Volume 11, Issue 7, July – 2026 International Journal of Innovative Science and Research Technology
ISSN No:-2456-2165 https://doi.org/10.38124/ijisrt/26jul568

The Role of Dietary Patterns in Hormonal Regulation and Breast Cancer Development: A Comprehensive Review

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Publication Date: 2026/07/28

Abstract: Breast cancer is one of the leading causes of cancer-related morbidity and mortality among women worldwide. Growing evidence suggests that dietary patterns influence endocrine function and may contribute to breast cancer development through hormonal modulation. Estrogen plays a central role in the initiation and progression of hormone receptor-positive breast cancer, while progesterone and thyroid hormones participate in regulating mammary gland physiology and cellular proliferation. This review aims to critically evaluate the current evidence on the relationship between dietary patterns, hormonal regulation, and breast cancer development. Published literature from peer-reviewed journals was analyzed to assess the effects of Mediterranean, plant-based, Western, high-fat, and high-fiber dietary patterns on estrogen metabolism, progesterone, thyroid hormones, insulin signaling, and inflammatory pathways. The evidence indicates that healthy dietary patterns rich in fruits, vegetables, whole grains, legumes, nuts, and omega-3 fatty acids are associated with improved metabolic health, reduced chronic inflammation, and favorable hormonal balance, which may lower breast cancer risk. Conversely, Western dietary patterns characterized by high consumption of processed foods, saturated fats, refined carbohydrates, and excessive alcohol intake are associated with obesity, increased estrogen exposure, insulin resistance, and chronic inflammation, all of which contribute to breast cancer progression. Current evidence linking dietary factors with thyroid hormone alterations remains limited and inconsistent, with no conclusive evidence that commonly consumed foods, including millets, directly disrupt endocrine function or increase breast cancer risk in healthy individuals. Overall, this review highlights the importance of dietary patterns as modifiable lifestyle factors that influence hormonal regulation and breast cancer risk. Further large-scale prospective studies and randomized clinical trials are required to clarify causal relationships and identify evidence-based nutritional strategies for breast cancer prevention and management.

Keywords: *Breast Cancer, Dietary Patterns, Hormonal Regulation, Estrogen, Progesterone, Thyroid Hormones, Endocrine System, Nutrition, Mediterranean Diet, Breast Cancer Prevention.*

How to Cite: J. Vanessa Francesca; D. Kavi Roshni; Vidhyashree L.; Latangi R.; Santhanalakshmi V.; Dr. D. P. Sivasakti Balan; R. J. Thayumanaswamy (2026) The Role of Dietary Patterns in Hormonal Regulation and Breast Cancer Development: A Comprehensive Review. *International Journal of Innovative Science and Research Technology*, 11(7), 1403-1409. https://doi.org/10.38124/ijisrt/26jul568

I. INTRODUCTION

Breast cancer is a disease in which abnormal cells in the breast grow uncontrollably and may form a tumor, some of which can spread to other parts of the body if not detected early. Although the exact cause is not fully understood, several risk factors are known to increase its likelihood. Genetic mutations, particularly in the BRCA1 and BRCA2 genes, can be inherited and significantly raise the risk of breast and ovarian cancers. Hormonal influences, such as prolonged exposure to estrogen due to early menstruation, late menopause, or long-term hormone replacement therapy, also contribute. The risk is higher in women and increases with age, especially after 50. Lifestyle factors like alcohol consumption, obesity, and lack of physical activity further elevate the risk, while reproductive factors such as late pregnancy, not having children, or not breastfeeding may also play a role. Additionally, previous exposure to radiation, especially during youth, and having dense breast tissue can increase susceptibility. Despite these risks, certain common beliefs, such as the use of bras or deodorants causing breast cancer, are not supported by scientific evidence. Therefore, awareness of risk factors, adoption of a healthy lifestyle, and regular screening are essential for early detection and better outcomes (World Health Organization, 2023; American Cancer Society, 2022).

