Review Article

Exploring the Intersection of Cosmetic Chemical Exposure and Health Disparities in Breast Cancer

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ABSTRACT

Parabens and phthalates are increasingly under scrutiny for their adverse biological effects owing to their ever-widening application in the fields of cosmetics and personal care products. As endocrine disruptors, these chemicals mimic estrogen, bind to hormone receptors, and cause changes in relevant cellular pathways that may lead to cancer development. Parabens and phthalates are believed to stimulate breast cancer cell proliferation, interfere with apoptosis, and activate signaling pathways such as PI3K/AKT, according to in vitro experiments. Such chemicals can be detected in breast tissues and urine, and concentrations associated with increased risk of hormone receptor-positive breast cancer have been confirmed in both studies of environmental contaminants and human population exposures. Moreover, the consequences of exposure extend far beyond personal health impacts, impacting more subtly upon public health and environmental justice. Culturally toxic advertising strategies contribute to restricted access to safer alternatives, hurting women of color and low-incidence populations the most. With inadequate testing on chemical components not being mandated by US regulations, vulnerable communities remain unprotected. The review will consider mechanisms of action for parabens and phthalate toxins, their impact on breast cancer biology, and how those risks may interact with patterns of social inequality. If these issues are truly to be addressed, comprehensive regulation, consumer awareness, and health equity research are required. The reckless use of these chemicals brings the urgency of public health intervention and policy conciseness without further delay to prevent any preventable harm and create a safer environment for all population strata.

Keywords: parabens; phthalates; cosmetics; breast cancer; public health

INTRODUCTION

Daily use is made of cosmetics and personal care products throughout the world. They add up as a proportion to the total consumables in society and are meant to beautify, scent, and prolong freshness with the use of many synthetic chemicals. Two of the most common are parabens and phthalates, which typically appear as preservatives, solvents, and fragrance stabilizers (1, 2). As such, concerns have risen about health effects on humans from the extensive use. Recent two-decadal research has verified that these are neither inert ingredients nor biologically active agents interfering with the endocrine signaling (3).

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It recognizes how today parabens and phthalates are regarded as public health issues emerging rather than isolated, toxicologically worrying agents (4).

Parabens and phthalates are found in creams, shampoos, deodorants, perfumes, nail lacquers, and many other personal care products that are used repeatedly throughout the day (1, 5). Because of their use through topical absorption, inhalation, and incidental ingestion, they become chronic exposures. Although they stabilize products in the chemical industry, their biological effects create concern. Parabens have a structure that is similar to that of estrogen and can bind to estrogen receptors, while phthalates are metabolized into monoesters, which interact with hormonal pathways (3, 6). These actions classify both types of substances as endocrine-disrupting chemicals. The damage to organs will include changes in reproductive function, metabolic disorders, and the possibility of developing hormone-sensitive cancers, including the breasts (2, 6).

There is another public health aspect regarding these substances: they are actually in allowed consumer products with relevance to health well beyond toxicity itself (7). It asserts rights to people living in poverty and deep within the margins of environmental health equity, regulatory policy, and disease prevention (7). Most consumers will remain unaware of such exposures because labels rarely mention them fully in the declaration and packaging (8). Risk management then becomes individual consumers, who are afflicted by a dearth of options available to create safer selections because of insufficient resources and knowledge (7). Cumulative exposures do not limit risk to an individual but actually give rise to risk for the whole population (9).

Emerging evidence reinforces the fact that exposure is not evenly distributed across the population. Researchers have shown that in America, the population group that consists mostly of women who experience more harm from parabens and phthalates is women of color and lower income (7, 10). While this is in part due to differential marketing, cultural beauty standards over history, and the lesser availability of safe alternatives also play their respective roles (10). Examples of products that have these chemicals at higher levels include hair relaxers, skin lightening agents, as well as heavily scented items; these are popularly marketed to women living in marginalized communities (10). Such distinctions manifest a classic case of environmental injustice under which disadvantaged people will structurally have a heavier load of toxic exposure (7).

