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PSYCHOLOGICAL STRESS AND CANCER PROGRESSION

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ABSTRACT

Background: Psychological stress, particularly in its chronic form, is increasingly recognized as a factor influencing cancer progression through complex neuroendocrine, immunological, and molecular mechanisms. Chronic activation of the hypothalamic-pituitary-adrenal (HPA) axis and the sympathetic nervous system (SNS) leads to dysregulated cortisol and catecholamine levels, which suppress anti-tumour immunity, promote angiogenesis, and enhance tumour cell invasiveness and metastasis.
Aim: To synthesise current evidence on the biological mechanisms linking psychological stress to cancer progression and to highlight the therapeutic potential of psychosocial interventions in oncology.
Material and methods: A narrative literature review was conducted using the PubMed database. Relevant preclinical and clinical studies published between 2000 and 2024 were selected to examine the influence of psychological stress on cancer biology and disease progression.
Results: Chronic stress contributes to cancer growth, metastasis, and therapy resistance through hormonal imbalance, immune suppression, and chronic inflammation. Disruption of the tumour microenvironment further facilitates malignant progression. Studies particularly emphasize these effects in breast and ovarian cancers. Psychosocial interventions such as cognitive behavioural therapy (CBT) and mindfulness-based stress reduction (MBSR) have demonstrated benefits in both psychological well-being and biological markers of disease activity.
Conclusions: Psychological stress significantly impacts cancer progression through multiple biological pathways. Integrating stress-reducing interventions into standard oncological care may enhance patient outcomes and offer a complementary strategy in cancer management.

KEYWORDS

Psychological Stress, Cancer Progression, HPA Axis, Chronic Inflammation, Psychosocial Intervention

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Introduction

Psychological stress is defined as a complex response of the body to stimuli perceived as a physical or emotional threat, which activates a number of neuroendocrine processes aimed at restoring homeostasis. The literature distinguishes between two basic types of stress: acute and chronic. Acute stress is the body's adaptive response to short-term and intense stimuli, leading to activation of the hypothalamic-pituitary-adrenal (HPA) axis and the sympathetic nervous system (SNS), resulting in, among other things, increased secretion of cortisol and catecholamines [1]. Chronic stress, on the other hand, resulting from prolonged exposure to psychological or social stressors, may lead to lasting alterations in the function of these systems and is associated with a range of negative health consequences [1, 2].

The pathophysiology of stress includes activation of the aforementioned neuroendocrine axes, resulting in effects on immune function, inflammatory processes, metabolism and cardiovascular function. Cortisol, with its immunosuppressive effect, can inhibit NK cell and T cell activity, impairing the body's ability to fight cancer cells [2, 3]. Additionally, chronic activation of the sympathetic nervous system can lead to the promotion of angiogenesis, tumour cell invasiveness and their ability to metastasise [3].

In recent years, there has been growing interest in the relationship between chronic stress and the development of chronic diseases, including cancer. Numerous experimental and clinical studies have indicated that stress may influence cancer initiation, progression, and the efficacy of oncological treatment [3, 4]. The complex interaction between the nervous, endocrine and immune systems creates conditions that favour not only the development, but also a more aggressive disease course.

The aim of this paper is to review the current knowledge of the biological mechanisms linking psychological stress to cancer progression. This review includes the effects of chronic stress on the immune response, the tumour microenvironment, molecular mechanisms promoting metastasis and potential therapeutic implications.

Psychological Stress and the Neuroendocrine System

Psychological stress, especially in its chronic form, plays an important role in the activation of two major neuroendocrine axes: the hypothalamic-pituitary-adrenal (HPA) axis and the sympathetic nervous system (SNS). Both of these pathways not only influence immune function, but may also modulate processes involved in tumour initiation, progression and metastasis.

The HPA axis is the body's main stress response pathway. In response to stress signals, the hypothalamus secretes corticolibrin (CRH), which stimulates the pituitary gland to release adrenocorticotropin (ACTH). ACTH acts on the adrenal cortex to stimulate the secretion of glucocorticoids, mainly cortisol [5]. Under conditions of chronic stress, this system is dysregulated - both overactivation and abnormal rhythmicity of cortisol secretion are observed. A review of the literature indicates that cancer patients have significant dysregulation of the HPA axis - manifested, among other things, elevated serum cortisol levels and a flattened diurnal rhythm [5].

