Review Article

General Overview of Broadly Discussed Lifestyle and Environmental Factors Casually Associated with Breast Cancer Development

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ABSTRACT

Background: breast cancer represents a significant global health burden, with a complex etiology extending beyond genetic predisposition to encompass modifiable lifestyle and environmental exposures. A myriad of these factors are frequently discussed in both scientific and public domains, yet the evidence supporting these associations varies widely in strength and consistency. Objective: This narrative review aims to broadly survey and synthesize the lifestyle and environmental factors that are casually mentioned in relation to breast cancer development, providing a panoramic overview of these superficial associations without delving into deep mechanistic analysis. Main Discussion Points: The review thematically explores factors including dietary patterns, alcohol and tobacco use, physical activity, body composition, exogenous hormones, environmental chemical exposures, circadian disruption, and socioeconomic influences. It highlights well-established causal links, such as those for alcohol and postmenopausal obesity, while also presenting the more ambiguous and inconsistently supported associations for elements like specific dietary components and endocrine-disrupting chemicals. Critical analysis underscores the methodological limitations inherent in the observational evidence base. Conclusion: The evidence for casually mentioned risk factors exists on a spectrum, with clear public health priorities emerging for risk reduction. Clinicians and policymakers should focus on advocating for interventions related to the most robustly evidence-based factors. The review calls for future research employing more rigorous, integrative study designs to clarify unresolved associations and advance primary prevention strategies.

Keywords: *Breast Cancer; Lifestyle Factors; Environmental Exposure; Risk Factors; Narrative Review; Prevention*

INTRODUCTION

Breast cancer remains the most commonly diagnosed malignancy among women globally, exerting an immense burden on public health systems and societies. With an estimated 2.3 million new cases reported in 2020, it constitutes a leading cause of cancer-related mortality in women worldwide, accounting for approximately 685,000 deaths annually (1). The incidence rates exhibit significant geographical variation, being highest in developed regions such as North America and Western Europe, though rising trends are increasingly observed in transitioning countries, likely due to changes in reproductive patterns and lifestyle adoption (2). This epidemiological profile underscores breast cancer not merely as a biomedical condition but as a complex disease intertwined with societal evolution. While advancements in screening and treatment have improved survival rates in high-income settings, the rising absolute numbers of cases necessitate a continued and intensified focus on primary prevention strategies. Understanding the modifiable contributors to risk is

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therefore paramount, as it holds the potential to shift the paradigm from early detection and treatment to averting the disease's onset altogether, a goal of immeasurable public health value. The established risk factors for breast cancer are multifactorial, encompassing non-modifiable elements such as age, genetic predispositions like BRCA1/2 mutations, and familial history, alongside modifiable hormonal and reproductive factors including early menarche, late menopause, nulliparity, and late age at first birth (3). However, these intrinsic and reproductive factors explain only a portion of the total disease burden, prompting scientific inquiry into the substantial role played by exogenous, lifestyle, and environmental influences. The last few decades have witnessed a burgeoning body of research exploring these associations, moving beyond the traditional focus to consider the totality of exposures an individual encounters—termed the exposome (4). This shift acknowledges that while genetic susceptibility lays the groundwork, it is often the interaction with environmental and behavioral factors that ultimately influences disease trajectory. Consequently, factors such as dietary patterns, physical activity levels, alcohol consumption, body fatness, exposure to endocrine-disrupting chemicals, and night shift work have entered the scientific discourse, frequently cited in both academic literature and public media as components of modern living that may alter breast cancer risk.