Law is left far behind by science. Cosmetic products in the United States, for instance, do not necessitate pre-market safety testing before use of most ingredients (11). The Food and Drug Administration has very limited power to restrict chemicals, even if increasing evidence of harm piles up (11). Thus, it is not that way in the European Union, as hundreds of substances are either banned or limited for use in cosmetics (12). The federal statute thus left the United States population, especially of the vulnerable communities within these nations, unguarded against chronic exposures (11, 12). Public health systems have oftentimes been slow to respond owing to limited national surveillance over long-term exposure, with scant effort put into the cumulative assessment of different products (9).

Toxicology and the study of parabens and phthalates are making tremendous strides, and quite some documentation has emerged on the disparities between cosmetic use (1, 2). However, the major gap still remains in integrating these two lines of evidence. The mechanism through which the endocrine-disrupting chemicals cause carcinogenicity is presented in most reviews (3, 6), or else the sociocultural patterns of cosmetic utilization (7, 10). Very few have brought the two perspectives together to analyze the field of the molecular underpinnings of toxicity with the view that they would intersect with race and socio-economic inequities influencing their risks of breast cancer (7, 10). This gap will thus throw a spanner in the efforts made by public health research in addressing biological determinants as well as structural ones, leading to disease.

This is intended to close the gap. Synthesizing present knowledge concerning mechanisms of action of parabens and phthalates with epidemiological data and evidence of unequal exposure would give a more complete understanding of the risks associated with cosmetics and these compounds (6, 13). This will recommend considering cosmetic safety to be a toxicological issue-if an environmental justice issue, as said (7, 10). This review adds novelty by tying endocrine disruption with the health disparities argument, thus focusing on populations that are both biologically vulnerable and socially disadvantaged. Change, therefore, must be systemic and should include better regulation, as well as culturally responsive education and equity-focused research (11, 12). This paper links cosmetic chemical exposure to a public health issue that needs urgent attention (7, 9).

PARABEN AND BREAST CANCER

Chemical Mechanism of Action

Parabens are alkyl derivatives of para-hydroxybenzoic acid, synthesized synthetically and commonly utilized as preservatives in cosmetic products, pharmaceuticals, and personal care commodities (14). They mainly restrict microbial growth and prolong the shelf life of products. In chemistry, parabens would have hydrophobic alkyl chains and hydrophilic hydroxyl groups. Such amphiphilic nature enables them to insert lipid bilayers with hydroxyl groups in touch with the aqueous environment. This characteristic favors their antimicrobial action through breaking the integrity and functions by disrupting membrane integrity and functions (14).

Apart from their action against microorganisms, parabens were also classified as endocrine-disrupting chemicals, which mimic the endogenous hormones, particularly the action of estrogens (3). This structural similarity between parabens and 17β-estradiol-molecular, the main female sex hormone, involves mainly the presence of their common phenolic ring structure (3). Similarity of structure permits esters to bind with estrogen receptors (ERα and ERβ), but at a lower affinity than that of natural estrogens (6). Subsequently, they may interfere with normal hormonal signaling pathways in metabolism, reproduction, and cell division (6).

Hence, this receptor binding is significant because it has the potential to activate or block transcription of estrogen-responsive genes. For example, genes for cellular proliferation and apoptosis suppression may sometimes be expressed by parabens (6). These effects raise serious concerns regarding tissues sensitive to estrogen, such as the breast, where they are most likely to occur. Thus, even a single such treatment has relatively lower estrogenic potency but is still alarming due to its high use, thereby causing chronic low-dose exposure, which raises questions about long-term biological effects, especially in hormone-sensitive tissues (1).

In vitro Effects on Breast Cancer

Experimental studies *In vitro* have shown for some time that parabens can alter breast cancer cell behavior. Salting human breast cancer cell lines like MCF-7 and BT-474, parabens enhance cell proliferation, inhibit apoptosis, and stimulate a variety of pathways leading to the expression of cellular activities related to tumor progression (6). Such responses are then considered biomarkers of their carcinogenic potential.