In parallel, activation of the sympathetic nervous system (SNS) in response to stress leads to the release of catecholamines - adrenaline and noradrenaline - from nerve endings and the adrenal medulla. Catecholamines interact with cells via adrenergic receptors, particularly β-adrenergic receptors, which can lead to increased angiogenesis, tumour cell invasion and metastasis [6]. Experimental studies have shown that chronic stress in mice with breast cancer significantly increased the number of lymph node and lung metastases. Importantly, administration of a non-selective β-blocker (propranolol) abolished this effect, indicating a key role for catecholamines in the metastatic process [6].

The dysfunction of the HPA and SNS axis under chronic stress is also closely related to the deregulation of diurnal rhythms, including the rhythm of cortisol secretion. In oncology patients, these disorders are common and have important clinical consequences, ranging from effects on quality of life to worsening of treatment tolerance and reduced survival time [7].

In ovarian cancer patients, a flattened diurnal rhythm of cortisol and elevated nocturnal concentrations of this hormone before treatment have been shown to be associated with shorter survival. A study of 113 women with ovarian cancer found that each standard deviation unit increase in nocturnal cortisol was associated with a 46% higher risk of death. Patients with high nocturnal cortisol levels survived an average of 3.3 years, compared to 7.3 years for patients with low levels of the hormone. In addition, elevated levels of interleukin-6 (IL-6) in the peritoneal fluid were correlated with each of the cortisol parameters measured, suggesting a link between HPA axis dysregulation and inflammation within the tumour [8].

In summary, chronic stress leads to persistent changes in the functioning of neuroendocrine axes, which in turn influence the biological environment that promotes cancer progression. Specifically, impaired cortisol secretion and excessive sympathetic nervous system activity may promote angiogenesis, suppress anti-tumour immunity and promote metastasis, making these pathways potential therapeutic targets.

Immunomodulatory Effects of Stress in Cancer

Psychological stress, especially when it takes on a chronic character, leads to profound changes in the functioning of the immune system. These changes play an important role in the process of cancer progression, affecting the body's anti-tumour response and the nature of the tumour microenvironment.

1) Effects of stress on the immune system: suppression of NK cells, T lymphocytes, altered cytokine profile

Chronic stress promotes immunosuppression by suppressing the function of cytotoxic T lymphocytes and natural killer (NK) cells, which constitute the first line of anti-tumour defence. The activity of these cells is significantly modulated by stress hormones such as glucocorticoids and catecholamines, which inhibit their cytotoxicity and ability to recognise and eliminate tumour cells [9, 10]. At the same time, there is an imbalance of cytokines - the production of pro-inflammatory cytokines, such as IL-6 and TNF-α, increases, while the production of cytokines that promote the cellular response, such as IL-2, decreases [4]. This change in cytokine profile promotes pro-tumorigenic inflammation and inhibits an effective immune response.

2) Chronic inflammation as a mediator between stress and cancer progression

One of the main mechanisms linking stress to cancer development is chronic low-grade inflammation. Stress disrupts signalling on the neuroendocrine-immune axis, leading to persistent activation of immune cells and increased secretion of pro-inflammatory cytokines [10]. Persistent inflammation creates an environment conducive to DNA damage, cell immortalisation and angiogenesis, which can lead to tumour initiation and progression [11]. Additionally, stress-related inflammation can promote tumour cell migration and metastasis by affecting cellular signal transduction pathways and remodelling the extracellular matrix.

3) Role of stress in the tumour microenvironment

The tumour microenvironment (TME) is a dynamic ecosystem of tumour cells, fibroblasts, immune cells and blood vessels. Chronic stress affects this complex system by increasing accumulation of immunosuppressive and pro-tumorigenic factors. Increased levels of catecholamines and glucocorticoids in the TME can lead to increased expression of factors that promote tumour cell survival, angiogenesis and resistance to chemotherapy [12]. Of particular relevance is the relationship between stress and the development of multidrug resistance (MDR). Stressors present in the tumour microenvironment can induce the expression of transport proteins (e.g. P-gp) that remove anticancer drugs from cells, significantly reducing the efficacy of therapy [13].

In summary, chronic stress influences tumour progression by suppressing the immune response, inducing chronic inflammation and modifying the tumour microenvironment. Understanding these mechanisms opens new perspectives for cancer therapy, including psychological and pharmacological interventions that can modulate the stress response.