Despite the proliferation of studies, the current knowledge landscape regarding these lifestyle and environmental factors is characterized by both compelling associations and persistent ambiguities. For instance, the link between alcohol consumption and increased breast cancer risk is supported by robust epidemiological evidence and deemed causal by several authoritative bodies, with a demonstrated dose-response relationship (5). Similarly, adult weight gain and postmenopausal obesity are consistently associated with heightened risk, largely mediated through estrogen metabolism in adipose tissue (6). Conversely, the evidence for other factors remains less definitive or superficially understood by the broader public. Discussions on dietary components, such as fat intake or fruit and vegetable consumption, often yield inconsistent findings across studies, complicated by methodological challenges in accurately measuring long-term dietary habits (7). The potential role of environmental xenobiotics, particularly endocrine-disrupting chemicals like bisphenol A (BPA) or certain pesticides, is a topic of intense research and public concern, yet translating findings from in vitro and animal models to clear human risk is fraught with complexity regarding exposure assessment windows and dosages (8). This landscape reveals a significant gap: while many factors are casually mentioned in relation to breast cancer in various contexts—from scientific commentaries to health journalism—a synthesized, broad overview that presents these superficial associations without delving into complex mechanistic pathways or attempting definitive causal inference is often lacking. There is a need to map the terrain of these frequently discussed links, acknowledging their presence in the scientific conversation while clarifying the current state of evidence. The objective of this narrative review, therefore, is to broadly survey and compile the lifestyle and environmental factors that are commonly and casually mentioned in the scientific and public discourse in relation to breast cancer development. The aim is not to provide a deep, critical appraisal of the biochemical mechanisms or to establish a hierarchical causal model, but rather to present a panoramic view of these superficial associations as they exist in contemporary discussion. This review will act as a catalogue of potential risk and protective factors that populate the etiological conversation surrounding breast cancer, serving to inform readers of the breadth of elements under investigation.

In terms of scope, this review will encompass factors broadly categorized into behavioral/lifestyle domains—including dietary habits, physical activity, alcohol and tobacco use, and body composition—and environmental exposures, such as ionizing and non-ionizing

radiation, occupational settings, and chemical pollutants with suspected endocrine-disrupting properties. The focus will be on factors frequently cited in review articles, consensus reports, and popular science literature over the last five years, ensuring relevance to current discourse. The inclusion of evidence will be intentionally superficial, drawing primarily on conclusions from large-scale meta-analyses, pooled analyses, and major consensus statements from bodies like the World Cancer Research Fund (WCRF) and the International Agency for Research on Cancer (IARC), rather than dissecting individual conflicting studies. The significance of this narrative review lies in its synthesis and contextualization. For clinicians, it offers a consolidated reference of factors patients may inquire about, enabling informed discussions. For public health practitioners and policymakers, it highlights areas where population-level interventions might be most plausible based on the strength of association and modifiability. For the research community and students, it provides a foundational map of the field, identifying which associations are well-substantiated and which remain in the realm of hypothesis and require further rigorous investigation. By collating these casually mentioned associations into a single coherent overview, this review aims to demystify the often-overwhelming array of risk factors presented in media and literature, offering clarity on what is broadly discussed, even as the depth of evidence varies considerably. Ultimately, it underscores the multifactorial nature of breast cancer etiology and reinforces the importance of a holistic, prevention-oriented approach to mitigating the global burden of this disease.

THEMATIC DISCUSSION

Dietary Patterns and Macronutrient Intake

The relationship between diet and breast cancer risk is one of the most extensively discussed, yet persistently nuanced, areas in lifestyle epidemiology. Broadly, high-quality dietary patterns, such as the Mediterranean diet rich in fruits, vegetables, whole grains, and olive oil, are casually associated with a modest reduction in risk, particularly for postmenopausal breast cancer (9). This association is often superficially attributed to the anti-inflammatory and antioxidant properties of such diets. Conversely, Western dietary patterns, characterized by high intakes of red and processed meats, refined grains, and sugars, are frequently mentioned as potentially adverse. Specific macronutrients also receive significant attention. For instance, high saturated fat intake is often casually linked to increased risk in public discourse, though large pooled analyses and meta-analyses present inconsistent findings, with some suggesting a very modest positive association and others showing null effects (10). The evidence for dietary fiber, however, is more coherent, with a protective association consistently observed, likely mediated through modulation of estrogen metabolism and improved glycemic control (11). A recurring gap in this theme is the challenge of isolating individual dietary components from the overall dietary matrix and the lifelong pattern of eating, leading to often superficial and sometimes contradictory public health messages.

