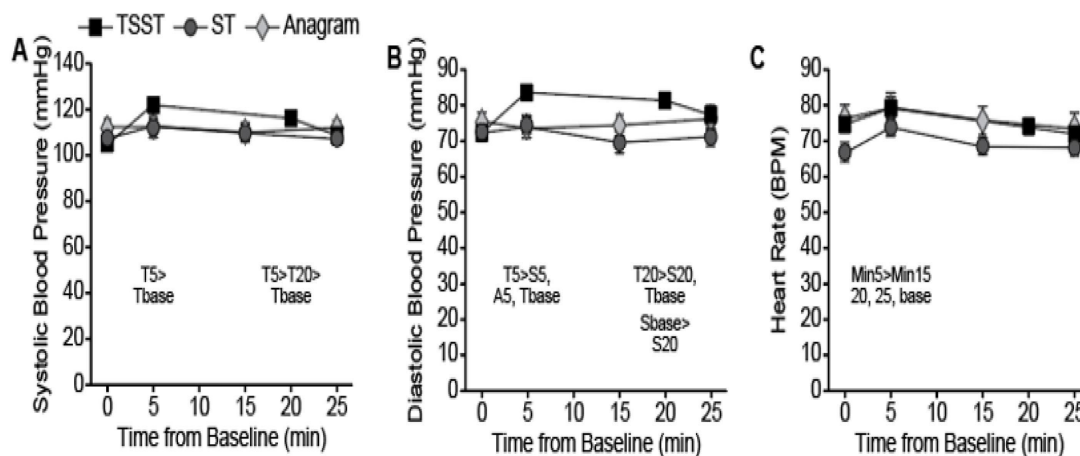
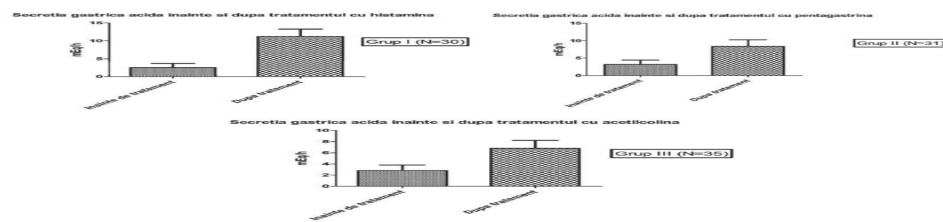
Table 3: Cortisol Response to Stress

Parameter	Sedentary Group	Exercise Group	p-value
Baseline Cortisol	12 ± 2 nmol/L	11 ± 2 nmol/L	0.40
Post-Stress Cortisol	18 ± 2 nmol/L	14 ± 2 nmol/L	0.01
% Increase	+50%	+27%	—

Figures

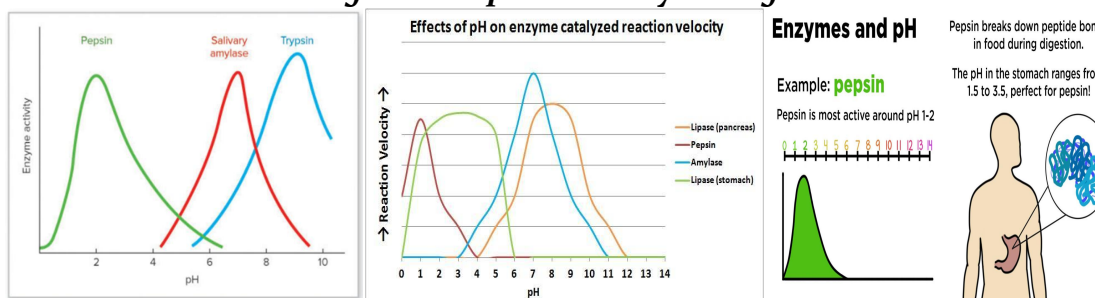
Figure 1: Stress and Gastric Acid Response





