From lifestyle to cancer: Cellular and molecular mechanisms of immunity and disease

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Abstract

Lifestyle-associated factors, including dietary patterns, physical inactivity, obesity, tobacco consumption, alcohol use, psychosocial stress, and environmental exposures, are increasingly recognized as critical determinants of immune regulation and cancer susceptibility. These modifiable factors influence host physiology through complex cellular and molecular processes that link metabolic imbalance, chronic inflammation, and immune dysfunction to disease initiation and progression. Emerging evidence indicates that prolonged exposure to adverse lifestyle conditions can disrupt immune homeostasis and promote a biological environment conducive to tumor development. At the cellular level, lifestyle-induced metabolic disturbances significantly alter the function and distribution of both innate and adaptive immune cells. Key immune populations such as macrophages, dendritic cells, natural killer cells, and T lymphocytes undergo functional reprogramming under conditions of chronic inflammation and metabolic stress. These changes may impair antigen presentation, reduce cytotoxic immune activity, and promote immune exhaustion, thereby weakening the body's ability to recognize and eliminate transformed cells. In parallel, sustained inflammatory signaling contributes to the establishment of a tumor-promoting microenvironment characterized by enhanced angiogenesis, immune suppression, tissue remodeling, and resistance to apoptosis.

At the molecular level, multiple signaling pathways act as central mediators linking lifestyle-associated exposures to immune dysregulation and oncogenic transformation. Prominent pathways include NF-κB, JAK/STAT, PI3K–AKT–mTOR, and MAPK, which regulate inflammatory responses, cell proliferation, survival, and metabolic adaptation. In addition, oxidative stress and mitochondrial dysfunction induced by lifestyle-related factors can lead to genomic instability and accumulation of somatic mutations. Epigenetic mechanisms—including DNA methylation, histone modifications, and the regulation of non-coding RNAs such as microRNAs and long non-coding RNAs—further modulate gene expression patterns associated with immune responses and tumorigenesis.

Recent studies also highlight the role of the gut microbiome and host metabolic reprogramming in shaping immune responses and influencing cancer risk. Lifestyle-driven alterations in microbial composition and metabolite production can impact immune signaling pathways and tumor–immune interactions. Collectively, these findings underscore the intricate relationship between lifestyle determinants, immune function, and cancer biology. A deeper understanding of these interconnected mechanisms may facilitate the identification of novel biomarkers and therapeutic targets. Integrating lifestyle-based preventive strategies with advances in immunology and molecular oncology could provide effective approaches for cancer prevention, early detection, and personalized treatment.

Keywords: Cancer; immunology; cancer biology; diseases.