Stress Hormones and Tumor Biology

Psychological stress, especially when chronic, has a significant impact on tumour biology through the action of stress hormones such as cortisol and catecholamines (adrenaline and noradrenaline). These hormones have a direct effect on tumour cells, modulating the processes of proliferation, apoptosis, angiogenesis and activating signalling pathways that promote tumour progression.

1) Effects of cortisol and catecholamines directly on tumour cells

Catecholamines, through activation of β-adrenergic receptors, increase tumour cell invasiveness, including by regulating the expression of matrix metalloproteinases (MMPs), which facilitates the penetration of tumour cells through the extracellular matrix. In studies in ovarian cancer models, β-adrenergic signalling was shown to significantly increase the invasive capacity of tumour cells through the regulation of MMPs [14]. In addition, catecholamines have been shown to have an anti-apoptotic effect on tumour cells, which can lead to resistance to chemotherapeutic drugs [15].

2) Regulation of angiogenesis (e.g. by VEGF), proliferation and apoptosis

Chronic stress increases levels of catecholamines, which stimulate the secretion of vascular endothelial growth factor (VEGF) by tumour cells, promoting angiogenesis. Studies in ovarian cancer models have shown that chronic stress led to increased angiogenesis through activation of the plexinA1/VEGFR2-JAK2-STAT3 signalling pathway [16]. In addition, activation of β-adrenergic receptors by catecholamines increased tumour cell proliferation and inhibited apoptosis, contributing to tumour progression [17].

3) Signalling pathways activated by stress: e.g. β-adrenergic, NF-κB, STAT3

Stress activates a number of signalling pathways that promote tumour progression. Activation of β-adrenergic receptors leads to the activation of NF-κB and STAT3 pathways, which regulate the expression of genes related to proliferation, angiogenesis and immunosuppression. Studies have shown that chronic stress increases NF-κB and STAT3 activity, which contributes to the development and progression of cancers such as gastric and colorectal cancer [18].

4) Potential for pharmacological intervention (e.g. beta-blockers)

The use of beta-blockers, which antagonise β-adrenergic receptors, shows therapeutic potential in oncology. Studies indicate that β-blockers may inhibit angiogenesis, proliferation and metastasis of tumours by blocking the action of catecholamines. A review of the literature highlights that beta-blockers may slow tumour growth and improve the efficacy of anticancer therapies [19]. Additionally, the use of beta-blockers may have a beneficial effect on the psychological state of cancer patients, reducing the stress associated with diagnosis and treatment [20].

Evidence from Clinical and Preclinical Studies

Both preclinical and clinical studies are providing increasing evidence of a link between psychological stress and cancer progression. In animal models, chronic stress has been shown to enhance tumour growth and increased incidence of metastasis. This effect is associated with activation of the hypothalamic-pituitary-adrenal (HPA) axis and the sympathetic nervous system, leading to increased levels of cortisol and catecholamines. These hormones affect the tumour microenvironment, promoting angiogenesis, invasion and immunosuppression [6, 21]. For example, in a mouse model of ovarian cancer, psychosocial stress was associated with increased angiogenesis and metastasis through activation of the β-adrenergic pathway [6].

Cohort studies in humans show a correlation between high levels of stress and poorer prognosis in various types of cancer. Chronic stress, depression and anxiety are associated with shorter survival times and accelerated disease progression, particularly in breast, ovarian, prostate and melanoma cancers [22-24]. However, the interpretation of these results faces methodological difficulties. Stress is subjective in nature and its impact may be modified by individual predisposition, the presence of psychiatric disorders, social support or socioeconomic status. In addition, stress may indirectly influence the course of the disease through reduced adherence to treatment, adverse lifestyle changes and impaired immunity [25, 26].

The strongest evidence for an association between stress and tumour progression relates to breast and ovarian cancer. In these cases, stress is associated with increased levels of pro-inflammatory cytokines, increased angiogenesis and changes in gene expression associated with tumour cell invasiveness [6, 23, 27]. Similar but less clear-cut relationships have also been observed in prostate cancer and melanoma [24, 25].

Psychological Interventions and Cancer Outcomes

A growing body of evidence suggests that psychological interventions can benefit not only the quality of life of cancer patients, but also the course of the disease. Cognitive behavioural therapy (CBT), mindfulness-based stress reduction (MBSR) techniques and structured social support reduce anxiety, depression and stress symptoms, resulting in improvements in patients' psychological well-being and physical functioning [28-30]. In breast cancer patients, participation in psychological support groups has been associated with improved quality of life, better adherence to treatment and, in some studies, prolonged survival [22].