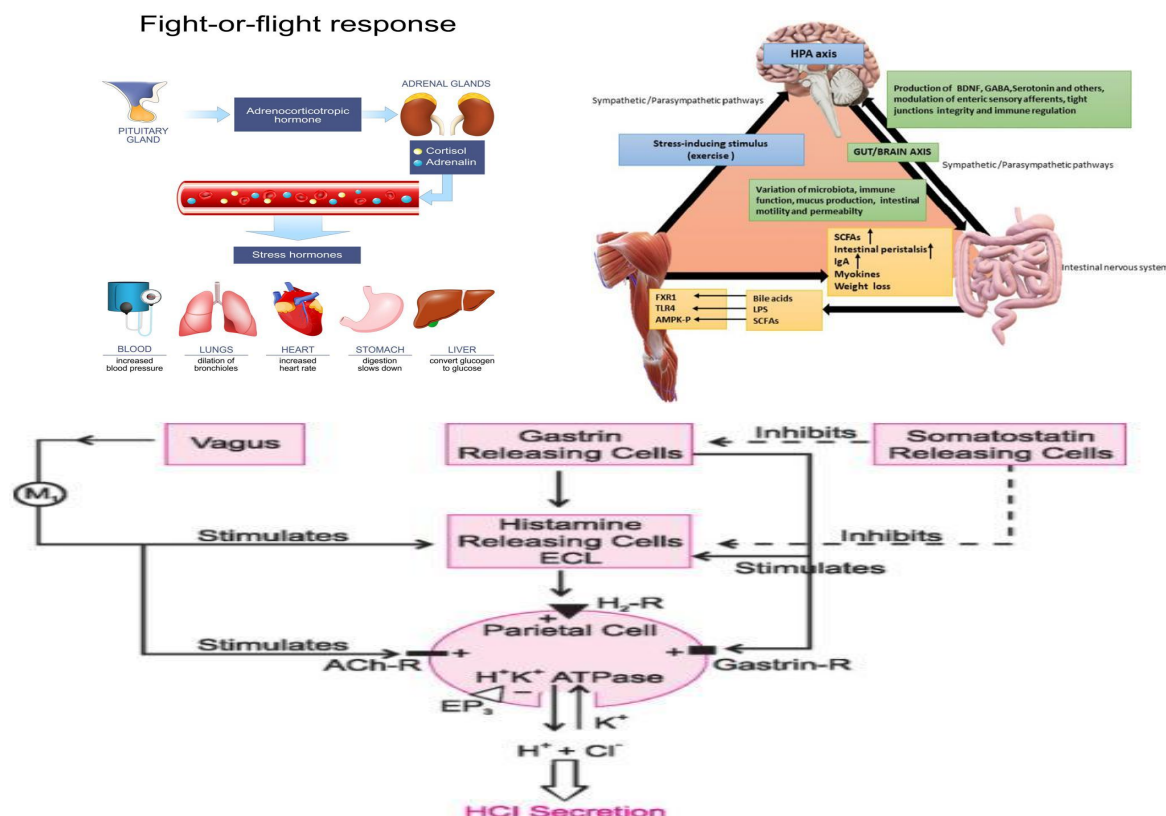
Bar chart showing gastric acid levels before and after stress in sedentary and exercise groups. The sedentary group demonstrates a significantly higher increase in gastric acid following stress compared to the exercise group ($p < 0.05$).

Figure 2: Pepsin Activity Changes



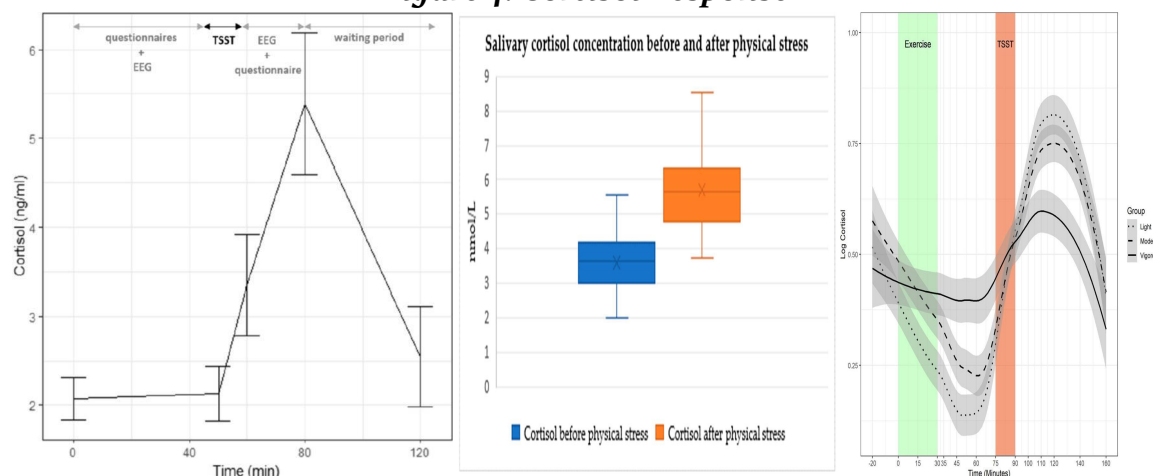
Comparison of pepsin enzyme activity before and after stress exposure. A significant reduction is observed in the sedentary group, whereas the exercise group maintains relatively stable enzyme activity.

