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Molecular Mechanisms of Endocrine-Disrupting Chemical–Induced Breast Carcinogenesis: Insights from Cellular, Animal, and Multi-Omics Studies

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Abstract
Breast cancer is the most commonly diagnosed malignancy in women across the world, with environmental factors accounting for about 90-95% of all cancers. Toxicants in the environment, including endocrine-disrupting chemicals prevalent in contemporary environments through plastics, pesticides, and personal care products, as well as industrial pollutants, have become significant factors in breast carcinogenesis. Such exogenous compounds imitate or inhibit natural hormones, which cause deviant cellular responses that favour malignant transformation. This is a systematic review of the molecular pathways of EDC-induced breast cancer surrounding the synthesis of the cellular research, animal models, and multi-omics tools.
The review summarises existing research that shows that EDCs have carcinogenic effects that occur via hormone receptor-mediated mechanisms, epigenetic alterations, induction of oxidative stress, and cell cycle regulation. Critical developmental windows of high susceptibility have been identified in animal models with transgenerational effects of cancer risk across generations. Omics technologies, such as genomics, transcriptomics, epigenomics, proteomics, and metabolomics, have uncovered comprehensive molecular indicators of EDC activity and discovered major signalling pathways and possible biomarkers. These mechanisms are vital in understanding preventive proxies, regulatory policies, and therapeutic interventions with the aim of dealing with this avoidable environmental risk factor.

Keywords: Endocrine-Disrupting Chemicals, Breast Cancer, Molecular Mechanisms, Multi-Omics, Carcinogenesis, Epigenetic Modifications

1. Introduction

1.1 Breast Cancer Epidemiology and Environmental Factors

Breast cancer is the most commonly diagnosed malignancy in women all over the world, with more than 2.3 million new cases being reported every year as per the latest global cancer statistics. Although the genetic predisposition is responsible for a range of 5 to 10%, most of the causes are non-hereditary, especially the environmental exposures that a woman accumulates over a life (Neagu *et al*., 2024) [10]. The increased rate of breast cancer in industrialised countries and the decreasing age of diagnosis in the last several decades led researchers to explore the impact of ubiquitous environmental chemicals in the development of breast cancer. Hormone receptor-positive breast cancer subtypes have been thought to be the result of environmental risk factors, such as the prolonged exposure to synthetic chemicals in both consumer products and food packaging and industrial emissions (Hong *et al*., 2025) [4].

1.2 Endocrine-Disrupting Chemicals: Definition and Classification

Exogenous substances or mixtures that change the activity of the endocrine system, and thus have undesirable health effects on an intact organism or its offspring, are called endocrine-disrupting chemicals. Hormone receptor blockers, natural hormone mimics, or interference with hormone synthesis, transport, metabolism, and elimination may be induced by these chemicals (Czaczkowska *et al*., 2025) [2]. Based on their chemical composition and source, EDCs are divided into a few major categories, including bisphenols, for example, bisphenol A in plastics and food containers, phthalates, which are plasticizers in consumer products, persistent organic pollutants, e.g. dioxins and polychlorinated biphenyls, per and polyfluoroalkyl compounds, which are non-stick coating and firefighting foam components, pesticides and herbicides, used in agriculture, parabens, used as preservatives in personal care products.

1.3 Scope and Objectives of the Review

- To understand molecular mechanisms by which endocrine-disrupting chemicals play a role in breast carcinogenesis by analysing both cellular and animal studies, as well as multi-omics studies.
- To assemble existing literature on the hormone receptor-mediated mechanisms, epigenetic alterations, oxidative stress reactions and disruption of cell cycle regulation that occurs due to exposure to EDC.
- To test critical periods of susceptibility and transgenerational outcomes shown in animals.
- To emphasise the role of genomics, transcriptomics, epigenomics, proteomics and metabolomics in showing the in-depth molecular signatures of the pathogenesis of breast cancer caused by EDC.