ISSN No:-2456-2165 https://doi.org/10.38124/ijisrt/26jul568

The hormonal basis of breast cancer is primarily linked to the influence of estrogen and progesterone, which regulate the growth and development of breast tissue. These hormones bind to specific receptors in breast cells, known as estrogen receptors (ER) and progesterone receptors (PR), stimulating cell division. When exposure to estrogen is prolonged or excessive, it can increase the chances of DNA damage during cell division, leading to abnormal cell growth and potentially cancer. Factors such as early onset of menstruation, late menopause, and hormone replacement therapy extend the duration of estrogen exposure, thereby raising the risk. In many breast cancers, especially hormone receptor-positive types, tumor growth is directly driven by these hormones. This understanding has led to effective treatments that target hormonal pathways, such as drugs that block estrogen receptors or reduce estrogen production. Thus, hormones play a crucial role not only in the development but also in the progression and treatment of breast cancer (American Cancer Society, 2022; National Cancer Institute, 2023).

Breast cancer in young women is less common, but it can occur due to a combination of genetic, hormonal, and lifestyle-related factors. One major reason is inherited genetic mutations, especially in the BRCA1 and BRCA2 genes, which significantly increase the risk at an early age. Hormonal influences also play a role, as higher levels of estrogen or increased sensitivity of breast cells to hormones can promote abnormal cell growth. In some cases, early onset of menstruation or certain hormonal conditions may contribute to this risk. Additionally, aggressive subtypes of breast cancer are more likely to occur in younger women, which can make the disease appear earlier and progress faster. Lifestyle factors such as obesity, lack of physical activity, alcohol consumption, and exposure to environmental pollutants may also increase susceptibility. Previous exposure to radiation therapy during childhood or adolescence is another known risk factor. Although many young women diagnosed with breast cancer may not have obvious risk factors, early awareness, regular self-examination, and timely medical consultation for unusual changes are important for early detection and better outcomes.

II. REVIEW OF LITERATURE

The association between thyroid hormones and the risk of breast cancer (BC) has been reported in epidemiological studies .A positive association has been reported between thyroxine (T4) and risk of BC, which is more pronounced in overweight and obese women.[Negative associations have been reported between triiodothyronine (T3) and BC among premenopausal women; in contrast, positive associations have been observed among postmenopausal women . It has been shown that in in vitro studies, thyroid hormones affect the growth of BC-derived cell lines , lung cancer , and glioblastoma . T4 has been shown to increase cell proliferation through the αvβ3 integrin receptor found on the plasma membrane of cells . In contrast, the effect of T3 on cell proliferation has not been well-established because it differs by the type of cell line used . These effects are important, since abnormalities in thyroid function tests have been observed in a variety of non-thyroidal illnesses, without preexisting thyroid or hypothalamic-pituitary disease . Furthermore, these abnormalities might change with body mass index (BMI) because thyroid hormones are involved in the regulation of various metabolic pathways (e.g., adaptive thermogenesis and glucose metabolism) that are relevant for resting energy expenditure and changes in body weight .

Research studies have shown that marital status is associated with differences in cancer risk and outcomes among women, with unmarried women (including those who are single, divorced, or widowed) generally having a slightly higher risk of certain cancers such as breast, ovarian, and uterine cancer compared to married women. This difference is not directly caused by marital status itself but is influenced by related factors such as reproductive patterns, since married women are more likely to have children earlier, which can offer some hormonal protection against certain cancers. In addition, married women often benefit from greater emotional, social, and financial support, which can lead to earlier detection, better access to healthcare, and improved adherence to treatment, thereby increasing survival rates. In contrast, unmarried women may experience higher levels of stress, delayed diagnosis, and reduced support during treatment, which can negatively affect outcomes. Overall, while marital status shows a statistical association with cancer risk and survival, it is the underlying social, biological, and lifestyle factors that play a more significant role.