Alcohol Consumption and Tobacco Use

Among lifestyle factors, alcohol consumption stands out for the strength and consistency of its association with breast cancer, which is frequently cited as a well-established causal relationship. The mechanism is superficially attributed to the metabolism of ethanol into acetaldehyde and the concomitant increase in circulating estrogen levels. Epidemiological evidence robustly indicates a linear dose-response relationship, where each additional 10 grams of alcohol consumed per day (approximately one standard drink) is associated with a 7-10% increase in risk (12). This association holds across different types of alcoholic beverages, underscoring the role of ethanol itself. In contrast, the association with tobacco smoking is more complex and often superficially generalized. While active smoking, especially when initiated early and sustained over a long duration, is associated with a moderate increase in risk, the evidence is less straightforward than for lung cancer (13). The interaction with genetic polymorphisms in detoxification pathways further complicates a simple public narrative. The controversy here often lies in distinguishing the effects of active versus passive

smoking and accounting for the confounding effect of alcohol, as the two behaviors are frequently correlated.

Physical Activity, Sedentary Behavior, and Body Composition

These three interrelated factors form a core triad in lifestyle discussions on breast cancer. Physical activity, both recreational and occupational, is consistently and casually presented as protective. The association is observed for both pre- and postmenopausal breast cancer, with postulated mechanisms superficially including reduced adiposity, lower chronic inflammation, and improved sex hormone profiles. Meta-analyses suggest that the most active women have a 10-20% lower risk compared to the least active (14). Conversely, prolonged sedentary time, independent of physical activity, is increasingly mentioned as a potential independent risk factor, possibly linked to metabolic dysfunction. The most dominant theme in this category, however, pertains to body composition. Adult weight gain and obesity in postmenopausal women are strongly and causally associated with an increased risk of hormone receptor-positive breast cancer, primarily due to the peripheral aromatization of androgens to estrogens in adipose tissue (15). The relationship is inverse for premenopausal breast cancer, where obesity is associated with a lower risk—a nuanced point often omitted in superficial discussions. This paradox highlights a significant gap in public understanding, where body fatness is frequently presented as a universally negative factor without acknowledgment of the differential effects by menopausal status.

Exogenous Hormones and Reproductive History

The use of exogenous hormones, primarily through oral contraceptives (OCs) and menopausal hormone therapy (MHT), is a classic and widely discussed environmental exposure. The association is superficially understood as a function of adding exogenous estrogen and/or progestin, thereby increasing cumulative lifetime exposure. Current and recent use of combined OCs is associated with a small but statistically significant increase in relative risk, which dissipates after cessation (16). For MHT, the discussion is more stratified. The use of estrogen-plus-progestin therapy is clearly associated with an increased risk, a finding solidified by the Women's Health Initiative trial, while estrogen-only therapy appears to confer little to no increased risk for women with a hysterectomy (17). These distinctions are crucial but are often blurred in casual mentions. Reproductive factors like nulliparity, late age at first full-term pregnancy, and lower parity are also consistently featured as risk-enhancing factors, framed within the concept of lifetime estrogen exposure and breast tissue differentiation.

Environmental Chemical Exposures

This theme encompasses a wide array of factors that generate significant public concern but where epidemiological evidence is often less definitive than for lifestyle behaviors. Endocrine-disrupting chemicals (EDCs) such as bisphenol A (BPA), phthalates, and certain pesticides (e.g., DDT) are frequently mentioned due to their potential to mimic or interfere with endogenous hormones. Laboratory studies robustly show these compounds can exert estrogenic effects, forming the basis for the casual association. However, human epidemiological studies face substantial challenges in accurately assessing long-term, low-dose exposures, leading to inconsistent findings (18). Another major environmental factor is ionizing radiation, a well-established causal risk factor, particularly when exposure occurs during adolescence when breast tissue is undifferentiated. This is most clearly demonstrated from studies of survivors of atomic bombs and therapeutic medical radiation (19). Non-ionizing radiation, such as that from radiofrequency fields (e.g., mobile phones), is a topic of ongoing public debate, but to date, major reviews by bodies like IARC have found the evidence for carcinogenicity to be limited and no causal link to breast cancer established (20).

Circadian Rhythm Disruption and Night Shift Work

The disruption of the circadian system, primarily through night shift work, has emerged as a prominent theme in discussions on modern environmental risks. The superficial mechanism posits that exposure to light-at-night suppresses melatonin production, a hormone with oncostatic and anti-estrogenic properties, while simultaneously dysregulating core circadian genes involved in cell cycle control. Based on mechanistic plausibility and epidemiological data suggesting an increased risk

among long-term night shift workers, the IARC classified shift work that involves circadian disruption as "probably carcinogenic to humans" (Group 2A) (21). However, this classification remains a point of controversy. Recent large-scale cohort studies and meta-analyses have yielded mixed results, with some showing a modest positive association, particularly for long durations, and others finding no significant link (22). The inconsistency highlights gaps related to the precise definition of "shift work," the intensity and rotation of schedules, and the difficulty in controlling for potential confounding lifestyle factors that may cluster in individuals working non-standard hours.