2. Cellular Mechanisms of Edc-Induced Breast Carcinogenesis

2.1 Hormone Receptor-Mediated Pathways

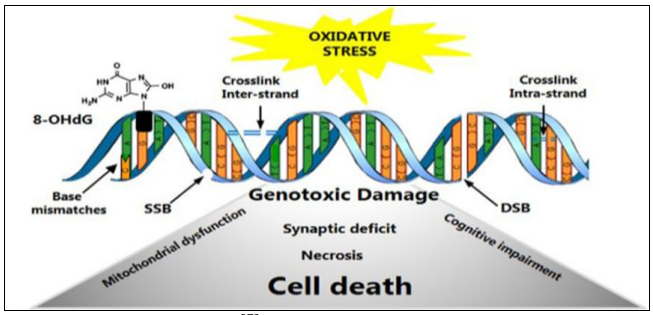
The first mechanism by which endocrine-disrupting chemicals cause carcinogenic effects on breast tissue is through the hormone receptor-mediated pathways. Several EDCs, especially bisphenols and some pesticides, have structural resemblance with endogenous estrogens and may be able to unite with estrogen receptors alpha and beta, thus triggering downstream signalling cascades that stimulate cellular multiplication (Yuan *et al*., 2025) [23]. Another study using molecular docking has found that certain compounds like di(2-ethylhexyl) phthalate have high binding affinities to estrogen receptor alpha, which may stimulate proliferative genes related to breast cancer development (Wang and Wang, 2025) [18].

2.2 Epigenetic Modifications

Endocrine-disrupting chemical exposure has been found to induce epigenetic alterations, which have been central in the connection between environmental exposures and changes in gene expression patterns typical of breast cancer. EDCs have the ability to cause abnormal DNA methylation, especially in the promoters of tumour suppressor genes and hormone metabolic genes, resulting in their silencing with no effect on the underlying DNA sequence (Singh, 2024) [14]. Exposure to EDC also causes changes in histone modifications such as acetylation, methylation, and phosphorylation, which modify chromatin structure and gene accessibility in the mammary epithelial cells. The most recent multi-omics research has found certain patterns of microRNA dysregulations after subjecting MCF-7 breast cancer cells to non-coding RNA compounds such as perfluorooctanoic acid and 4-hydroxybenzophenone, indicating that non-coding RNA changes play a role in EDC-induced carcinogenesis (Yang *et al*., 2024) [21].

2.3 Oxidative Stress and DNA Damage

Oxidative stress is a significant pathway through which cellular damage and carcinogenesis of breast tissue are caused by endocrine-disrupting chemicals. Halophenolic disinfection byproducts and other EDCs in the exposure cause excess production of reactive oxygen species that overwhelm antioxidant defence systems inside the cells, causing oxidative damage to lipids, proteins, and nucleic acids (Li *et al*., 2023) [6].



Source: Liu *et al*., 2025 [7]

Fig 1: Types of DNA damage due to oxidative stress

The analysis of the transcriptomes has shown that EDCs induce oxidative stress that triggers stress response pathways and results in significant DNA damage, such as single and double-strand breaks, base changes, and DNA-protein crosslinks (Liu *et al*., 2025) [7]. Failure of cells to effectively repair oxidative DNA damage enhances the chances of oncogenic mutations being entrenched in the genome.

2.4 Disruption of Cell Cycle and Apoptosis

The endocrine-disrupting chemicals severely impair the normal cell cycle control and apoptotic systems, which ensure against unregulated proliferation and malignant transformation. Exposure to EDC causes cyclin-dependent kinases and their inhibitors to become dysregulated, which causes abnormal cell cycle checkpoint progression and the ability to proliferate mammary epithelial cells (Rizzo *et al*., 2023) [13]. Network toxicological studies have identified CCND1, CDK4, and CDK6 as key cell cycle control genes, which are of primary interest in the action of EDC on breast cancer cells. Moreover, EDCs disrupt pro-apoptotic (BAX) and anti-apoptotic (BCL2) protein expression and activation, which leads to salvaging of damaged cells as they avoid programmed cell death (Zhang *et al*., 2025) [24].