The association between uterine disorders and breast cancer is largely influenced by common hormonal and biological factors rather than a direct causal link. Both the uterus and breast are sensitive to estrogen, and prolonged exposure to this hormone can contribute to conditions such as endometrial hyperplasia, uterine fibroids, and an increased risk of breast cancer. Factors like early onset of menstruation, late menopause, obesity, and use of hormone therapy can elevate estrogen levels, thereby increasing the likelihood of abnormalities in both organs. In addition, certain genetic predispositions and metabolic conditions may further connect the development of uterine problems and breast malignancies. Thus, the relationship reflects shared risk factors, particularly hormonal imbalance, rather than one disease directly leading to the other.

Uterine polyps, fibroids, and Polycystic Ovary Syndrome (PCOS) are closely linked to hormonal imbalance, particularly involving estrogen and progesterone. Research shows that uterine polyps develop due to abnormal overgrowth of the endometrial lining, often influenced by increased sensitivity to estrogen and altered hormone receptor activity. Similarly, uterine fibroids are strongly hormone-dependent tumors, where estrogen and progesterone promote the growth and proliferation of smooth muscle cells in the uterus. PCOS, on the other hand, is an endocrine disorder characterized by excess androgen levels, irregular ovulation, and prolonged estrogen exposure without adequate progesterone balance, which can lead to endometrial changes, hyperplasia, and increased risk of polyps. Studies also indicate that women with PCOS frequently exhibit associated conditions such as obesity and menstrual irregularities, which further contribute to a hyperestrogenic state and increase the

ISSN No:-2456-2165 https://doi.org/10.38124/ijisrt/26jul568

likelihood of developing uterine abnormalities like polyps and fibroids. Overall, these conditions are interconnected through a common pathway of hormonal dysregulation, particularly involving estrogen dominance and progesterone resistance, rather than occurring independently.

Estrogen plays a major role in influencing the risk of Breast cancer because it directly affects the growth and division of breast cells. Estrogen binds to receptors in breast tissue and stimulates cell proliferation; while this is a normal biological process, prolonged or excessive exposure increases the chance of DNA damage and mutations, which can lead to cancer. Women who experience longer lifetime exposure to estrogen—such as early onset of menstruation, late menopause, or hormone replacement therapy—have a higher risk because their breast cells are stimulated for a longer period. Conditions like Polycystic Ovary Syndrome, obesity, and certain hormonal imbalances can also elevate estrogen levels, further increasing risk. Additionally, some breast cancers are classified as estrogen receptor-positive (ER+), meaning the cancer cells rely on estrogen to grow, which is why treatments often aim to block estrogen production or its action. In contrast, factors that reduce lifetime estrogen exposure—such as early pregnancy or breastfeeding—can lower the risk by limiting prolonged hormonal stimulation of breast tissue.

Thyroid hormone levels can influence the risk and behavior of Breast cancer, but the relationship is indirect and still being actively researched. The thyroid gland regulates metabolism through hormones like T3 and T4, and these hormones interact with other endocrine systems—especially estrogen, which plays a key role in breast tissue growth.

In conditions such as Hypothyroidism (low thyroid hormone levels), metabolism slows down and there may be changes in estrogen metabolism, sometimes leading to relatively higher circulating estrogen levels. This prolonged estrogen exposure can increase stimulation of breast cells, which may raise cancer risk. On the other hand, Hyperthyroidism (high thyroid hormone levels) increases metabolic activity and may enhance cell turnover and oxidative stress, which could also contribute to abnormal cell changes in breast tissue.

Some studies have found that women with thyroid disorders—especially autoimmune thyroid diseases—have a slightly higher incidence of breast cancer, possibly due to shared hormonal and immune system pathways. Thyroid hormones may also directly affect breast cells by binding to receptors that influence cell growth, similar to estrogen-like effects in certain contexts.