Amongst other interesting findings was that butylparaben was shown to upregulate c-Myc oncogene expression and induce ERα-mediated proliferation in HER2-positive BT-474 cells. These activities were markedly increased in the presence of neuregulin-a growth factor thought to be involved in HER2 signaling-which may indicate a possible interaction between paraben exposure and existing oncogenic pathways in breast tissue (6). Those findings indicate a strong preparedness in parabens to enhance the already precarious disease state of cells, particularly in subjects genetically predisposed or already receptor prone.

In addition to acting on estrogen receptors, parabens seem to stimulate receptor-independent pathways within the signaling cascades. For instance, propylparaben promotes increased phosphorylation of the key protein AKT within the PI3K/Akt pathway, which is known to promote cell survival and growth (15). This indicates that parabens can potentially alter pathways that affect the proliferation and motility of breast cancer cells by more than one mechanism and thus raise questions about their impact on the onset and progression of cancer.

Human Clinical and Epidemiological Evidence

Evidence from tissue studies and epidemiology in humans supports the biological plausibility of parabens being implicated in breast cancer (3). They have been widely reported in human biological fluids such as serum, urine, breast milk, and even semen, corroborating that the general population has been widely exposed to this product (3). One highly cited study by Darbre *et al*. reported that parabens were found in every single one of the 100 breast tumors analyzed, with a mean concentration of 20.6 ± 4.2 ng/g tissue (3). Although this study does not provide a demonstration of direct causation, it raises alarming possibilities of paraben accumulation in breast tissue and its biological effects thereafter over time.

The second avenue of support is epidemiological studies linking abuse paraben levels with breast cancer risk. One study conveyed that women in the highest quintile of paraben exposure were 30 to 50 percent more likely to develop breast cancer than those in the lowest quintile (16). The defined risk was stronger for women with a BMI of less than 25 kg/m², suggesting that body composition may modify the effect of exposure (16). In addition, although associations are found between paraben levels and a reduction in all-cause mortality

following breast cancer diagnosis, the very broad confidence intervals suggest a degree of statistical imprecision warranting further investigation (16).

Limitations

Epidemiological studies indicate paraben exposure is associated with breast cancer risk. However, results are not always consistent. A major reason for this is the differences in exposure assessment (3, 9). Parabens are rapidly metabolized and excreted, with half-lives measured in hours, rendering a single spot urine sample a poor indicator of long-term exposure (3, 9). Some studies rely on urinary concentrations, others on breast tissue concentrations, and some others on self-reporting the use of cosmetics (3, 17). Thus, these differences in methodology result in divergent results because a short-term biomarker measurement may wrongly represent or underestimate chronic exposure patterns (3, 17). In addition, population characteristics affect the outcomes of the study (17, 18). For instance, a specific study reported a stronger association between urinary paraben levels and breast cancer among women with a body mass index below 25 kg/m² (17). An example is the possible confusing factors in the connection, such as postmenopausal state, genetic susceptibility, and lifestyle factors like alcohol consumption or smoking (17, 18). Another complication arises from the fact that parabens are seldom administered alone, due to their actual use in commercial cosmetic mixtures, making it difficult to pinpoint their independent effects (1,5). The small sample sizes with heterogeneous designs further limit one's interpretation, as retrospectively designed case-control studies may be more likely to be biased within particular situations where predefined large cohorts are established (13, 17). These factors further explain why some studies show a significant association of paraben exposure to the incidence of breast cancer, while others show null or inconclusive findings (13, 17). A thorough understanding of the limitations indicates the need for longitudinal studies with repeated biomarker collection, uniform exposure assessment, and adequate control for confounding (13, 17). Such methods would bring clarification on whether the links observed were truly causal effects or the result of methodological variability (13, 17).

On the balance of empirical data, laboratory or population study findings undoubtedly provide evidence verifying the hypothesis of parabens functioning in breast carcinogenesis under chronic exposure (3, 6). As concerning mechanisms of action, the use of parabens from activation at the estrogen receptors to modulation of critical survival pathways is consistent with the currently known processes involved in tumor development (6, 15). Thus, the cumulative use of such parabens through ongoing use in cosmetics and their topical application to sites adjacent to breast tissue raises further concerns in public health over cumulative use, especially for the susceptible populace (7).

