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Perspective

The Influence of Sleep Disruption on the Immune Landscape of Metastatic Breast Cancer

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DESCRIPTION

Sleep plays a fundamental role in maintaining physiological homeostasis, with profound effects on metabolic, endocrine, and immune functions. Disruption of sleep, whether due to lifestyle factors, stress, or pathological conditions, can significantly impair immune regulation and inflammatory balance. In recent years, increasing attention has been given to the relationship between sleep quality and cancer progression. Among malignancies, Metastatic Breast Cancer (MBC) represents a particularly critical context, as the immune system's capacity to recognize and eliminate tumor cells is already compromised. Understanding how sleep disruption alters the immune landscape in metastatic breast cancer may offer insights into disease progression and therapeutic outcomes.

Melatonin, the hormone primarily secreted by the pineal gland during the night, serves as a crucial mediator between sleep and immune function. Beyond its role in regulating circadian rhythms, melatonin possesses potent antioxidant and immunomodulatory properties. It enhances the activity of Natural killer (NK) cells and T lymphocytes, modulates cytokine production, and protects immune cells from oxidative damage. In metastatic breast cancer, diminished nocturnal melatonin secretion caused by sleep disruption or exposure to light at night has been associated with increased tumor growth and metastasis. Experimental studies have shown that melatonin can inhibit estrogen receptor signaling and limit angiogenesis by downregulating Vascular Endothelial Growth Factor (VEGF). Thus, sleep loss not only deprives the immune system of a key regulator but also removes a natural inhibitor of tumor-promoting pathways.

At the cellular level, the impact of sleep disruption on the immune landscape extends to alterations in immune cell metabolism and function. Sleep deprivation can shift immune cell populations toward a more exhausted and dysfunctional phenotype. These exhausted T cells are less capable of mounting effective antitumor responses. Similarly, macrophages under sleep-deprived conditions tend to polarize toward the M2 phenotype, which promotes tissue repair and tumor progression rather than immune-mediated cytotoxicity. This skewed macrophage polarization contributes to the establishment of an immunosuppressive microenvironment favorable for metastatic dissemination.

Sleep disruption also influences the efficacy of immunotherapies and chemotherapy. Immune checkpoint inhibitors, which rely on reactivating T-cell function, may be less effective in patients with chronic sleep disturbances due to baseline immune exhaustion. Moreover, disrupted circadian rhythms can affect the pharmacokinetics and pharmacodynamics of anticancer drugs. Chronotherapy-administering treatment according to the body's circadian cycle-has been proposed as a potential approach to mitigate these effects. Ensuring optimal sleep hygiene could therefore be an essential component of integrative cancer care, enhancing the immune system's responsiveness and improving therapeutic outcomes.

The psychological burden of metastatic breast cancer further compounds the problem of sleep disruption. Anxiety, depression, and pain are prevalent in this patient population and contribute to poor sleep quality, creating a vicious cycle of stress-induced immune suppression. Chronic activation of the stress response increases glucocorticoid levels, which not only suppress immune function but also enhance tumor cell survival and resistance to apoptosis. Addressing psychological factors through Cognitive Behavioral Therapy for Insomnia (CBT-I), mindfulness-based interventions, or pharmacological treatments targeting sleep may thus have beneficial effects not only on quality of life but also on immune competence.

Emerging research using transcriptomic and single-cell sequencing technologies has begun to map how sleep disruption reprograms immune cell gene expression in cancer. In preclinical models of breast cancer, sleep fragmentation has been shown to alter the expression of genes involved in antigen presentation, cytokine signaling, and immune checkpoint regulation. These findings suggest that sleep loss may fundamentally reshape the immune architecture within the tumor microenvironment, reducing immune surveillance and facilitating metastatic spread. Future studies integrating immune profiling with longitudinal sleep assessments in patients will be crucial to elucidate causal

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mechanisms and identify potential biomarkers linking sleep disruption with immune dysfunction in metastatic breast cancer.

CONCLUSION

Sleep disruption exerts profound effects on the immune landscape of metastatic breast cancer through hormonal, inflammatory, and metabolic pathways. By impairing cytotoxic immune responses, promoting systemic inflammation, and fostering an immunosuppressive tumor microenvironment, sleep loss accelerates disease progression and undermines treatment efficacy. Recognizing sleep health as a critical component of cancer management could have significant implications for patient outcomes. Integrating sleep restoration strategies into oncologic care-through behavioral interventions, circadian rhythm regulation, and potentially melatonin supplementation may represent an underutilized yet promising approach to enhance immune resilience and improve prognosis in metastatic breast cancer.