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CONTRACEPTIVE CHOICES AND CANCER RISK: UNDERSTANDING THE LINK BETWEEN OCPS AND BREAST CANCER

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Abstract

Background: Oral contraceptive pills [OCPs] have transformed reproductive health since their introduction in the 1960s, offering women autonomy and therapeutic benefits beyond contraception. However, concerns about a potential link between OCP use and breast cancer risk have persisted, particularly in light of evolving hormonal formulations and emerging immunological strategies in cancer prevention. Objective: This review aimed to evaluate the association between oral contraceptive use and breast cancer risk, considering key modifying factors such as duration, age at initiation, hormonal potency, genetic predisposition, and familial history. Methods: A systematic search was conducted using Google Scholar, Scopus and PubMed to identify relevant studies published between 2000 and 2020. From an initial pool of 23 articles, 8 peer-reviewed studies were selected based on inclusion criteria focused on quantitative risk assessment and detailed reporting of OC exposure. Data were extracted and synthesized narratively to highlight patterns and subgroup-specific risks. Results: The cumulative evidence indicates a modest but consistent increase in breast cancer risk associated with oral contraceptive use, particularly among women who began use at a young age, used OCs for extended durations, or had a genetic predisposition [e.g., BRCA1/2 mutations]. High-dose estragon and progestin formulations were linked to higher risk estimates [ORs up to 3.1], while modern low-dose pills appeared safer. Risk was elevated among recent users and those

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with a first-degree family history of breast cancer. Regional data from non-Western populations, such as Jordan, supported the generalizability of these findings. Conclusion: Oral contraceptive use is associated with a moderately increased risk of breast cancer, especially in high-risk subgroups. While the absolute risk remains low for most users, personalized contraceptive counselling is essential. Ongoing refinement of OC formulations and consideration of individual risk profiles can help optimize safety while maintaining contraceptive efficacy. Further research should explore how hormonal exposure may intersect with novel immune-preventive strategies, such as breast cancer vaccines, to inform future directions in women's health.

Keywords: Oral contraceptives, Breast cancer, Hormonal contraception, Estrogen and progestin, Cancer risk, BRCA1, BRCA2 mutations, Family history.

Introduction

Oral contraceptive pills [OCPs], introduced in the 1960s, revolutionized reproductive health and women's autonomy by providing effective means of birth control. Over the decades, their widespread use has extended beyond contraception to encompass therapeutic applications in conditions such as dysmenorrhea, polycystic ovary syndrome [PCOS], and endometriosis. However, concerns have persisted regarding their potential association with various cancers, particularly breast cancer. Simultaneously, advances in immunotherapy have ushered in a promising frontier in the fight against breast cancer: therapeutic vaccines. The intersection of hormonal contraceptive use and immunoprophylactic strategies raises critical questions in women's health, demanding a nuanced understanding of risk, benefit, and emerging preventive paradigms.

The relationship between OCP use and breast cancer has been the subject of extensive epidemiological research. Numerous studies have identified a modestly elevated risk of breast cancer among recent and long-term users of oral contraceptives. For instance, Trivers et al. found that recent OC use was associated with a higher likelihood of developing breast cancer in women aged

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20 to 54 years (Trivers et al.)[9]. Similarly, Rosenberg et al., in a comprehensive case-control study, reported a statistically significant increase in breast cancer risk with OCP use, particularly in younger women and those with prolonged exposure (Rosenberg et al.){10}.

The risk appears to be influenced by various factors, including the age at first use, duration of use, and hormonal composition of the pills. Pike et al. observed that women who began using OCPs at an early age or used them before their first full-term pregnancy were at increased risk (Pike et al.)[11]. However, these risks tend to decline after cessation of use. A study by Michels et al. revealed that the elevated risk of breast cancer associated with OCPs diminished within 10 years after discontinuation (Michels et al.)[12].

Moreover, the impact of OCPs on cancer risk is not uniformly negative. Some formulations offer a protective effect against ovarian and endometrial cancers, highlighting the complex interplay of estrogen and progestin on different tissues. Kamani et al. reviewed the dual nature of OCPs, emphasizing their role in reducing the incidence of certain gynaecological cancers while raising concerns over breast cancer, particularly in high-risk populations (Kamani et al.)[13]. The evolving formulations of OCPs—with lower hormone doses—may offer improved safety profiles compared to earlier high-dose variants.