3. Animal Model Insights into Edc-Mediated Breast Tumorigenesis

3.1 Rodent Models and Key Findings

The rodent models, especially the rats and mice, have given priceless information on the carcinogenicity of the endocrine-disrupting chemicals as well as the mechanisms behind the EDC-induced breast tumorigenesis. Such animal models have shown that exposure to such compounds as di (2-ethylhexyl) phthalate can cause such changes in mammary gland morphology, such as more terminal end buds, ductal elongation, and abnormal patterns of branching, which are antecedents of neoplastic transformation (Xu *et al*., 2024) [19]. Moreover, rodent models have been useful in determining certain molecular pathways that are being activated in response to EDC-induced mammary carcinogenesis, such as changes in hormone receptor expression, growth factor signalling, and inflammatory responses (Czaczkowska *et al*., 2025) [2].

3.2 Developmental Programming and Critical Windows

Critical developmental stages, especially prenatal, perinatal, and pubertal developmental stages, are the times of elevated susceptibility to exposure to endocrine-disrupting chemicals

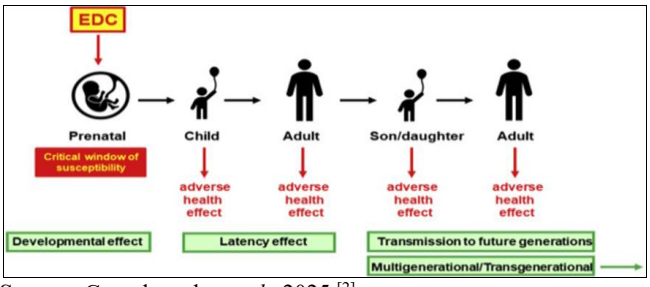
owing to rapid cell differentiation and tissue remodelling in the mammary gland (Wan *et al*., 2022) [16]. Exposure of rodents to bisphenol A during gestation and lactation leads to maldevelopment of the mammary glands, such as ductal malformation, high epithelial density, and terminal end bud persistent development in animals (Ogidi & Tawariowei, 2023) [12].

3.3 Transgenerational Effects

Among the most threatening outcomes of animal model research is that the transgenerational effects of exposing living organisms to endocrine-disrupting chemicals have been shown, meaning that the carcinogenic effect is not only passed on to the specific person who is directly exposed but also passed on to the next generations. Research has demonstrated that the development of breast cancer in daughters and granddaughters of a female who was exposed to EDCs can occur under the influence of epigenetic mechanisms, which are passed on via the germline (Singh, 2024) [14]. Such transgenerational effects include heritable shifts in DNA methylation, histone states, and non-coding RNA expression that change the development of the mammary gland and risk of cancer in the offspring who have not experienced exposure (Caserta *et al*., 2022) [1].

3.4 Relationships of Dose-Response and Low-Dose Effects

The use of animal models has questioned the conventional toxicological beliefs that endocrine-disrupting chemicals usually have non-monotonic dose-response curves and that low doses can have effects other than or even stronger than the effects of higher doses. This is especially applicable to estrogenic EDCs, which may stimulate various receptor subtypes and signalling systems with the concentration (Mafe, 2025).



Source: Czaczkowska *et al*., 2025 [2]

Fig 2: Endocrine-disrupting chemicals (EDCs)

It has been demonstrated that exposure to environmentally relevant low doses of bisphenol A at sensitive developmental stages will cause mammary gland changes and more tumour vulnerability than high doses, which can either cause compensatory or toxic reactions. Also, animal model mixture effects indicate that additive or synergistic effects may occur when many EDCs are combined in doses that are low on an individual basis, which makes derived safe levels of exposure difficult to determine (Czaczkowska *et al*., 2025) [2].