Socioeconomic and Psychosocial Factors

While not traditional "lifestyle" factors in a behavioral sense, socioeconomic status (SES) and psychosocial stress are increasingly mentioned in holistic discussions of breast cancer etiology. Higher SES is inconsistently associated with increased incidence but lower mortality, a pattern largely attributed to differences in reproductive patterns (e.g., later age at first birth), greater use of MHT in the past, and, crucially, better access to screening (23). The association is therefore more reflective of detection and historical risk factor prevalence than a direct biological effect of wealth. The role of chronic psychological stress and adverse life events is a topic of enduring public interest, often linked superficially to immune dysfunction or elevated cortisol. However, epidemiological evidence for a direct causal link between stress and breast cancer incidence remains elusive and inconclusive, with studies plagued by methodological issues in measuring stress retrospectively and over the lifecourse (24). This represents a significant gap where public perception, fueled by anecdote and mechanistic plausibility, often outpaces the available scientific evidence.

This broad survey illustrates the vast landscape of factors casually mentioned in connection to breast cancer development. The strength and consistency of evidence vary dramatically, from the causal, dose-response relationship of alcohol and postmenopausal obesity to the highly plausible but epidemiologically complex associations for night shift work and EDCs, and further to the areas of persistent inconsistency or weak evidence such as specific dietary components and psychosocial stress. What emerges is a picture of a disease for which risk is woven from multiple, interacting threads of biology, personal behavior, and environmental context. Superficial associations, while useful for raising public awareness, often mask this complexity. The gaps and controversies identified—particularly the differential effects by menopausal status, hormone receptor subtype, and the challenges in measuring lifelong exposures—point to the limitations of current knowledge and underscore the need for more sophisticated, integrative approaches in future research, such as life-course epidemiology and exposomics, to move beyond casual association toward a more precise understanding of preventable risk.

Critical Analysis and Limitations

While the broad associations between lifestyle, environmental factors, and breast cancer are frequently discussed, a critical appraisal of the underlying literature reveals significant methodological constraints that temper the certainty of these casual links and complicate straightforward public health messaging. A fundamental limitation permeating this field is the overwhelming reliance on observational study designs, primarily cohort and case-control studies. The very nature of the exposures in question—diet, physical activity, chemical exposures—precludes the ethical and practical feasibility of large-scale randomized controlled trials (RCTs) with cancer incidence as an endpoint. Consequently, the evidence base is inherently correlational, making definitive causal inferences challenging. Observational studies are susceptible to unmeasured or residual confounding, a critical weakness where an unrecognized third variable influences both the exposure and the outcome. For instance, the association between high fruit and vegetable consumption and reduced risk may be confounded by a broader constellation of health-seeking behaviors, including regular screening, lower alcohol intake, and higher physical activity, which are difficult to fully account for statistically (25). This limitation is particularly acute for studies on socioeconomic status and psychosocial stress, where disentangling the biological effects of stress from correlated factors like poorer diet, limited healthcare access, and higher rates of smoking is immensely complex. Beyond overarching design constraints, specific methodological biases recurrently cloud the literature. Measurement error is a pervasive and often underappreciated issue, varying significantly across themes. In dietary research, the use of food frequency questionnaires

(FFQs) is standard but introduces recall bias and misclassification, as individuals struggle to accurately report long-term eating patterns. This non-differential misclassification typically biases results toward the null, potentially obscuring genuine associations, such as those for specific fats or food groups (26). Similarly, assessing exposure to endocrine-disrupting chemicals relies heavily on single or few point-in-time biomarker measurements (e.g., urinary BPA), which may poorly reflect chronic, low-dose exposure over the etiologically relevant time windows, which could span decades preceding diagnosis. This measurement variability leads to inconsistencies across studies and weakens the overall evidence base. Selection bias also poses a threat; large prospective cohorts, while valuable, often comprise volunteers who are more health-conscious and socioeconomically advantaged than the general population, potentially limiting the generalizability of findings to broader, more diverse populations (27).