PHTHALATE & BREAST CANCER

Chemical Mechanism of Action

Phthalates are made from synthetic ingredients of 1,2-benzenedicarboxylic acid. Mostly, they can be found in industrial plastics and personal care items. These chemicals are used primarily as plasticizers and solvents for cosmetics; for example, common cosmetics containing phthalates are fragrances, hair sprays, and nail polishes (18). Structurally, phthalates consist of a benzene ring having two ester functional groups in ortho position, imitating the positioning of the natural hormones; this structure can bind phthalates to hormone receptors, especially those that participate in the estrogen signaling pathway (5, 19).

Once absorbed by humans either orally, through inhalation, or through dermal absorption, the phthalates quickly metabolize into monoester derivatives like monoethylhexyl phthalate and monobutyl phthalate (20), which are known to interfere with endocrine activity. The results of studies indicated that certain phthalates might bind dose dependently to the estrogen receptors (6), most particularly in alkyl chains with long or side-chain structures in a dose-dependent manner. For instance, ortho-substituted diallyl phthalates exhibited a higher binding affinity for estrogen receptors alpha (ERα) than that of their other isomers (21).

Molecular modeling and receptor binding assays confirm that dibutyl phthalate and dicyclohexyl phthalate mimic the binding mode of natural estrogens. Such phthalate species can enhance specific conformational changes in the receptor, which may result in an altered function (22). This property of receptor-specific interaction permits phthalates to disturb proper hormonal control, which causes much worry regarding possible impacts of such agents on hormone-sensitive diseases like breast cancer (18, 22).

***In vitro* Effects on Breast Cancer**

In vitro studies provide some pretty compelling evidence that the effect that phthalates have on cellular

behavior potentially has implications in the development of cancer or the development of a cancer phenotype. Associated with the aforementioned, some phthalates such as benzyl butyl phthalate (BBP), di-n-butyl phthalate (DBP), and di-2-ethylhexyl phthalate (DEHP) have propagated the proliferation of estrogen receptor-positive breast cancer cell lines, specifically MCF-7 (23). Growth factors then induced the expression of PCNA (proliferating cell nuclear antigen), a marker of cell replication and cell cycle transition. Furthermore, these growth factors activated the PI3K/AKT signaling pathway, an important pathway needed for the survival and growth enhancement of the cell (23).

One of the most alarming patterns would be that phthalates counteract the effects of tamoxifen-induced apoptosis. Tamoxifen is used to block the signaling of estrogen receptors in breast cancer. It is indicated that when the effect of tamoxifen on the resistant MCF-7 cells is considered, certain actions of phthalates might inhibit the action of an important therapeutic (21). Another study showed that 9 out of 22 phthalates tested were as estrogenic as or more so than β-estradiol, the natural ligand of estrogen in the body, especially true for those having lateral alkyl chains composed of 3 to 6 carbons (22).

So these studies conclude that the phthalates stimulate proliferative responses in breast cancer cells, which may reduce the efficiency of hormone therapies in the future. The suggested biological responses are consistent with tumor-promoting mechanisms like increased mitotic activity, inhibition of programmed cell death, and increased activation of estrogen receptors (19, 22).

Human Clinical and Epidemiological Evidence

Phthalates, indeed, are found to be ubiquitous in human exposure as they have been consistently found in urine samples across some of the largest population cohorts (9). Epidemiological studies have been initiated to find possible associations of such metabolites with breast cancer. One of the studies involved more than 700 patients who were diagnosed with breast cancer and demonstrated prognostically relevant correlates indicating that such patients had urinary levels of phthalates associated with a greater tendency to present with aggressive disease, a greater requirement for chemotherapy, and lower values of estrogen and progesterone receptor positivity (24). Thus, the influence of phthalate exposure on both the development and severity of disease progression is corroborated.

Another cohort study conducted in Denmark involved more than one million women and recorded those having high cumulative exposure to dibutyl phthalate, which showed a significantly increased risk of developing estrogen receptor-positive breast cancer (13). Interestingly, results showed that neither low nor moderate exposures were associated with increases in risk, making dosage and exposure time the crucial parameters concerning health effects (13, 24).