4. Multi-Omics Approaches to Understanding Edc Effects

4.1 Genomics and Transcriptomics

The use of genomics and transcriptomics has transformed the realisation of the endocrine-disrupting chemical impact on breast cancer because it allows a thorough evaluation of genetic changes and modifications in gene expression under EDC exposure. Genomic evidence has been offered by expression quantitative trait loci analysis to identify environmental risk factors that lead to breast cancer being estrogen receptor-positive, specifically endocrine disruptors that are susceptible to genetic variants (Hong *et al*., 2025) [4]. The analysis of transcriptomes after exposure to per- and polyfluoroalkyl substances has shown a broad dysregulation of genes related to cell proliferation, apoptosis, hormone signals, and immune response, which are the molecular signatures of PFAS-induced breast carcinogenesis (Fu *et al*., 2025) [3].

4.2 Epigenomics

Epigenomic profiling has become an effective instrument for defining the hereditary modifications in the control of genes by exposure to endocrine-disrupting chemicals without modification of the DNA sequences. The analysis of genome-wide DNA methylation has shown that EDCs cause comprehensive changes in the methylation patterns of thousands of CpG sites of the genome, with a specific increase in enrichment of genes related to hormone response, cell cycle regulation, and developmental processes (Singh, 2024) [14]. Multi-omics studies of the combined effects of perfluorooctanoic acid and hydroxybenzophenone have recently identified that mTORC1 signalling is an important consolidator of EDC-related epigenetic changes (Yang *et al*., 2024) [21].

4.3 Proteomics and Phosphoproteomics

The technologies of proteomics and phosphoproteomics have given an important understanding of the functional impact of exposure to the endocrine-disrupting chemical by directly quantifying the abundance of proteins and their post-translational modifications. Proteomics is a mass spectrometry technique that has revealed hundreds of proteins that change their expression in response to exposure to EDC, and these include hormone receptors, growth factor signalling, metabolic enzymes, and structural proteins (Yang *et al*., 2024) [21]. Phosphoproteomics studies have shown EDCs cause widespread alterations in protein phosphorylation states, including most major signalling pathways, including MAPK, PI3K-AKT, and mTOR, which control cell growth, survival, and metabolism. Proteomics has been used to define changes in the mammary proteome in studies evaluating the effects of exposure to DEHP in lactating mice; these studies identified dysregulated proteins that contribute to lipid metabolism, immune response, and cellular stress response to DEHP exposure (Xu *et al*., 2024) [19].

4.4 Metabolomics

Metabolomics has become a vital part of the multi-omics studies, which offer an understanding of the biochemical implications of the endocrine-disrupting chemical exposure by fully profiling small molecule metabolites. Research done with the use of metabolomics to examine the DEHP-induced biotoxicity showed that several disruptions in the metabolic profile of mammary tissue, serum, and faeces occurred, indicating that there was systemic metabolic reprogramming after the EDC exposure (Xu *et al*., 2024) [19]. Specialised metabolomics involving hormone metabolite markers has revealed that EDCs disrupt estrogen,

progesterone, and androgen metabolite ratios, which may induce hormonal disequilibrium that facilitates the formation of breast cancer (Neagu *et al*., 2024) [10].