However, it's important to understand that thyroid imbalance alone is not a direct cause of breast cancer. It acts more as a modifying factor that may influence risk when combined with other elements such as genetics, lifestyle, and hormonal exposure.

Radiation therapy helps cancer patients recover by using high-energy rays (like X-rays or gamma rays) to destroy or damage cancer cells. These rays target the DNA inside cancer cells, preventing them from growing and dividing. Since cancer cells divide much faster than normal cells, they are more sensitive to radiation, so the treatment mainly affects the tumor while nearby healthy cells are better able to repair themselves.

In diseases like Breast cancer, radiation therapy is often used after surgery to kill any remaining cancer cells and reduce the chances of the cancer coming back (recurrence). It can also be used before surgery to shrink tumors, making them easier to remove. In advanced stages, radiation is used as palliative treatment to relieve symptoms such as pain or pressure caused by tumors.

Radiation therapy can be delivered externally (from a machine outside the body) or internally (placing radioactive material inside or near the tumor, called brachytherapy). The treatment is usually given in small doses over several sessions to maximize cancer cell damage while minimizing harm to normal tissues.

Overall, radiation therapy improves recovery by controlling or eliminating cancer cells, reducing recurrence, and helping patients maintain a better quality of life during and after treatment.

Surgery plays a major role in the recovery of cancer patients by physically removing the tumor from the body. In conditions like Breast cancer, surgery is often one of the primary treatments and can significantly improve survival, especially when the cancer is detected early.

The main way surgery helps recovery is by eliminating the bulk of cancer cells at once. Procedures such as tumor removal (lumpectomy) or complete removal of the affected organ (mastectomy) reduce or completely stop the spread of cancer if it has not already metastasized. Removing nearby lymph nodes also helps doctors check whether the cancer has spread and decide further treatment.

Surgery also influences recovery by improving the effectiveness of other treatments. After surgery, therapies like Radiation therapy or chemotherapy are often used to destroy any remaining microscopic cancer cells, reducing the chance of recurrence.

However, recovery depends on several factors such as the stage of cancer, the patient's overall health, and how extensive the surgery is. While surgery can lead to physical side effects like pain, fatigue, or temporary loss of function, proper post-operative care, rehabilitation, and follow-up treatments help patients regain strength and return to normal life.

Overall, surgery improves recovery by removing cancer, preventing its spread, guiding further treatment, and increasing the chances of long-term survival.

Hormone therapy plays an important role in the recovery of patients with Breast cancer, especially when the

ISSN No:-2456-2165 https://doi.org/10.38124/ijisrt/26jul568

cancer is hormone receptor–positive (ER+ or PR+), meaning the tumor grows in response to hormones like estrogen or progesterone.

Hormone therapy works by either lowering the levels of estrogen in the body or blocking its action on breast cancer cells. Since these cancer cells depend on hormones to grow, cutting off this supply slows down or stops their growth and can even cause them to die. This significantly reduces the risk of cancer coming back after primary treatments like surgery or Radiation therapy.

Common treatments include drugs such as Tamoxifen, which blocks estrogen receptors on cancer cells, and Aromatase inhibitors, which reduce estrogen production in the body. These therapies are usually taken for several years after initial treatment to provide long-term protection against recurrence.

Hormone therapy also helps in advanced or metastatic cases by controlling tumor growth and improving quality of life. However, its effectiveness depends on the cancer type—it does not work for hormone receptor–negative cancers.

Overall, hormone therapy improves recovery by preventing cancer regrowth, increasing survival rates, and supporting long-term disease control in breast cancer patients.

Food plays an important role in regulating hormone balance, including thyroxine (thyroid hormone) and estrogen, and imbalances in these hormones can indirectly influence the risk of conditions like Breast cancer.

