

Thyroid Disorders in Pregnancy: Maternal and Fetal Outcomes

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Introduction

While some studies suggest a protective role for melatonin against thyroid oxidative damage caused by carcinogens, more research is needed to fully understand the mechanisms involved and to determine the optimal use of melatonin as a preventive or therapeutic agent in thyroid cancer. The thyroid gland is a critical organ responsible for regulating metabolism, growth, and energy balance in the body. It is susceptible to damage from various factors, including exposure to carcinogens. Carcinogens are substances that can promote the formation of cancer by inducing oxidative stress, which can lead to DNA damage and mutations. Melatonin, a hormone primarily produced by the pineal gland, has emerged as a potential protective agent against oxidative damage in the thyroid gland. This article explores the mechanisms behind melatonin's protective effects and its potential as a therapeutic agent in preventing thyroid cancer. Oxidative stress occurs when there is an imbalance between the production of reactive oxygen species and the body's ability to neutralize them with antioxidants. ROS are highly reactive molecules that can damage cellular components, including DNA, proteins, and lipids. Chronic oxidative stress can lead to the development of cancer by promoting mutations in key genes involved in cell growth and proliferation. The thyroid gland is particularly vulnerable to oxidative stress due to its high metabolic activity and the presence of high levels of unsaturated fatty acids, which are susceptible to oxidation. Inflammation is closely linked to oxidative stress and carcinogenesis. Chronic inflammation can promote the production of ROS and reactive nitrogen species, leading to oxidative damage in tissues. Melatonin has anti-inflammatory properties and can suppress the production of pro-inflammatory cytokines and mediators, such as tumor necrosis factor, interleukin-6 and cyclooxygenase-2. By reducing inflammation, melatonin can indirectly reduce oxidative stress in the thyroid gland and mitigate the damaging effects of carcinogens. Its anti-inflammatory effects may also contribute to its cancer-preventive properties by inhibiting the growth and proliferation of cancer cells [1].

Description

Carcinogens, such as ionizing radiation, environmental toxins, and certain chemicals, can induce oxidative stress in the thyroid gland, leading to DNA damage and potentially cancerous changes in thyroid cells. Melatonin is a potent antioxidant that can neutralize ROS and protect cells from oxidative damage. It scavenges free radicals, such as hydroxyl radicals and singlet oxygen, thereby reducing oxidative stress and its damaging effects. Melatonin also stimulates the activity of antioxidant enzymes, such as superoxide dismutase, glutathione peroxidase, and catalase, which further enhance its antioxidant capacity. In addition to its direct antioxidant effects, melatonin has been shown to modulate the expression of genes involved in antioxidant defense and DNA repair. It can upregulate the expression of antioxidant enzymes and downregulate the

expression of pro-oxidant enzymes, thereby enhancing the cell's ability to cope with oxidative stress and maintain genomic stability [2].

Conclusion

Melatonin may also interact with enzymes involved in the metabolism of carcinogens, thereby altering their effects or enhancing their detoxification. For example, melatonin has been shown to inhibit the activity of cytochrome P450 enzymes, which are involved in the activation of certain carcinogens. By inhibiting these enzymes, melatonin may reduce the formation of reactive metabolites that can damage DNA and promote cancer development. Melatonin has emerged as a promising agent for protecting the thyroid gland against oxidative damage caused by carcinogens. Its antioxidant, anti-inflammatory, and immune-modulating effects, along with its ability to regulate cell proliferation and apoptosis, make it a potentially valuable therapeutic agent in preventing thyroid cancer. Further research is needed to elucidate the precise mechanisms of melatonin's protective effects and to determine its optimal use in clinical settings. Nonetheless, the evidence suggests that melatonin may offer a novel approach to reducing the risk of thyroid cancer in individuals exposed to carcinogens.

References

1. Szanto, Ildiko, Marc Pusztaszeri and Maria Mavromati. "H2O2 metabolism in normal thyroid cells and in thyroid tumorigenesis: Focus on NADPH oxidases." *Antioxidants* 8 (2019): 126.
2. Karbownik-Lewińska, Małgorzata and Agnieszka Kokoszko-Bilska. "Oxidative damage to macromolecules in the thyroid-experimental evidence." *Thyroid Res* 5 (2012): 1-6.

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