5. Key Molecular Pathways and Signaling Networks

5.1 Estrogen Receptor Signalling Network

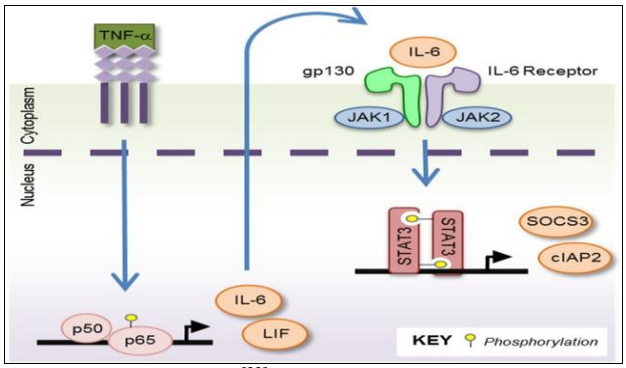
The estrogen receptor signalling network is the main molecular pathway through which most endocrine-disrupting chemicals have their carcinogenic actions on breast tissue (Ning *et al*., 2025) [11]. The research on network toxicology has shown that EDCs stimulate genomic and non-genomic estrogen receptor signalling that induces transcription of estrogen-responsive genes that regulate cell proliferation, survival, and differentiation (Hong *et al*., 2025) [4]. Other than activating direct receptors, EDCs regulate the expression and action of estrogen receptor coactivators and corepressors, amplifying hormonal signals and changing the response to endogenous estrogen transcription. The network analysis has identified the estrogen receptor signalling as a hub that links various pathways derailed by EDC exposure, such as PI3K-AKT-mTOR, MAPK, and JAK-STAT signalling cascades (Fu *et al*., 2025) [3].

5.2 Growth Factor Signalling Pathways

The targets of endocrine disruption by chemical action are growth factor signalling pathways, such as epidermal growth factor receptor, insulin-like growth factor and fibroblast growth factor signalling pathways, which play a central role in breast carcinogenesis. The PI3K-AKT-mTOR pathway has become one of the major agents in the development of EDC-induced breast cancer, and multi-omics studies have found mTORC1 as a major signal integrator between PFOA and hydroxybenzophenone exposure treatment in MCF-7 cells (Yang *et al*., 2024) [21]. EDC also triggers MAPK signalling cascades such as ERK, JNK, and p38 and mediates proliferative, survival, and stress responses (Li *et al*., 2023) [6].

5.3 Inflammatory and Immune Pathways

Inflammatory and immune responses are important in endocrine-disrupting chemical-mediated breast carcinogenesis by the formation of a pro-tumorigenic microenvironment and inhibition of anti-tumour immunity. Exposure to EDC causes the activation of the inflammatory signalling pathways, which include the NF-kB and STAT3, which contribute to the increase of pro-inflammatory cytokine synthesis, such as interleukin-6, interleukin-8, and tumour necrosis factor-alpha (Liu *et al*., 2025) [7].



Source: Yin *et al*., 2025 [22]

Fig 3: NF-κB and STAT3

The multi-omics analysis of microplastic exposure indicated that the environmental polluters can trigger immune evasion and malignant remodelling by stimulating immune checkpoint molecules, including CD47 and PD-L1 (Yin *et al*., 2025) [22]. There has been a report of a wide crosstalk between inflammatory pathways and hormone receptor signalling, where estrogen receptor activation mediated inflammatory responses, and inflammatory mediators mediated hormone receptor expression and activity.

5.4 Signalling Pathways of Development

WNT, hedgehog, notch, and transforming growth factor-beta signalling pathways are abnormally stimulated by exposure to endocrine-disrupting chemicals and implicated in the pathogenesis of breast cancer by acting on stem cell activity and tissue remodelling (Yang *et al*., 2025) [20]. The alteration of the TGF-beta pathway by EDCs disrupts the tumour suppressive properties of the system in the initial stages of carcinogenesis and augments the pro-metastatic properties of the system in the later stages (Ning *et al*., 2025) [11]. It has been found using network toxicology methods that the exposure to parabens leads to a dysregulation of multiple developmental pathways that manifest as radical changes in tissue architecture and cellular differentiation programs (Zhang *et al*., 2025) [24]. It is especially noteworthy that these changes in developmental pathways are most pronounced in cases of exposure to EDC that happens at critical points in the development of the mammary gland, setting the stage for permanent alteration in tissue structure and cancer risks.