Furthermore, the focus of much research on populations of European descent in high-income countries creates a significant gap in understanding how these risk associations manifest across different genetic ancestries and cultural contexts, where prevalence of risk factors and genetic susceptibility may differ. Publication bias represents another critical limitation that skews the published narrative. There is a well-documented tendency for journals to publish studies with statistically significant positive findings over those with null or negative results. This creates an inflated perception of the strength and ubiquity of certain associations in the scientific record. For example, early smaller studies suggesting a strong link between a specific dietary component and breast cancer risk may garner publication, while larger, later studies finding no association might struggle for visibility, creating a distorted literature that takes years to correct through cumulative meta-analyses (28). This bias is particularly relevant for novel or controversial topics, such as the effects of specific environmental chemicals or non-ionizing radiation, where a few positive studies can disproportionately influence public and scientific opinion despite a larger body of inconclusive evidence. The file drawer problem, wherein negative data remain unpublished, impedes a truly balanced synthesis and can lead to persistent myths or undue alarm regarding certain factors. The issue of generalizability is multifaceted and extends beyond selection bias in cohort recruitment. Many of the established associations, particularly those concerning reproductive factors and hormone therapy, were derived from studies conducted in specific historical and demographic contexts. The applicability of findings from studies on MHT use in the 1980s and 1990s to contemporary formulations and prescribing practices is uncertain. Moreover, the evolving nature of exposures—such as the rapid change in the chemical landscape of plastics or the patterns of night shift work in the gig economy—means that studies conducted even a decade ago may not accurately reflect current risk profiles.

The majority of evidence also pertains to invasive ductal carcinoma, with far less data on rarer but often more aggressive subtypes like triple-negative breast cancer, which may have distinct etiologies. This subtype-specific gap is a major shortcoming, as preventive strategies informed by data on estrogen receptor-positive disease may not be effective for all women (29). Finally, a profound limitation lies in the literature's frequent treatment of these diverse factors in isolation. The dominant research paradigm investigates single exposures, yet in reality, individuals are exposed to a complex mixture of lifestyle and environmental factors simultaneously throughout their lifecourse. The failure to adequately model these interactions—such as how diet might modify the effect of chemical exposures, or how physical activity might offset the risk associated with obesity—presents a simplified and potentially misleading picture of etiology. The emerging field of exposomics, which aims to assess all exposures from conception onward, represents a promising but methodologically daunting response to this limitation (30). In conclusion, while the reviewed literature provides a essential map of potential risk associations, its observational foundation, susceptibility to bias and confounding, problems with exposure measurement, issues of publication and generalizability, and reductionist approach collectively underscore that the casually mentioned links between lifestyle, environment, and breast cancer, while often biologically plausible, are supported by evidence of varying and often limited robustness. This critical perspective is vital for interpreting the evidence and directing future research toward more integrative and methodologically rigorous approaches.

Implications and Future Directions

The broad synthesis of casually associated lifestyle and environmental factors, tempered by a critical analysis of the literature's limitations, yields several concrete implications for practice, policy, and future scientific inquiry. For clinical practice, this overview serves not as a prescriptive checklist but as a framework for informed, nuanced patient counseling. Healthcare providers, particularly in primary care and oncology, are positioned to translate this complex landscape into actionable, individualized advice. The strongest evidence—supporting the causal roles of alcohol consumption and postmenopausal obesity—should form the cornerstone of preventive discussions. Clinicians can confidently advocate for limiting alcohol intake and maintaining a healthy weight through diet and physical activity as evidence-based risk-reduction strategies. However, the review equally underscores the necessity for caution. When patients inquire about more ambiguous associations, such as those with specific dietary chemicals or plastic use, clinicians can provide context, explaining the biological plausibility while clarifying the current limitations of human evidence and avoiding the propagation of undue anxiety or unsubstantiated claims. This balanced approach empowers patients to focus their efforts on modifiable factors with the strongest empirical support, thereby optimizing the clinical utility of lifestyle medicine in breast cancer prevention. At the policy and public health level, the findings highlight a clear hierarchy for intervention priorities. National and international health agencies should continue to strengthen and disseminate guidelines focused on population-level reductions in alcohol consumption and the combating of obesity through structural measures, such as fiscal policies on sugar-sweetened beverages, improved urban design to promote active living, and regulations on food marketing. The evidence for these factors is sufficiently robust to justify such investments. For more contentious areas like environmental chemicals, the precautionary principle may guide policy in the absence of definitive carcinogenic proof in humans, particularly when interventions also confer other health or ecological benefits. For instance, policies that reduce the population’s exposure to endocrine-disrupting chemicals through stricter material regulations can be advocated not solely on breast cancer grounds but as part of a broader commitment to environmental health. Furthermore, the persistent association of night shift work with risk, despite ongoing controversy, suggests that labor policies should consider minimizing circadian disruption where feasible and ensuring that workers in such roles have equitable access to regular health screening, acknowledging their potential status as a higher-risk group within occupational health frameworks.