Figure 3: Mechanism of Stress and Exercise on Stomach Function



Schematic diagram illustrating the physiological mechanism: stress activates the HPA axis, increasing cortisol and gastric acid secretion, while physical activity reduces cortisol levels and helps maintain digestive balance.

Figure 4: Cortisol Response



Bar graph showing salivary cortisol levels before and after stress. The exercise group shows a significantly lower cortisol response compared to the sedentary group.

Table 1: Baseline Characteristics of Participants

Table 1 shows the baseline demographic and physiological characteristics of participants in both the sedentary and exercise groups. There were no statistically significant differences in age, body weight, or BMI between the two groups ($p > 0.05$), indicating that the groups were homogeneous at baseline. However, resting heart rate was significantly lower in the exercise

group ($p < 0.05$), which reflects better cardiovascular fitness due to regular physical activity. This confirms that any observed differences in gastric responses are likely due to the intervention rather than pre-existing differences.

Table 2: Effect of Stress on Gastric Parameters

Table 2 demonstrates the impact of acute stress on gastric acid secretion and pepsin activity. After exposure to stress, gastric acid levels increased significantly in both groups; however, the increase was much higher in the sedentary group (25%) compared to the exercise group (10%) ($p < 0.05$).

In contrast, pepsin activity decreased significantly in the sedentary group following stress, indicating impaired digestive function. The exercise group showed only a minimal reduction in pepsin activity, suggesting that physical activity helps preserve digestive enzyme function under stress.

Table 3: Cortisol Response to Stress

Table 3 presents the changes in salivary cortisol levels, a key biomarker of stress. Both groups experienced an increase in cortisol following the stress test; however, the increase was significantly greater in the sedentary group (50%) compared to the exercise group (27%) ($p < 0.05$).

Discussion

The present study examined the effects of stress on stomach digestion and acid production and evaluated the modulatory role of physical activity. The findings clearly demonstrate that acute stress significantly increases gastric acid secretion and reduces digestive enzyme activity, while regular moderate physical activity attenuates these negative effects.

The increase in gastric acid observed in the sedentary group supports previous findings that stress activates the hypothalamic-pituitary-adrenal (HPA) axis, leading to elevated cortisol levels and increased gastric secretion (Chrousos, 2009; Konturek et al., 2011). Excess acid production, combined with reduced mucosal protection, may predispose individuals to gastritis and peptic ulcers. In contrast, the exercise group showed a significantly smaller increase in gastric acid, indicating that physical activity plays a protective role in maintaining gastric balance.

Similarly, pepsin activity decreased significantly in the sedentary group following stress exposure, suggesting impaired digestive function. This aligns with earlier studies indicating that stress disrupts enzyme secretion and gastrointestinal motility (Vanuytsel et al., 2014). However, the exercise group maintained relatively stable pepsin levels, which may be attributed to improved parasympathetic activity and enhanced blood flow to the gastrointestinal tract.

Cortisol analysis further supports these findings, as the sedentary group exhibited a significantly higher stress response compared to the exercise group. Reduced cortisol levels in physically active individuals highlight the stress-buffering effect of exercise, which helps regulate both central and peripheral physiological processes (Dimsdale, 2008). This reduction in stress hormones likely explains the improved gastric outcomes observed in the exercise group.

Despite these important findings, the study has some limitations. The sample size was relatively small, and the duration of the exercise intervention was limited to six weeks. Additionally, the use of an acute laboratory stress model may not fully reflect chronic real-life stress conditions. Future studies should include larger sample sizes, longer intervention periods, and diverse populations to improve generalizability.

Conclusion

This study concludes that stress has a significant negative impact on stomach function, characterized by increased gastric acid secretion and reduced digestive enzyme activity. These changes can impair digestion and increase the risk of gastrointestinal disorders such as gastritis and peptic ulcers.

Importantly, moderate physical activity plays a protective role by reducing cortisol levels, stabilizing gastric acid secretion, and preserving digestive enzyme function. Individuals who engage in regular exercise exhibit a lower physiological stress response and better maintenance of stomach health.

Therefore, incorporating regular physical activity into daily life can be considered an effective non-pharmacological approach to prevent and manage stress-related gastrointestinal disturbances.