For thyroxine, diet directly affects thyroid function. Iodine-rich foods such as iodized salt, seafood, and dairy are essential for the production of thyroid hormones; deficiency can lead to Hypothyroidism. On the other hand, excessive intake of goitrogenic foods like cabbage, broccoli, and soy (especially when consumed raw and in large amounts) may interfere with thyroid hormone synthesis by affecting iodine uptake. Selenium and zinc, found in nuts, whole grains, and legumes, are also crucial because they help convert inactive thyroid hormone (T4) into its active form (T3).

Diet also strongly influences estrogen levels. High-fat diets, particularly those rich in processed foods and red meat, can increase estrogen levels by promoting fat accumulation, as fat tissue produces estrogen. This may contribute to hormonal imbalance and increase breast cancer risk. In contrast, fiber-rich foods such as fruits, vegetables, and whole grains help regulate estrogen by improving its excretion from the body. Phytoestrogens found in foods like flaxseeds and soy can have a mild estrogen-like effect, which may help balance hormone levels when consumed in moderate amounts.

Additionally, lifestyle-related dietary patterns—such as high sugar intake, low nutrient consumption, and obesity—can disrupt both thyroid and estrogen balance by affecting metabolism and endocrine function. Antioxidant-rich foods (like berries and leafy greens) help reduce oxidative stress, which also supports healthy hormone regulation.

Overall, a balanced diet rich in essential nutrients, moderate in processed foods, and supportive of healthy body weight can help maintain proper thyroxine and estrogen levels, thereby reducing the risk of hormonal imbalance and related diseases.

- *Rising Consumption of Soy-Based Products*

Items such as tofu, soy milk, and processed soy foods have become more popular, especially among vegetarians. Excessive soy intake may interfere with thyroid hormone synthesis and absorption, particularly in people with low iodine intake.

- *Frequent Consumption of Cruciferous Vegetables*

Vegetables like cabbage, cauliflower, and broccoli are commonly eaten year-round. While healthy, excessive intake (especially raw) can mildly inhibit iodine uptake due to goitrogens, affecting thyroxine production.

- *Increased Consumption of Millets and Plant-Based Diets*

Millets (such as ragi and bajra) are being promoted as healthy alternatives and are consumed more frequently. However, in excess and without proper iodine intake, they may have mild goitrogenic effects.

- *Low Intake of Micronutrient-Rich Foods*

Modern diets sometimes lack nuts, seeds, and whole foods rich in selenium and zinc, which are essential for converting T4 into active T3.

- *Dairy Products (Widely Consumed Daily)*

Milk, cheese, and butter consumption has increased. Some studies suggest that dairy products may contain natural hormones or stimulate estrogen production, especially when consumed in large amounts.

- *Soy-Based Foods (Rapidly Increasing Trend)*

Foods like soy milk, tofu, and soy nuggets are more popular now. Soy contains phytoestrogens (plant-based estrogen-like compounds) that can mimic weak estrogen activity in the body. Their effect depends on quantity—moderate intake may help balance hormones, while excessive intake may influence estrogen levels.

- *Refined Carbohydrates and Sugary Foods*

White bread, pastries, sweets, and sugary drinks are commonly consumed. These foods increase insulin levels, which can indirectly raise estrogen production and disrupt hormonal balance.

Fiber-rich foods (such as whole grains, fruits, vegetables, and legumes) play an important role in regulating hormones like thyroid hormones and estrogen by influencing digestion, metabolism, and hormone clearance in the body.

For thyroid function, especially in conditions like Hypothyroidism, fiber helps indirectly. A high-fiber diet

ISSN No:-2456-2165 https://doi.org/10.38124/ijisrt/26jul568

improves gut health and supports better metabolism, which is important for proper conversion of thyroid hormone T4 into its active form T3. It also helps maintain a healthy body weight, and since obesity can disrupt thyroid balance, fiber contributes to hormonal stability. However, excessive fiber intake (very high amounts) may slightly reduce the absorption of thyroid medications, so balance is important.