The critical analysis naturally reveals substantial unanswered questions and research gaps that must steer the future agenda. A paramount gap is the need for a more nuanced understanding of how these factors interact with tumor biology, particularly breast cancer subtypes. Future research must move beyond aggregating all breast cancers and instead interrogate whether associations differ for hormone receptor-positive versus triple-negative diseases, as early indications suggest they might (31). The lifecourse timing of exposures represents another critical frontier. The influence of factors like diet, physical activity, or chemical exposure during childhood, adolescence, pregnancy, or menopause may be profoundly different, yet most epidemiological studies capture exposure only in mid-to-late adulthood. Unraveling these critical windows is essential for developing life-stage-specific prevention strategies. Additionally, the role of the gut microbiome as a potential mediator between lifestyle factors (diet, antibiotics) and breast cancer risk is an emerging field ripe for exploration, potentially explaining some of the inconsistencies observed in nutritional epidemiology (32). To address these gaps, future research must employ more sophisticated and rigorous study designs. While RCTs for most primary exposures remain impractical, innovative methodologies can strengthen causal inference. Mendelian randomization studies, which use genetic variants as proxies for modifiable exposures, offer a powerful tool to limit confounding and reverse causality, providing stronger evidence for or against causal relationships for factors like alcohol, BMI, and circulating vitamin levels (33). There is also a pressing need for large, well-characterized prospective cohorts that begin enrollment in early life and incorporate repeated, high-fidelity exposure assessments. Such cohorts should employ biomonitoring for chemical exposures, accelerometry for physical activity and sedentary behavior, and dietary biomarkers alongside traditional questionnaires to minimize measurement error. Investment in the science of the exposome is crucial; utilizing high-resolution mass spectrometry and advanced data analytics to characterize the totality of environmental exposures will move the field beyond a one-chemical-at-a-time approach to a more holistic understanding of mixture effects (34). Finally, fostering international consortia that pool data from

diverse populations across different geographic and socioeconomic contexts is imperative to ensure findings are generalizable and to understand how genetic ancestry and cultural practices modify risk associations. By integrating these advanced methodologies, future research can transform the current landscape of superficial associations into a precise, mechanistic, and actionable map for the primary prevention of breast cancer.

CONCLUSION

In conclusion, this narrative review has broadly mapped the extensive terrain of lifestyle and environmental factors casually associated with breast cancer development, revealing a hierarchy of evidence that ranges from well-established causal relationships to more speculative links. The synthesis underscores that factors such as alcohol consumption, postmenopausal obesity, and physical inactivity carry the most robust and consistent epidemiological support, while associations involving specific dietary components, endocrine-disrupting chemicals, and psychosocial factors are often supported by biological plausibility but remain mired in inconsistent human data and methodological challenges. The overall strength of the existing literature is therefore mixed; it provides a compelling, population-level narrative about modifiable risks but is fundamentally constrained by its observational nature, susceptibility to confounding, and frequent inability to account for the complex interplay of exposures over a lifetime. Given this landscape, the most pragmatic recommendation for clinical practice and public health is to prioritize communication and intervention on the factors with the strongest evidence base—specifically, advocating for reduced alcohol intake, the maintenance of a healthy body weight, and regular physical activity—as these actions offer the clearest path to risk reduction without awaiting further proof. Simultaneously, this review necessitates a clear call for more sophisticated, longitudinal research employing exposomic frameworks, lifecourse perspectives, and advanced causal inference methods to move beyond superficial association toward a definitive understanding of how modern living shapes breast cancer risk, ultimately enabling more precise and effective primary prevention strategies.

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