While risk evaluation remains central to contraceptive counselling, the landscape of breast cancer prevention is being transformed by emerging immunological interventions, most notably therapeutic vaccines. Unlike prophylactic vaccines such as the HPV vaccine, breast cancer vaccines aim to train the immune system to recognize and destroy cancerous cells by targeting tumor-associated antigens. Candidates like HER2/neu-derived peptides, MUC1, and mammaglobin are currently under investigation in clinical trials, showing promise in eliciting tumor-specific immune responses with limited systemic toxicity.

Breast cancers are especially relevant for individuals at high risk due to genetic predispositions [e.g., BRCA mutations] or familial history. Their development complements traditional treatments like chemotherapy and hormone therapy, offering a novel strategy to prevent recurrence and possibly provide long-term remission. Some vaccine approaches aim to prime T-cell responses, while others

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utilize dendritic cells or viral vectors to enhance antigen presentation. As noted in clinical evaluations, the efficacy of vaccines is often improved when used in conjunction with immune checkpoint inhibitors, suggesting a future of personalized, combinatorial cancer immunotherapy.

The potential convergence of these two domains—OCP use and immunization—raises intriguing possibilities and challenges. Could long-term hormonal modulation influence the efficacy of cancer vaccines? What implications might hormonal contraceptive use have for immune surveillance in breast tissue? These questions remain open for investigation but underscore the importance of integrated, multidisciplinary approaches to women's health.

In summary, while oral contraceptive pills offer significant reproductive and gynaecological benefits, they are not without risks, particularly concerning breast cancer. However, with ongoing refinement in hormonal formulations and increasing awareness of individualized risk factors, these risks can be managed judiciously. At the same time, breast cancer vaccines represent a paradigm shift in oncology, offering new hope for prevention and treatment. Future research must address the intersection of these areas, ensuring that advancements in one domain do not inadvertently undermine progress in another.

This review aims to investigate the relationship between oral contraceptive use and breast cancer risk, with a focus on identifying how factors such as duration of use, age at initiation, hormonal composition, genetic predisposition, and family history may influence this association.

Methodology

A systematic search was conducted to examine the relationship between oral contraceptive [OC] use and breast cancer risk. The search strategy involved screening research articles published in English using reputable academic databases, primarily **Google Scholar , Scopus** and **PubMed**, between the years 2000 and 2020. A total of **23 studies** were initially identified based on relevance to the topic, using keywords such as "oral contraceptives," "birth control pills," "breast cancer risk," and "hormonal contraception." After reviewing titles, abstracts, and full texts, **8 studies** were selected that met the inclusion criteria.

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These criteria required that studies be peer-reviewed, report quantitative risk estimates [e.g., odds ratios, relative risks, or hazard ratios] for the association between OCs and breast cancer, and include clear methodological detail on OC exposure [duration, timing, or formulation]. Studies that did not focus on breast cancer as a primary outcome or lacked stratified OC data were excluded. Additionally, **3 duplicate reports** involving overlapping study populations were identified and excluded to avoid redundancy. Ultimately, **8 original and distinct studies** were included in the final analysis, offering a diverse range of populations and study designs, including meta-analyses, case–control studies, prospective cohorts, and genetic risk subgroup analyses. Data from each selected study were extracted manually, focusing on study design, population characteristics, OC usage parameters, and reported risk estimates. These findings were synthesized narratively to identify common patterns, subgroup differences, and risk modifiers.

Results

The cumulative evidence from 8 peer-reviewed studies suggests a consistent, though moderate, association between oral contraceptive [OC] use and increased risk of breast cancer [BC]. Across diverse populations and study designs—including case–control studies, cohort analyses, and meta-analyses—the risk of BC appears to be influenced by multiple factors such as duration of OC use, age at initiation, hormonal formulation, reproductive timing, genetic predisposition, and familial history of cancer.