For estrogen, fiber has a more direct effect. Fiber binds to excess estrogen in the digestive tract and helps remove it from the body through excretion. This reduces the amount of circulating estrogen, preventing hormonal overload. Lower and well-regulated estrogen levels are beneficial in reducing the risk of hormone-related conditions such as Breast cancer. Additionally, fiber improves insulin sensitivity, and since high insulin levels can increase estrogen production, this further helps maintain hormonal balance.

Fiber-rich diets also promote a healthy gut microbiome. Certain gut bacteria help regulate estrogen metabolism (known as the “estrobolome”), and a fiber-rich diet supports these beneficial bacteria, improving hormone regulation.

Overall, fiber-rich foods help maintain hormonal balance by improving metabolism, supporting gut health, regulating estrogen levels, and indirectly supporting thyroid function, making them essential for overall endocrine health.

Fiber regulates hormones through several biological mechanisms involving digestion, metabolism, and gut microbiota. Its effects on estrogen are more direct, while its influence on thyroid hormones is mostly indirect but still important.

For estrogen, fiber acts primarily in the intestine. After estrogen is processed in the liver, it is released into the gut in a conjugated (inactive) form. Certain gut bacteria can convert it back into its active form through an enzyme called β-glucuronidase, allowing it to be reabsorbed into the bloodstream (enterohepatic circulation). Fiber-rich foods reduce this reabsorption in two ways: first, fiber binds to estrogen in the intestine and promotes its excretion through feces; second, a high-fiber diet supports beneficial gut bacteria that lower β-glucuronidase activity, reducing the amount of estrogen that gets reactivated. This leads to lower circulating estrogen levels, which is protective in conditions like Breast cancer.

For thyroid hormones, the mechanism is indirect. Fiber improves gut health and metabolic efficiency, which supports the conversion of thyroxine (T4) into the active hormone triiodothyronine (T3). A healthy gut microbiome also contributes to better nutrient absorption, including iodine, selenium, and zinc—key elements required for thyroid hormone synthesis and activation. In addition, fiber helps regulate blood glucose and insulin levels; stable insulin reduces metabolic stress on the endocrine system, supporting overall thyroid function. This is particularly helpful in managing disorders like Hypothyroidism.

However, excessive fiber intake can slightly reduce the absorption of thyroid medications and some minerals, which may affect hormone levels if not properly managed.

In summary, fiber regulates estrogen by enhancing its elimination and reducing reabsorption, while it supports thyroid hormone balance by improving gut health, nutrient absorption, and metabolic stability.

III. METHODOLOGY

Secondary data collection is the process of gathering and using data that has already been collected, recorded, and published by others for a different purpose. Instead of collecting new (primary) data directly from participants, researchers rely on existing sources such as books, research articles, government reports, census data, medical records, and databases. This method is widely used in fields like Research methodology because it saves time, cost, and effort, while still providing valuable information for analysis.

Details of breast cancer patients female aged between 18 and 30 were collected from the authentic source to analyse the corelation between the occurance of the cancer with the hormonal change which is expressed with uterine and thyroid symptoms

Analysis also made considering the condition all the patient exhibitibg normal condition and they silently develop the breast cancer.

- *Data Analysis*

Breast cancer has become the most frequently diagnosed cancer among women worldwide, with its incidence continuing to increase across both developed and developing countries. According to the Global Cancer Observatory (GLOBOCAN 2022), approximately **2.3 million new breast cancer cases** and **670,000 deaths** were reported globally, making breast cancer the leading cancer diagnosed in women. Although improved screening and early detection have contributed to increased diagnosis, the rising incidence is also associated with changes in reproductive patterns, urbanization, obesity, physical inactivity, alcohol consumption, and the adoption of energy-dense Western dietary habits. Unlike non-modifiable risk factors such as age, genetics, and family history, dietary habits represent an important modifiable factor that may influence breast cancer risk through effects on body weight, chronic inflammation, oxidative stress, insulin resistance, and hormone metabolism.