According to these observations, it is understandable how chronic exposure to long durations of phthalates could affect human health. In fact, longitudinal studies are insufficient to validate causation, while the signs to date indicate a trend that is more worrying (24). Phthalates are characterized by bioaccumulation and interference with hormonal signaling pathways, making them potentially contributory to the risk of breast cancer in the manner of potential direct receptor interaction and disruption of cellular homeostasis (19, 22).

Limitations

Epidemiological studies of phthalates and breast cancer have been inconsistent, with the gaps often attributed to differences in study methodology and population characteristics. Phthalates are non-persistent compounds with half-lives below a day (20), so spot urine samples only reflect short-term exposure and may not mirror chronic use more relevant to cancer development. Therefore, findings from one study may not be replicated in another study that collects samples at a different time point or with a different frequency (20). From basically small case-control studies in the United States, which rather frequently report mixed or null results (24), to large prospective cohorts like the Danish study associating cumulative dibutyl phthalate exposure with estrogen receptor-positive breast cancer (13), study designs vary. Further variability is introduced by differences in genetics, lifestyle behaviors, and socioeconomic contexts that are determinants of both product use and disease susceptibility (17, 18). One more challenge to weigh is the fact that phthalates almost never exist alone. Other endocrine-disrupting compounds with which phthalates co-occur in cosmetics and plastics make it difficult to isolate their independent effects (5). Biological activity could also differ among phthalates with varying side-chain lengths and molecular structures, rendering the interpretation of effects across different metabolites all the more complicated (21). In concert, these explanations account for the contradictory findings found in epidemiology.

While some studies suggest that high exposure is associated with more aggressive tumor features and increased therapy needs (24), confirmation of this link is lacking in other studies (24). This will necessitate large multiracial cohorts with biomarker measurements repeated over time, standardized laboratory techniques, and careful adjustments for genetic, lifestyle, and environmental confounding factors (24).

This phthalate remains a big issue in the field of public health. They are virtually everywhere in consumer products, and since therein lies great transparency for their labeling, the long periods of exposure at low concentration became something of importance, too difficult for the consumer to avoid. Risk will then be alleviated through research and consideration for further regulations supported by culturally relevant public education to eliminate equity gaps in exposure and disease results (7, 11).

EXPOSURE TO PARABENS & PHTHALATES AS A PUBLIC HEALTH CONCERN

Parabens and phthalates present a double jeopardy nexus on one hand as toxicological hazards and on the other as social determinants of health. Their presence in cosmetics used every day by millions establishes a chronic poisoning link to endocrine disruptors. While laboratory and epidemiological evidence suggest a contribution toward breast carcinogenesis, more detailed consideration must be given to confounding factors, methodological considerations, and the social context of such exposures.

Genetic susceptibility is an additional source of variation in epidemiological findings. Mutations in BRCA1 and BRCA2, along with HER2 amplification, confer a high breast cancer risk and may alter susceptibility to xenoendocrine disruption (25). The concern is raised that parabens may augment ERα-mediated proliferation in HER2-positive cell lines, and thus genetically predisposed women may be facing compounded risks (6). Nonetheless, the consideration of genetic parameters is rarely included when many population studies are conducted, obstructing the preferred mechanism of categorizing independent as well as interactive factors.

Lifestyle factors stand to potentially confound. Alcohol use, smoking, obesity, and lack of exercise are some of the established risk factors for breast cancer (17). Obesity is believed to influence endogenous hormone metabolism, which may serve to amplify the biological effectiveness of parabens and phthalates, while alcohol consumption and smoking are themselves additional endocrine-disrupting exposures. Besides, these behaviors cluster together often within marginalized populations, making it difficult to delineate chemical risks from lifestyle ones. For example, Parada *et al*. observed that the association between breast cancer and paraben exposure was greater for women with a low body mass index, suggesting that body composition and chemical metabolism have a more complex interaction (16).