Longer duration and recent use of OCs emerged as significant predictors of elevated breast cancer risk in several studies. Beaber et al. [1] found that women aged 20 to 44 who used OCs for 15 or more years had a significantly increased risk [OR = 1.5], and current use for five or more years raised the risk even further [OR = 1.6]. These risks were more pronounced in younger women and appeared to be higher for estrogen receptor–negative and triple-negative breast cancer subtypes, although the differences between subtypes were not statistically significant. Similarly, Kumle et al. [5], in a large prospective cohort of over 100,000 Norwegian and Swedish women, observed a relative risk of 1.6 among

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current or recent OC users, with comparable risks reported for both combined and progestin-only pills. These findings were reinforced by a nested case–control study by Beaber et al. [7], which linked recent OC use [within the past year] with an odds ratio of 1.5, particularly for estrogen receptor–positive cancers, though low-dose formulations were not associated with significant increases.

The timing of OC initiation relative to reproductive milestones also influenced breast cancer risk. Ji et al. [2] conducted a meta-analysis of ten studies, comprising more than 686,000 participants and over 8,500 BC cases, and confirmed a significant linear association between younger age at first OC use and increased BC risk. In line with this, Brohet et al. [3], analysing a cohort of BRCA1 and BRCA2 mutation carriers, reported that ever-use of OCs was associated with a 47% increased hazard of breast cancer [HR = 1.47], and that usage for four or more years before a woman's first full-term pregnancy notably raised the risk, especially among BRCA2 carriers [HR = 2.58]. This pattern was mirrored in the general population, where Kumle et al. noted a borderline significant increase in risk among short-term users before age 20 or first pregnancy. The findings collectively suggest that hormonal exposure prior to breast tissue maturation may have a more pronounced carcinogenic impact.

The potency and formulation of OCs were also shown to be significant modifiers of risk. Althuis et al. [4] reported that women using high-dose estrogen pills [>35 µg ethinylestradiol] had a nearly two-fold increased risk of breast cancer [RR = 1.99] compared to those using lower-dose formulations [RR = 1.27], with the strongest associations found in women under 35 years old [RR = 3.62 for high-dose use]. These findings were supported by Beaber et al. [7], who showed that specific formulations, such as triphasic pills containing 0.75 mg of norethindrone, and those with ethynodiol diacetate or high-dose estrogen, were associated with substantially increased risks [ORs between 2.6 and 3.1], whereas low-dose estrogen pills were not associated with significant risk increases.

Genetic predisposition, particularly among women with BRCA1/2 mutations, further amplified the risk associated with OC use. Brohet et al. [3,7] emphasized that OC use in BRCA mutation carriers increased breast cancer risk regardless of timing or age at use, although risk was notably greater when use occurred before

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first childbirth. The results suggest that exogenous hormone exposure has a more pronounced effect in genetically susceptible individuals, and that early, prolonged use further compounds this risk.

Familial history, even in the absence of BRCA mutations, also contributed to increased susceptibility. Grabrick et al. [7] reported that among women with a first-degree family history of breast cancer, those who had ever used OCs—particularly older formulations used before 1975—had a significantly elevated risk [RR = 3.3], whereas no significant association was seen in granddaughters, nieces, or women married into the families. This supports the role of gene–environment interaction and historical differences in hormonal concentrations as important contributors to risk stratification.

Geographical and ethnic variability was explored in a case–control study by Bardaweel et al. [8] conducted in Jordan, which found that regular OC use was associated with a significant increase in breast cancer risk [OR = 2.25]. Although the duration of use did not significantly affect outcomes in this population, other factors such as age at puberty, menopausal status, and family history were significantly associated with breast cancer, indicating that regional and cultural reproductive health patterns may mediate the effects of OC exposure.

Collectively, these studies indicate that oral contraceptive use is associated with a modest but non-negligible increase in breast cancer risk. The degree of risk varies by dosage, duration, timing of use, genetic background, and family history. While newer, lower-dose formulations appear to carry less risk than earlier high-potency pills, caution is still warranted, particularly for women with early OC initiation, long-term use, or a strong personal or familial predisposition to breast cancer. These findings support the need for individualized risk-benefit discussions in clinical decision-making regarding contraceptive choices.