Clinical studies also point to biological effects of psychological interventions. Participation in MBSR or CBT programmes was associated with reduced levels of pro-inflammatory cytokines (e.g. IL-6, TNF-α), decreased HPA axis activity and reduced expression of genes related to cellular stress and inflammation [31-33]. In a randomised trial involving women with breast cancer, a CBT-based intervention led to beneficial changes in leukocyte gene expression profiles and stabilisation of telomere length, indicating potential modulation of biological processes associated with ageing and cancer progression [32].

The use of a holistic approach in oncology, including both pharmacological treatment and psychological support, is increasingly recognised as an important part of comprehensive patient care. Interventions targeting mental health can enhance immune function, improve the ability to cope with the disease and reduce the risk of depression, which is itself associated with a poorer prognosis [34]. Therefore, the integration of psychology into routine oncology care is becoming increasingly justified on the basis of both clinical and biological research findings.

Limitations and Future Perspectives

Despite a growing body of evidence suggesting a link between psychological stress and the course of cancer, current research faces important methodological limitations. First of all, many are observational in nature, precluding definitive conclusions about causality. The full heterogeneity of the study populations - such as differences in cancer type, disease stage, age, gender or level of social support - is often not taken into account, limiting the generalisability of the results [35, 36]. Additionally, subjective ways of measuring stress and variability in definitions of psychological interventions make it difficult to compare studies and meta-analyse them [37].

Therefore, there is an urgent need for well-designed translational and prospective studies that integrate biological with psychosocial research approaches. Future studies should include standardised interventions, measurement of stress biomarkers and evaluation of long-term clinical outcomes [38]. Biomarkers such as salivary cortisol, levels of pro-inflammatory cytokines (IL-6, TNF-α), or the expression profile of stress-related genes - which may act as prognostic and predictive indicators in the future - could play a particular role here [39-41].

In the same time, new therapeutic options are being developed that target physiological stress pathways. β-adrenergic receptor inhibitors (β-blockers) have shown preliminary anti-tumour effects in some observational studies, particularly in the context of breast, ovarian and melanoma cancer [42-44]. Other approaches include pharmacological modulation of the HPA axis and the integration of psychoneuroimmunological therapies as support for cancer treatment. Ultimately, the future of cancer treatment may include both biologically targeted and psychological interventions - as part of an integrated, holistic approach to the cancer patient.

Conclusions

Available preclinical and clinical data indicate that psychological stress may influence the course and progression of cancer through a number of interrelated biological mechanisms, such as activation of the hypothalamic-pituitary-adrenal (HPA) axis, excessive stimulation of the sympathetic nervous system, chronic inflammation and impaired immune response [6, 45, 46]. Stress promotes angiogenesis, tumour cell invasion, metastasis and impaired immune surveillance, which can lead to faster disease progression and a poorer prognosis [45, 47].

Therefore, the psychological aspect should be an integral part of oncology patient care. Ignoring a patient's psychological burden may not only impair quality of life, but also negatively affect treatment outcomes through reduced adherence to therapy, increased susceptibility to side effects and weakened immune mechanisms [48, 49]. Therefore, systematic assessment of stress, anxiety and depression should be included in the standard diagnostic and therapeutic management in oncology.

Incorporating psychotherapy - particularly cognitive behavioural therapy - and stress-reduction techniques, such as mindfulness training, into the comprehensive treatment of patients with cancer may have significant benefits in both quality of life and potential biological parameters related to prognosis [22, 50, 51]. In the future, an integrated biopsychosocial approach to cancer treatment may form the foundation of personalised cancer medicine, which accounts not only for tumour characteristics, but also the mental state and emotional needs of the patient.

Disclosure

This manuscript is a narrative literature review and does not present original clinical or experimental data. All information was obtained from publicly available peer-reviewed sources.

Supplementary Materials

No supplementary materials are provided with this article.

Author Contributions

Conceptualization, [Kwiatkowska Agnieszka, Khiralla-Gawlik Sandra]; literature search and analysis, [Konrad Strużek, Basak Aleksandra]; writing—original draft preparation, ; [Strzelecka Aleksandra, Tracz Wiktor, Mączka Ewelina]; writing—review and editing, [Świercz Patrycja, Teper Kinga, Agnieszka Kwiatkowska]. The authors has read and agreed to the published version of the manuscript.

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