Further occupational exposures create even more difficulty in establishing the risk profiles. The women working in hair and nail salons and in cosmetics manufacturing experience repeated and high exposures to parabens and phthalates, and are routinely exposed to other toxicants (26). Women of color and immigrants comprise a disproportionately large share of these working populations, many of whom experience structural barriers to health care. Once again, occupational exposures rarely figure into breast cancer studies, thereby obscuring the combined effects of professional and personal use of cosmetic products.

Social and cultural factors introduce another layer. Historically, marketing has targeted women of color with relaxers, skin lighteners, and heavily scented cosmetics that are high in endocrine disruptors (10). Structural racism and colorism mold beauty norms for the promotion of these products, while economic inequities restrict access to safer substitutes (27). Such disparities render chemical exposure in cosmetics a clear instance of environmental injustice, whereby structurally disadvantaged groups bear an inordinate toxic burden.

A wider policy implication arises. With respect to the cosmetic ingredients outcome, the U.S. is still trailing the European Union, which, in its precautionary approach, banned or restricted over 1,000 substances (12). In contrast, the U.S. Food and Drug Administration does not mandate pre-market safety testing, nor does it require comprehensive ingredient information (11). Hence, this gaping regulatory hole promotes forever exposure, even while scientific evidence has been piling up. Some urgently needed actions include introducing stronger regulatory oversight, including the requirement of safety testing and disclosure of labels.

Also, interventions must be culturally sensitive. Unless they consider the cultural norms, political economy, and other barriers that shape cosmetic use, generic campaigns for consumer education are unlikely to reach the women most affected. Initiatives

that specifically target that community are essential to present meaningful risk information and safer alternatives (7). Research should also change. Large, longitudinal, multi-ethnic cohorts with repeated biomarker measurements are required to clarify associations. These studies should account for genetic predisposition, lifestyle exposures, and occupational exposures to rule out confounding and improve causal inference. An emphasis on environmental justice will ensure that any disparities in exposure and health outcomes will not merely be an afterthought but rather a key tenet of future investigations.

All in all, far from merely being an issue of toxicology pertaining to parabens and phthalates, their influences must be seen through the different intersecting lenses of genetics, lifestyle, occupation, culture, and policy. What the evidence emphasizes is that the question of cosmetic chemical safety is a structuring determinant of breast cancer disparities. In addressing this issue of greater regulation, culturally relevant interventions and equity-focused research would be required in order to avert further health inequities.

CONCLUSION

Parabens and phthalates are still predominantly used in cosmetics and personal care products as preservatives, solvents, and stabilizers for fragrances. *In vitro* evidence suggests that these chemicals mimic estrogen, activate proliferative pathways like PI3K/AKT, and inhibit apoptosis in breast cancer cells, as summarized by Figure 1 and Table 1 (15). While the results from epidemiology aren't always consistent, the majority suggest that chronic exposures may result

Table 1. Summary of key cosmetic chemicals, sources of exposure, mechanisms, and evidence linking them to breast cancer

Chemical	Common Cosmetic Sources	Mechanisms of Action	Evidence for Breast Cancer Relevance	Key References
Parabens (methyl-, propyl-, butyl-)	Lotions, shampoos, deodorants, makeup, sunscreens, perfumes	Estrogen receptor binding (ERa, ERB); mimic estradiol; activation of PI3K/AKT pathway; suppression of apoptosis; increased proliferation	Detected in human breast tumors; urinary concentrations associated with increased breast cancer risk; stronger effects in women with lower BMI	Darbre & Harvey, 2008 (3); Pan *et al*, 2016 (6); Wrobel & Gregoraszczuk, 2015 (15); Parada *et al*., 2019 (16)
Phthalates (DBP, DEHP, BBP)	Fragrances, nail polish, hair sprays, skin lotions	Estrogen receptor binding; interference with tamoxifen-induced apoptosis; activation of PI3K/AKT pathway; increased PCNA expression	Urinary metabolites linked with more aggressive breast cancer and higher chemotherapy needs; cohort study found cumulative DBP exposure associated with higher ER+ breast cancer risk	Mittermeier *et al*., 2016 (20); Kim *et al*, 2004 (21); Zhang *et al*., 2016 (23); Wu *et al*., 2021 (24); Ahern et at., 2019 (13)
Other endocrine-disrupting agents (not focus of this review)	Hair relaxers, skin lighteners, lipsticks, fragrances	Hormone mimicry, oxidative stress, DNA damage (varies by compound)	Limited or mixed epidemiological evidence, but concern raised in environmental justice context	Helm *et al*, 2018 (10); Scott & Wilson, 2021 (27)

