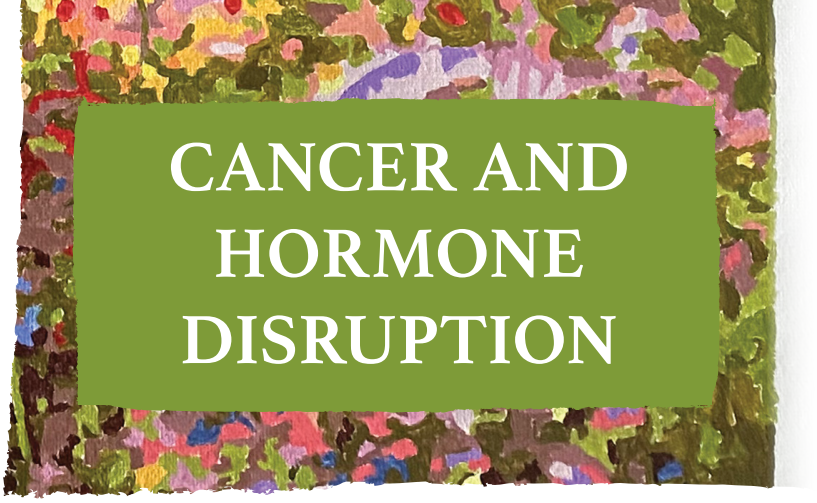
Cleveland's Center for Healing
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CANCER AND HORMONE DISRUPTION

Hormones that are well-known to have an impact on cancer include estrogen, progesterone, and testosterone. In addition to these more common hormones, insulin, thyroid hormones, and melatonin also play a role. The hormone cortisol, which has a significant impact on cancer, is addressed under the HPA Axis Cancer Root.

For cancers that are hormone-sensitive or hormone-dependent, their cells have hormone sensitive proteins on their surfaces called receptors. They link to hormones like a lock and a key making a connection. The hormone acts as a "key" opening the "lock" of the receptor, the cancer cells then grow and spread.

Typical hormones that we are all familiar with include estrogen and progesterone which can cause some types of breast, ovarian, and uterine cancers to grow. Testosterone can also help prostate cancer grow and spread. Hormones can drive cancer proliferation, which can lead to random genetic errors. With cancer cell production many cells are made over and over, errors can occur during cell formation and division that lead to mutations which can eventually lead to a malignancy of some type.

Estrogen is the hormone that drives cancers typically seen in breast, ovarian, and endometrial (uterine) cancers. But estrogen receptors are also involved in cancers of the stomach. Dysregulation of estrogen receptors play a role[1]. And hormones are also impacting cancers in the colon. Research supports the role of estrogen and its receptors in the initiation and progression of colorectal cancer. Estrogen effects can also be protective in colon cancer. Hormone replacement therapy (HRT) in postmenopausal women as well as consumption of soy reduces the incidence of colorectal cancer[2].

In healthy cells, one of the most common hormones involved in cancers is estrogen. This hormone helps with normal cell growth and function, such as in pregnancy, but when other factors are present, such as chronic inflammation from an unhealthy diet and other inflammatory drivers, toxin exposures, and even obesity, genetic changes can occur. Estrogen can then cause cancer cells to multiply. For example, estrogen can change and directly alter how cells repair their DNA, which makes it a more central factor in cancer's origin than simply promoting cancer cell proliferation.

Estrogen driven cancers can be stimulated by xenoestrogens. Xenoestrogens are covered under the Toxic Exposure Cancer Root in breast cancer. Please refer to that cancer root to learn more. Xenoestrogens are synthetic or natural chemical compounds that mimic estrogen and bind to estrogen receptors. They are also known as endocrine disrupting compounds. Exposure to Bisphenol A (BPA), a chemical found in many of our household products including cosmetics, and food and beverage containers, has been shown to increase the risk of breast cancer in women[3]. And prostate cancer in men[4]. Bisphenol A is a chemical compound used to make plastics and resins that began in the 1950s.