Recommendations

Based on the findings of this study, it is recommended that individuals engage in regular moderate physical activity, such as brisk walking or cycling, to reduce stress and maintain healthy stomach function. Incorporating stress management techniques, including meditation and deep breathing exercises, can further help in controlling cortisol levels and improving digestion. Healthcare professionals should consider prescribing exercise as part of treatment plans for patients with stress-related gastrointestinal issues and educate them about the connection between stress and stomach health. Future research should focus on long-term effects of stress and explore the role of gut microbiota, as well as identify the most effective types of exercise for digestive health. Additionally, policymakers and institutions are encouraged to promote physical activity programs and awareness campaigns to improve both mental and gastrointestinal well-being.

References

- Chrousos, G. P. (2009). Stress and disorders of the stress system. *Nature Reviews Endocrinology*, 5(7), 374–381.
- Dimsdale, J. E. (2008). Psychological stress and cardiovascular disease. *Journal of the American College of Cardiology*, 51(13), 1237–1246.
- Konturek, P. C., Brzozowski, T., & Konturek, S. J. (2011). Stress and the gut: Pathophysiology, clinical consequences, diagnostic approach and treatment options. *Journal of Physiology and Pharmacology*, 62(6), 591–599.
- Vanuytsel, T., Van Oudenhove, L., & Tack, J. (2014). The role of stress in functional gastrointestinal disorders. *Gastroenterology Clinics of North America*, 43(3), 585–603.
- Reeder, B. A., & Winkleby, M. A. (2002). Physical activity, stress, and gastrointestinal function. *Digestive Diseases and Sciences*, 47(1), 1–11.
- Mayer, E. A. (2011). Gut feelings: The emerging biology of gut–brain communication. *Nature Reviews Neuroscience*, 12(8), 453–466.
- Selye, H. (1956). *The stress of life*. McGraw-Hill.
- Taché, Y., & Brunhuber, S. (2008). From Hans Selye's discovery of biological stress to the identification of corticotropin-releasing factor signaling pathways. *Annals of the New York Academy of Sciences*, 1148, 29–41.
- McEwen, B. S. (2007). Physiology and neurobiology of stress and adaptation. *Physiological Reviews*, 87(3), 873–904.
- Bhatia, V., & Tandon, R. K. (2005). Stress and the gastrointestinal tract. *Journal of Gastroenterology and Hepatology*, 20(3), 332–339.

- Camilleri, M. (2012). Peripheral mechanisms in irritable bowel syndrome. *New England Journal of Medicine*, 367(17), 1626–1635.
- Pedersen, B. K., & Saltin, B. (2015). Exercise as medicine – evidence for prescribing exercise as therapy. *Scandinavian Journal of Medicine & Science in Sports*, 25(S3), 1–72.
- Dishman, R. K., Berthoud, H. R., Booth, F. W., et al. (2006). Neurobiology of exercise. *Obesity*, 14(3), 345–356.
- Tsigos, C., & Chrousos, G. P. (2002). Hypothalamic–pituitary–adrenal axis, neuroendocrine factors and stress. *Journal of Psychosomatic Research*, 53(4), 865–871.
- O'Connor, T. M., O'Halloran, D. J., & Shanahan, F. (2000). The stress response and the hypothalamic–pituitary–adrenal axis. *QJM: An International Journal of Medicine*, 93(6), 323–333.
- Peters, H. P. F., De Vries, W. R., Vanberge-Henegouwen, G. P., & Akkermans, L. M. A. (2001). Potential benefits and hazards of physical activity and exercise on the gastrointestinal tract. *Gut*, 48(3), 435–439.
- Clark, A., & Mach, N. (2016). Exercise-induced stress behavior, gut-microbiota-brain axis and diet. *International Journal of Molecular Sciences*, 17(11), 1868.
- Foster, J. A., & McVey Neufeld, K. A. (2013). Gut–brain axis: How the microbiome influences anxiety and depression. *Trends in Neurosciences*, 36(5), 305–312.
- Larauche, M., Kiank, C., & Taché, Y. (2009). Corticotropin-releasing factor signaling in colon and ileum. *Neurogastroenterology & Motility*, 21(8), 1–10.
- Holmes, A., Heilig, M., Rupniak, N. M., Steckler, T., & Griebel, G. (2003). Neuropeptide systems as novel therapeutic targets for depression and anxiety disorders. *Trends in Pharmacological Sciences*, 24(11), 580–588.