This table provides an overview of major endocrine-disrupting chemicals commonly used in cosmetic and personal care products. Parabens and phthalates are highlighted as primary agents of concern, with details on their widespread sources, biological mechanisms of action, and supporting epidemiological or experimental evidence connecting them to breast cancer risk. Additional cosmetic agents, while not the central focus of this review, are included to illustrate the broader context of chemical exposures in beauty products. Collectively, the table emphasizes how these chemicals contribute to both toxicological risk and disparities in breast cancer outcomes.

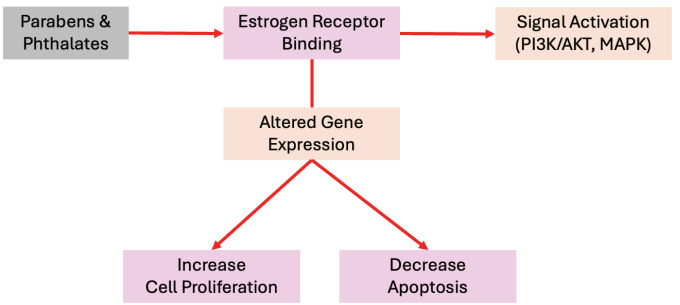

Figure 1. Mechanisms of Endocrine Disruption by Parabens and Phthalates. This schematic illustrates the cellular mechanisms by which parabens and phthalates may contribute to breast carcinogenesis. Both classes of chemicals can mimic endogenous estrogens due to their phenolic or aromatic structures, allowing them to bind to estrogen receptors (ERα and ERβ). Once bound, they may activate estrogen-responsive signaling pathways such as PI3K/AKT and MAPK, leading to altered gene expression, increased cell proliferation, and reduced apoptosis in hormone-sensitive tissues. These processes are implicated in breast cancer development and progression. *In vitro* studies using MCF-7 and BT-474 cells have shown that these chemicals promote oncogenic signaling and interfere with apoptosis. Some phthalates have also been shown to reduce tamoxifen-induced apoptosis, suggesting interference with endocrine therapy. These findings underscore the importance of understanding how daily exposure to endocrine-disrupting chemicals can influence hormone-regulated cellular pathways.

in increased risk for breast cancer that is hormone receptor-positive, especially in patients with additional genetic or lifestyle susceptibilities (3, 16).

Of significance is that exposure varies between populations. Women who are Black and Latina and those who belong to low-income communities face increased exposure because of targeted marketing and little access to safer alternatives (7, 10). This burden is increased by occupational exposures in the beauty and manufacturing industries (26). These differences underscore how exposure to cosmetic chemicals is a hazard at a biological level as well as a social justice problem.

Many recommendations emerge towards risk mitigation. To start with, regulatory reform is paramount. The FDA needs to be empowered to require pre-market testing and enforce full disclosure of ingredient contents in an effort to harmonize such standards with those of the European Union (11, 12). Moreover, consumer education campaigns on safer alternatives and chemical risks should take into consideration cultural competencies and sensitize diverse communities (7). Third, equity-driven research must integrate toxicology with social determinants of health: longitudinal and multiethnic cohorts should consider genetic, lifestyle, and occupational confounders (17, 25, 26). Finally, intervention efforts should be informed by partnerships with community organizations to ensure the relevancy and accessibility of solutions (7, 27).

Concluding, parabens and phthalates are examples of the major linkages between cosmetic safety and regulation, culture, and equity. Formulating exposure as both a toxicological and environmental justice problem demonstrates the need for systemic change. For these reasons, stronger oversight, informed consumers, and equity-centered research are needed to advance cosmetic safety as a means of preventing breast cancer and advancing health justice.

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