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Other hormones involved in cancer cell proliferation include the thyroid hormones: thyroid stimulating hormone (TSH), Free T4, and free T3. These hormones affect nearly every function of the body and are associated with thyroid cancer. Exposure to high doses of radiation or living near a nuclear accident site increases the risk of thyroid cancer. People with low iodine levels may be at higher risk for developing cancer of the thyroid after radiation exposure. Having a close relative with thyroid cancer, especially papillary thyroid cancer, is known to increase the risk of developing thyroid cancer. Having a benign thyroid condition, such as an enlarged thyroid (goiter) or thyroid nodules, can increase your risk for developing cancer. Most thyroid nodules are benign, but about 2–3 in 20 are cancerous. Other conditions that may increase your risk include acromegaly, a rare condition where the body produces too much growth hormone, and the autoimmune disease systemic lupus erythematosus. The risk of thyroid cancer can go up with obesity as well[5].

Melatonin, which is a neuro- hormone, also plays a role in cancer. The effects of melatonin, specifically its role in the sleep–wake cycle is well known, but in addition to that it has been identified as an antioxidant, an immunomodulator, and has a significant role in anti-inflammation. Melatonin has anticancer effects[6]. Patients in the Phase 1 Functional Oncology Program with Hyperion Functional Medicine are placed on appropriate doses of melatonin based on demonstrated research for their type of cancer.

INSULIN'S ROLE IN CANCER:

The insulin and the insulin growth factor signaling system plays a key role in the development and progression of many cancers. Insulin levels should not be overlooked in cancer patients. Insulin can promote tumor cell proliferation, cancer cell survival, migration, and invasion leading to metastasis by signaling through the insulin receptor and insulin growth factors. These signaling pathways can lead to: Abnormal protein synthesis, progression of the cancer cell cycle resulting in cancer cell growth, and prevention of apoptosis (natural cell death). Insulin plays a role in an increased risk of cancer in people with type 2 diabetes and obesity, related in part to hyperinsulinemia (elevated insulin levels), and because of the resulting development of insulin resistance. Insulin resistance occurs when cells in the muscles, fat, and liver don't respond well to insulin that should help regulate blood sugar levels. When this normal response to insulin goes away, cells can't easily absorb glucose from the blood, so the pancreas produces more and more insulin to help glucose enter the cells. This continues to occur over time, the cells may continue to resist insulin, and the pancreas may eventually wear out and be unable to produce enough insulin. This can lead to high blood sugar levels, and elevated hemoglobin A1c which can eventually reach diabetic levels. Hyperinsulinemia leads to increased expression of insulin-like growth factor expression. Increased insulin, and insulin growth factors levels are associated with tumor growth ***in vitro***, in animal models, and in epidemiological studies in humans[7]. In many cancer cells, the insulin receptor is overexpressed. In cancers where the insulin receptor is overexpressed, a selective growth advantage to cancer cells when exposed to insulin occurs. For this reason, patients with conditions of hyperinsulinemia (includes prediabetes, metabolic syndrome, obesity, type 2 diabetes before the pancreas is exhausted, polycystic ovary syndrome and type 1 diabetes) will have an increased risk of cancer. Cancer-related mortality is also increased in patients exposed to elevated insulin[8].

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Having too much insulin in the body, can contribute to the development of several cancers: Pancreatic cancer because hyperinsulinemia can cause the pancreas to produce more digestive enzymes, which can lead to inflammation and precancerous pancreatic cells. The risk of Breast, prostate, and colorectal cancers is increased as well with hyperinsulinemia. Hyperinsulinemia can allow tumor cells to absorb glucose and use it to fuel rapid growth. Studies have also shown that hyperinsulinemia can increase the risk of cancer mortality in participants without obesity, diabetes, or a history of cancer[9].

Symptoms of hyperinsulinemia include fatigue and increased hunger, as glucose remains in the bloodstream and isn't being converted into energy.

All patients in the phase 1 oncology program with Hyperion functional medicine have insulin levels tested initially and revaluated through the functional oncology program to monitor for elevations. Hemoglobin A1c and glucose levels are also evaluated which can indicate hyperinsulinemia and insulin resistance. Off label pharmaceuticals are used if indicated for specific cancers, such as metformin, if indicated to manage elevations in insulin, glucose and hemoglobin A1c that can drive cancer growth.

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