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Exploring the Therapeutic Potential of Vitamin B3 in Cancer

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Abstract: Niacin (vitamin B3), comprising nicotinic acid and nicotinamide, is a precursor to the essential coenzymes NAD^+ and $NADP^+$, which play central roles in cellular redox reactions, energy metabolism, DNA repair, and epigenetic regulation. A growing body of research highlights the link between dietary niacin intake and the bioavailability of $NAD(P)^+$, implicating niacin in maintaining genomic stability and modulating age-related and metabolic processes. In the context of cancer, niacin deficiency is frequently observed and is associated with reduced treatment efficacy and increased genomic instability. Niacin supplementation has shown potential to improve cancer outcomes and mitigate chemotherapy side effects. Furthermore, niacin metabolism intersects with tryptophan catabolism and inflammatory pathways, suggesting broader roles in immune regulation and tumor progression. This review explores the multifaceted roles of niacin and its derivatives in health and disease, with particular emphasis on cancer biology, metabolic regulation, and therapeutic implications.

Keywords: Vitamin B3, Nicotinamide, Tumor biology, Cancer prevention, Cancer therapy.

Research Paper

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1. INTRODUCTION

Niacin, or vitamin B3, exists in two primary forms—nicotinic acid and nicotinamide—which serve as vital precursors for the biosynthesis of the coenzymes nicotinamide adenine dinucleotide (NAD^+) and nicotinamide adenine dinucleotide phosphate ($NADP^+$). These coenzymes are indispensable for a wide range of metabolic and redox reactions in cells. Beyond their metabolic roles, NAD^+ also functions as a co-substrate for several enzyme families, including poly (ADP-ribose) polymerases (PARPs) and sirtuins, which regulate gene expression, DNA repair, and chromatin remodeling.

Recent studies have emphasized the regulatory connection between dietary niacin intake and $NAD(P)^+$-dependent epigenetic enzymes, suggesting that niacin has a broader physiological role in maintaining genetic stability, metabolic homeostasis, and aging processes. Additionally, the interaction between PARP activity and SIRT1-dependent deacetylation appears to influence chromatin structure and function, affecting diverse biological pathways. These findings have led to renewed interest in niacin not only as an essential nutrient but also as a potential therapeutic agent for conditions ranging from cancer to metabolic and cardiovascular diseases [1–3].

2. DISCUSSION

2.1. Niacin Deficiency and Cancer Progression

Niacin deficiency is a common finding among cancer patients and has been correlated with poorer clinical outcomes, including reduced efficacy of treatment and increased susceptibility to DNA damage. As a key factor in energy metabolism and DNA repair, insufficient niacin levels can lead to genomic instability, impaired DNA repair mechanisms, and decreased apoptotic response to genotoxic stress. Supplementation has demonstrated potential in enhancing cancer cell death, reducing chemotherapy-induced toxicity, and possibly improving overall survival rates in specific patient populations [3, 4].

2.2. NAD^+, $NADP^+$, and Redox Balance in Cancer

Both NAD^+ and its phosphorylated form $NADP^+$ play critical roles in the metabolic reprogramming of cancer cells. $NADP^+$, particularly in its reduced form NADPH, contributes to antioxidant defense and supports biosynthetic demands, such as nucleic acid and lipid synthesis. NAD^+, meanwhile, is indispensable for DNA repair and metabolic reactions.

Notably, alterations in NAD⁺ metabolism are frequently observed in cancer, further reinforcing the importance of niacin in oncological contexts [5, 6].

2.3. Niacin and Immune–Inflammatory Modulation

Niacin also intersects with inflammatory pathways. Extracellular NAD^+ has been shown to function as a pro-inflammatory signaling molecule, enhancing granulocyte activation and chemotaxis. However, NAD^+ biosynthesis via NAMPT in mesenchymal stem cells contributes to anti-inflammatory effects, highlighting the dual roles of NAD^+ in immune regulation. NAD^+ precursors, such as nicotinamide riboside (NR), have been shown to lower levels of circulating inflammatory cytokines [7, 8].

2.4. Tryptophan Metabolism and Niacin Biosynthesis

In mammals, niacin is synthesized from tryptophan via the kynurenine pathway, involving enzymes such as tryptophan 2,3-dioxygenase (TDO) and indoleamine 2,3-dioxygenase (IDO) [9, 10]. Tryptophan catabolism has been identified as a significant regulator of tumor growth and immune evasion, influencing both cancer cell proliferation and anti-tumor immune responses [11]. Although TDO and IDO both metabolize tryptophan, they differ in substrate specificity and regulation. These enzymes are hemoproteins; whose activity depends on the redox state of their heme moieties [10, 12].

2.5. HemoProteins and HO-1 in Cancer

Hemoproteins play important roles in cancer progression, particularly in energy metabolism, cellular proliferation, and immune modulation. Cancer cells often upregulate heme biosynthesis and uptake, which facilitates mitochondrial respiration and growth [13, 14]. Niacin has been shown to induce heme oxygenase-1 (HO-1), an enzyme involved in heme degradation. While HO-1 is associated with anti-inflammatory and cardioprotective effects [15, 16], it also contributes to tumor progression and immune suppression in the tumor microenvironment (TME) via its enzymatic products—carbon monoxide, biliverdin, and ferrous iron [17, 18].

3. CONCLUSION

Niacin (vitamin B3), encompassing both nicotinic acid and nicotinamide, plays a multifaceted and essential role in human physiology, particularly through its conversion into the bioactive cofactors NAD and NADP. These molecules are critical for cellular redox reactions, energy metabolism, DNA repair, epigenetic regulation, and immune modulation. Increasing evidence links niacin status to cancer progression, treatment outcomes, and overall cellular health.

Niacin deficiency is not only common in certain cancer patients but may also exacerbate genomic instability, impair DNA repair, and promote tumor development. Conversely, supplementation has shown promise in improving therapeutic responses and reducing chemotherapy-induced toxicity. Niacin also impacts inflammation and tumor immunity through its influence on NAD+ metabolism and related pathways, including the activation of enzymes like sirtuins, PARPs, and HO-1.

Furthermore, the interplay between niacin metabolism and tryptophan catabolism—especially through enzymes like IDO and TDO—adds another layer of complexity in cancer biology, highlighting its influence on both tumor cell behavior and the immune microenvironment.

Overall, niacin is emerging as more than a basic nutritional requirement; it is a potential therapeutic agent influencing metabolic, genetic, and immunological aspects of disease. Understanding its broader biological roles could pave the way for novel strategies in cancer prevention and treatment, metabolic health, and aging-related conditions.

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