Recent systematic reviews indicate that dietary patterns characterized by high consumption of fruits, vegetables, legumes, whole grains, nuts, and dietary fiber, together with limited intake of red and processed meats, refined carbohydrates, sugar-sweetened beverages, and ultra-processed foods, are associated with a lower risk of postmenopausal breast cancer. Evidence from the World Cancer Research Fund’s Global Cancer Update Programme further demonstrates that greater adherence to healthy dietary and lifestyle recommendations is associated with a reduced

ISSN No:-2456-2165 https://doi.org/10.38124/ijisrt/26jul568

risk of breast cancer. These findings emphasize that dietary modification should be considered an integral component of breast cancer prevention strategies alongside regular physical activity, maintenance of a healthy body weight, and avoidance of alcohol and tobacco. While no single food can prevent breast cancer, adopting a balanced dietary pattern rich in plant-based foods may contribute to reducing modifiable risk factors and improving overall metabolic and hormonal health.

IV. DISCUSSION AND RESULTS

Breast cancer is a hormone-dependent malignancy in which endocrine regulation plays a fundamental role in tumor initiation, progression, and therapeutic response. Approximately 70–75% of breast cancers express estrogen receptors (ER-positive), indicating that estrogen signaling is a major driver of disease progression, while progesterone receptor (PR) status is also used to determine prognosis and endocrine treatment strategies. In addition to reproductive hormones, thyroid hormones contribute to breast physiology through interactions with estrogen signaling pathways, and disturbances in thyroid hormone homeostasis have been associated with alterations in breast cell proliferation and tumor biology. Recent reviews describe a complex hormonal crosstalk between thyroid hormones (triiodothyronine [T3] and thyroxine [T4]), thyroid-stimulating hormone (TSH), estrogen, and progesterone that may influence breast cancer development, although the underlying mechanisms remain under investigation. Experimental evidence has shown that thyroid hormones can regulate the expression of genes involved in cell proliferation and may interact with estrogen receptor signaling, but epidemiological findings remain inconsistent regarding whether thyroid dysfunction directly increases breast cancer risk. ([PubMed][1])

Millets have gained considerable attention as nutrient-dense cereals because of their high dietary fiber, polyphenol, antioxidant, and micronutrient contents. However, concerns have been raised regarding the presence of naturally occurring goitrogenic compounds, particularly C-glycosyl flavones in pearl millet (Pennisetum glaucum). These compounds have demonstrated antithyroid activity in laboratory and animal studies by inhibiting thyroid peroxidase activity and interfering with iodine utilization. Nevertheless, the most recent systematic review evaluating nine published studies concluded that the available human evidence is insufficient to establish that pearl millet consumption, when included as part of a balanced diet, causes thyroid dysfunction or goiter in iodine-sufficient populations. Importantly, no well-designed human clinical trials have demonstrated that millet consumption significantly alters circulating estrogen, progesterone, testosterone, luteinizing hormone (LH), or follicle-stimulating hormone (FSH) concentrations. Consequently, there is currently no scientific evidence to support the hypothesis that dietary millet intake directly modifies sex hormone production or influences hormone-dependent breast cancer through endocrine disruption. Instead, the potential health benefits of millet consumption—including improved glycemic control, reduced oxidative stress, enhanced dietary fiber intake, and better metabolic health—may indirectly contribute to lowering chronic disease risk, including factors associated with breast cancer, although direct clinical evidence linking millet intake with reduced breast cancer incidence through hormone regulation is currently lacking. Therefore, existing evidence suggests that moderate millet consumption is safe for healthy individuals with adequate iodine intake and should not be considered a dietary factor that adversely affects hormone regulation or breast cancer risk. ([PubMed][1])