6. Mechanisms of Specific Edc Classes

6.1 Bisphenol A (BPA) and Analogues

Bisphenol A and analogues have carcinogenic effects that occur via several molecular pathways that include hormone receptor stimulation, reprogramming of the epigenome, and disruption of metabolism. It has been demonstrated that bisphenols cause breast cancer through progesterone receptor-mediated pathways, so PGR is a key molecular target that enables this interaction (Sun *et al*., 2025) [15]. The exposure to BPA causes extensive epigenomic modifications, such as DNA hypomethylation of the oncogenes and hypermethylation of the tumour suppressor genes (Singh, 2024) [14].

6.2 Phthalates

Phthalates, and in the case of di(2-ethylhexyl) phthalate, they favour breast carcinogenesis by regulating hormone receptors, causing oxidative stress, and interfering with metabolic processes. Several molecular targets of DEHP have been determined in the breast cancer cells by network toxicology and molecular docking analysis, such as estrogen receptors, androgen receptors, and key metabolic enzymes (Wang & Wang, 2025) [18]. Network toxicology investigations of combined exposures to bisphenol A and phthalates have shown that both share common processes of action, including cell cycle deregulation and promotion of proliferative signalling (Wang *et al*., 2026) [17].

6.3 Persistent Organic Pollutants (Dioxins, PCBs)

Chronic organic pollutants such as dioxins and polychlorinated biphenyls cause carcinogenicity by the activation of the aryl hydrocarbon receptor and the long-term disruption of endocrine homeostasis. The

carcinogenicity of polybrominated diphenyl ethers detected in fatty tissues of breast cancer patients has been explored in terms of network toxicology and molecular docking methods, and found various molecular targets of the hormone signalling and cell division (Zhao *et al*., 2025) [25]. This can be attributed to their resistance to degradation, which leads to chronic low-level exposure, which could be especially pertinent to EDC effects after non-monotonic dose-response relationships.

6.4 Pesticides and Herbicides

The contribution of pesticides and herbicides to breast carcinogenesis has a variety of mechanisms that are due to the chemical heterogeneity of these substances. The endocrine-disrupting properties of the fluorinated pesticides have molecular mechanisms that have been identified via cell model research, including the interference with hormone receptors and the induction of oxidative stress (Liu *et al*., 2025) [7]. Some herbicides alter the aromatase, which influences the local estrogen production in the breast tissue (Ogadi & Tawariowei, 2023). The compounded mixtures to which human beings are subjected by food and environmental pollution, most probably have combinatorial effects which are not predicted with speculations made based on individual chemical tests.

7. Challenges and Future Directions

The complexity of mixture exposures (that are found in real-world contexts) is a major difficulty in comprehending endocrine-disrupting chemical-induced breast carcinogenesis because people are concomitantly exposed to dozens of EDCs with possibly synergistic or antagonistic exposures (Molinari *et al*., 2024) [9]. Next-generation directions are advised to focus on creating more complex *in vitro* models, such as three-dimensional culture models, organoids and microfluidics models, with a better representation of breast tissue architecture and microenvironment (Li *et al*., 2025) [5].

8. Conclusions

Endocrine-disrupting chemicals are highly relevant in environmental factors that lead to breast cancer in various and interconnected molecular pathways that have been discovered via cellular biology, animal models, and multi-omics technology. Animal models have defined critical periods of susceptibility at the developmental stages where exposure to EDCs can permanently and irreversibly change the structure of the mammary glands and cancer risks, and even potentially be passed on between generations. Multi-omics techniques have also transformed the knowledge by offering the molecular portraits of the EDC action, which disclose the concerted disturbance of the genomic, epigenomic, proteomic, and metabolomic networks. The pervasive nature of the EDCs in contemporary settings and the growing body of knowledge of their carcinogenicity status warrant the strengthening of regulatory measures, creation of less harmful substitutes of chemicals, and further research to gain a better understanding of the mechanisms and devise intervention measures to prevent this threat that is prevented by environmental factors to breast cancer.

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