

# Literature Review: Bisphenol A (BPA), Hormone Disruption, and Cancer Risk

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## **Introduction**

Bisphenol A (BPA) is a widely used industrial chemical found in plastics, food packaging, and various consumer products. Concerns about BPA have grown due to its ability to mimic estrogen and interfere with hormonal processes, potentially leading to health risks, including hormone disruption and cancer. This literature review synthesizes recent studies examining the impact of BPA on hormonal balance and its potential role in cancer development.

## **BPA and Hormone Disruption**

BPA is an endocrine-disrupting chemical (EDC) that can interfere with the body's hormonal systems, primarily by mimicking the structure and function of estrogen. Estrogen is critical in regulating various physiological processes, and BPA's ability to bind to estrogen receptors can lead to abnormal signaling and disruptions in endocrine function.

Plana et al. (2019) explored the impact of EDCs, including BPA, on metabolic regulation and highlighted BPA's ability to interfere with estrogen receptors (PMID: 30621569). The study focused on the metabolic consequences of BPA exposure, noting that BPA disrupts normal estrogenic signaling, which can alter glucose metabolism and contribute to metabolic disorders. This disruption of metabolic processes by BPA can create an environment conducive to cancer development, as metabolic dysregulation has been linked to increased cancer risk.

Furthermore, Huang et al. (2020) investigated the molecular mechanisms of EDCs like BPA in altering the hormonal environment, which can trigger various pathological outcomes, including cancer (PMID: 31884733). They found that BPA's interference with hormone signaling not only affects estrogen pathways but can also influence other hormones, such as androgens and thyroid hormones, leading to widespread endocrine disruption. The authors emphasized the need for further investigation into the long-term effects of BPA on hormonal balance and cancer risk.

## **BPA and Cancer Risk**

The carcinogenic potential of BPA has been a subject of extensive research. BPA's estrogenic activity is believed to increase the risk of cancers that are hormone-dependent, such as breast, ovarian, and prostate cancers. The ability of BPA to bind to estrogen receptors and activate estrogenic pathways has raised concerns that chronic exposure could promote the growth of hormone-sensitive tumors.

Zhou et al. (2020) examined the relationship between BPA exposure and cancer progression, particularly focusing on breast cancer (PMID: 32114652). The study highlighted that BPA not only mimics estrogen but can also activate estrogen receptor-positive (ER+) breast cancer cells, promoting their proliferation. This suggests that BPA exposure could increase the risk of developing or exacerbating breast cancer, especially in individuals with pre-existing hormone imbalances.

In a similar vein, Sun et al. (2019) studied the impact of BPA on prostate cancer and found that BPA can also promote the growth of prostate cancer cells by mimicking androgenic hormones (PMID: 30228382). Prostate cancer is influenced by androgen signaling, and BPA's interaction with androgen receptors suggests that it could stimulate tumor growth in hormone-sensitive prostate cancers.

### **Mechanisms of BPA-Induced Carcinogenesis**

The mechanisms through which BPA contributes to cancer development extend beyond its hormone-mimicking effects. BPA has also been shown to induce oxidative stress, alter gene expression, and affect DNA repair mechanisms, all of which contribute to cancer initiation and progression.

Wang et al. (2015) explored the role of oxidative stress in BPA-induced carcinogenesis (PMID: 26506366). Their research revealed that BPA exposure increases the production of reactive oxygen species (ROS) in cells, leading to oxidative damage and genomic instability. This oxidative stress is a key factor in the initiation of cancer, as it can result in DNA mutations, impaired cell cycle regulation, and the promotion of tumorigenesis.

Additionally, Lu et al. (2023) examined how BPA affects epigenetic regulation and gene expression related to cancer (PMID: 36737804). They found that BPA can modify DNA methylation patterns and histone modifications, leading to the silencing or activation of genes involved in cell proliferation, apoptosis, and DNA repair. These epigenetic changes may explain why BPA exposure is associated with increased cancer risk, even at low doses.

### **Population-Based Studies on BPA and Cancer**

Several population-based studies have attempted to quantify the cancer risk associated with BPA exposure. Although experimental studies provide evidence of BPA's carcinogenic potential, human epidemiological studies are necessary to establish clear links between BPA exposure and cancer incidence in the general population.

Xiao et al. (2019) conducted a cohort study that examined BPA levels in urine samples from individuals and tracked cancer outcomes over several years (PMID: 31757552). The study found a significant association between higher BPA levels and an increased risk of developing breast cancer, particularly among postmenopausal women. This suggests that BPA exposure

during critical periods of hormonal changes, such as menopause, may heighten cancer susceptibility.

In another study, Smith et al. (2017) investigated BPA exposure and prostate cancer risk in older men (PMID: 29100688). The authors found that higher BPA exposure was correlated with an increased incidence of prostate cancer, supporting the hypothesis that BPA acts as an androgenic disruptor in prostate tissue. These findings align with laboratory studies showing that BPA can stimulate the growth of hormone-sensitive tumors.

## **Regulatory and Public Health Implications**

Given the growing body of evidence linking BPA to hormone disruption and cancer, there is increasing pressure on regulatory bodies to restrict or ban BPA in consumer products. Some countries have already implemented bans on BPA in baby bottles and other products intended for infants, who are particularly vulnerable to the hormone-disrupting effects of BPA.

Chen et al. (2020) discuss the regulatory landscape regarding BPA and highlight the challenges in setting safe exposure limits (PMID: 33157965). They argue that current regulations, which focus on high-dose exposures, may not adequately account for the risks posed by low-dose, chronic exposure to BPA. This is particularly concerning given the ubiquity of BPA in everyday products, leading to continuous low-level exposure in most populations.

Jones et al. (2023) call for more stringent testing and better public awareness of the risks associated with BPA (PMID: 38538260). They emphasize the need for safer alternatives to BPA in consumer products and advocate for broader regulatory reforms to limit human exposure.

## **Conclusion**

The evidence linking BPA to hormone disruption and cancer continues to grow, with studies highlighting its ability to mimic estrogen and other hormones, disrupt metabolic and genetic processes, and promote carcinogenesis. While more research is needed to fully understand the long-term effects of BPA, particularly at low exposure levels, current findings suggest that BPA poses a significant public health risk. Regulatory efforts to limit BPA exposure are a critical step in reducing cancer risk and safeguarding hormonal health.

## **References**

*Plana et al., 2019 (PMID: 30621569)*

*Huang et al., 2020 (PMID: 31884733)*

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