- *Results*

Dietary modification has emerged as an important component of breast cancer prevention because diet influences obesity, chronic inflammation, oxidative stress, insulin resistance, and hormone metabolism, all of which are associated with breast carcinogenesis. Current evidence supports a dietary pattern rich in vegetables, fruits, whole grains, legumes, nuts, seeds, and healthy fats such as those derived from olive oil, while limiting red and processed meats, refined carbohydrates, sugar-sweetened beverages, and ultra-processed foods. The Mediterranean dietary pattern has consistently been associated with a lower risk of breast cancer, particularly among postmenopausal women, owing to its high content of dietary fiber, antioxidants, polyphenols, and unsaturated fatty acids. Whole grains, including millets, have also attracted attention because they provide complex carbohydrates, dietary fiber, B vitamins, minerals, and bioactive phytochemicals that may improve metabolic health and reduce oxidative stress. Although current evidence does not demonstrate that millet consumption directly prevents breast cancer through hormonal regulation, incorporating millets as part of a balanced, plant-based dietary pattern may contribute to overall cancer prevention by supporting healthy body weight, improving glycemic control, and enhancing antioxidant intake. Therefore, a healthy diet should be viewed as one component of a multifaceted breast cancer prevention strategy rather than as a standalone preventive intervention.

V. SUMMARY

Breast cancer is a hormone-dependent disease, with approximately 70–75% of cases being estrogen receptor (ER)-positive, indicating that estrogen plays a major role in tumor growth. Progesterone receptor status is also important for prognosis and treatment. Thyroid hormones (T3 and T4) interact with estrogen signaling and may influence breast cell proliferation, but current evidence is inconsistent regarding whether thyroid dysfunction directly increases breast cancer risk. Millets are nutrient-rich cereals containing dietary fiber, antioxidants, and polyphenols. Although pearl millet contains natural goitrogenic compounds that can affect thyroid function in laboratory and animal studies, current human studies do not provide sufficient evidence that millet consumption causes thyroid disorders in iodine-sufficient individuals. Furthermore, no clinical studies have shown that millet intake significantly alters estrogen, progesterone, or other reproductive hormones. Instead, millets may indirectly reduce breast cancer risk by improving metabolic health, controlling blood glucose, and reducing oxidative stress. Overall, moderate millet consumption is considered safe and

ISSN No:-2456-2165 https://doi.org/10.38124/ijisrt/26jul568

is not associated with adverse hormonal changes or increased breast cancer risk.

RECOMMENDATION

Integrating genetic and hormonal research is essential for a deeper understanding of how Breast cancer develops and progresses, particularly among high-risk individuals. Genetic factors such as mutations in BRCA1 and BRCA2 significantly increase a woman's lifetime risk of developing breast cancer. However, these genetic mutations do not act in isolation; their effects are often influenced by hormonal exposure, especially estrogen and progesterone, which regulate breast tissue growth and differentiation.

Future research should focus on how hormonal environments interact with genetic susceptibility. For instance, individuals with BRCA mutations may be more sensitive to prolonged estrogen exposure, which can accelerate abnormal cell proliferation and increase the likelihood of tumor formation. Studying how factors such as early menarche, late menopause, or hormone replacement therapy affect genetically predisposed individuals can provide valuable insights into disease onset and progression.

Additionally, integrating molecular biology techniques with endocrine studies can help identify biomarkers that reflect both genetic mutations and hormonal activity. This approach can improve early detection by identifying women who are not only genetically at risk but also hormonally vulnerable. It can also guide personalized prevention strategies, such as targeted screening schedules or preventive therapies that regulate hormone levels.

Furthermore, this integration can enhance treatment outcomes by supporting precision medicine. For example, patients with hormone receptor–positive tumors and BRCA mutations may benefit from combined therapies that target both hormonal pathways and genetic repair mechanisms. Understanding these interactions can also help in developing new drugs that specifically address gene–hormone pathways.

Overall, combining genetic and hormonal research will enable the identification of highly vulnerable populations, improve risk prediction models, and support more effective, individualized prevention and treatment strategies for breast cancer.

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