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Website: usajournals.org

Table:1 OC Use and Breast Cancer Risk

S.no	Study (Author et al.)	Study Design	Population	Key Risk Factors	Risk Estimates
1	Beaber et al.	Case-Control	Women 20â€“44, USA	Long-term use (â‰¥15 yrs), current use, ER-/TNBC	OR = 1.5â€“1.6
2	Ji et al.	Meta-Analysis	Global (686,305)	Early age at first use, dose response pattern	RR = 1.24
3	Brohet et al.	Cohort (BRCA1/2)	BRCA1/2 carriers	Use before first pregnancy, long-term use	HR = 2.58 (BRCA2), HR = 1.47
4	Althuis et al.	Case-Control	Women 20â€“44, USA	High-dose estrogen, high-potency progestins	RR = 1.99; RR = 3.62 (<35 yrs)
5	Kumle et al.	Prospective Cohort	103,027 Nordic women	Current/recent use, early use before 20 or pregnancy	RR = 1.6
6	Beaber et al. (formulations)	Nested Case-Control	Women 20â€“49, USA	Formulation-specific risk (triphasic, ethynodiol)	OR = 2.6â€“3.1
7	Grabrick et al.	Historical Cohort	US women with family history	Use before 1975, first-degree relatives	RR = 3.3
8	Bardaweel et al.	Case-Control (Jordan)	Jordanian women 18â€“65	Regular use, puberty, menopause, family history	OR = 2.25

OR = Odds Ratio, RR = Relative Risk, HR = Hazard Ratio.

Discussion

The findings from this review provide consistent evidence that oral contraceptive use is associated with a modest but significant increase in breast cancer risk, particularly in certain populations and under specific conditions of use. While the overall magnitude of risk remains relatively small in the general population, specific patterns emerge that highlight subgroups of women who may face a higher degree of susceptibility.

One of the most robust and recurrent findings across studies is that recent and prolonged use of OCs contributes to elevated breast cancer risk. Studies by Beaber et al. and Kumle et al. observed that current or recent users, particularly those who used OCs for ≥5 years, faced significantly increased odds [ORs and RRs ranging from 1.5 to 1.6] of developing breast cancer. This risk was especially pronounced among younger women aged 20–39 years and in those who initiated use before their first full-term pregnancy.

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The timing of use relative to reproductive milestones, particularly first full-term pregnancy, appears critical. Ji et al.'s meta-analysis and the cohort study by Brohet et al. among BRCA1/2 carriers confirm that initiating OC use at an early age and before childbearing significantly amplifies risk. The biological plausibility behind this is well-supported: breast tissue may be more vulnerable to carcinogenic effects of hormones before it undergoes full maturation during pregnancy.

Moreover, hormonal composition and potency of OCs are important factors. High-estrogen dose pills and those with high progestin potency were shown to confer greater risks, with ORs as high as 3.1 for some formulations [e.g., triphasic norethindrone] as reported by Beaber et al. and Althuis et al. These findings suggest that not all OCs carry equal risk and that modern low-dose formulations may offer a relatively safer alternative compared to earlier, more potent versions.

A particularly vulnerable group identified in this review are women with a genetic predisposition to breast cancer, especially BRCA1/2 mutation carriers. In this population, even short-term OC use before the first pregnancy was associated with substantially elevated hazard ratios, with BRCA2 carriers facing up to a 2.6-fold increased risk. These findings warrant special consideration in clinical decision-making for genetically predisposed individuals.

Family history of breast cancer, even in the absence of known BRCA mutations, also contributes to increased risk. As shown in Grabrick et al., women with a first-degree family history who used OCs—especially older, high-dose versions—faced significantly greater risks, underscoring a possible gene–environment interaction.

Importantly, regional data from Jordanian women [Bardaweel et al.] support these findings, suggesting that the increased risk associated with OCs is not confined to Western populations, though cultural and biological factors may influence the magnitude and nature of this risk.

Despite consistent findings, the absolute risk increase remains modest for most women, and OCs still offer significant benefits, including effective contraception, regulation of menstrual cycles, and protection against ovarian and endometrial cancers. Therefore, the decision to use OCs should involve individualized risk

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assessment, particularly in young women, those with a strong family history, or known genetic mutations.

Conclusion

This comprehensive review of 8 studies confirms that oral contraceptive use is associated with a modestly increased risk of breast cancer, particularly with early initiation, long-term use, high-dose formulations, and in genetically or familiarly predisposed women. These findings highlight the importance of personalized contraceptive counselling. While newer, lower-dose OCs may mitigate some of the risk, clinicians should carefully weigh the benefits and risks for each patient, especially those with early reproductive use or a strong familial/genetic background. Future research should continue to monitor evolving OC formulations and explore strategies to minimize risk while preserving contraceptive efficacy.

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