Exploring DDT's Interlinked Impacts on Maternal and Child Health: Hormonal Dynamics and the Intersection with Obesity and Breast Cancer

Arushi Neravetla, Trisha Sengupta, Raylene A Sulivar-Monis, Emily Son, and Meghaa Ravichandran.

Public Health and Health Sciences Division at Undergraduate Laboratory, Berkeley

University of California, Berkeley

❖ Abstract:

Our study focuses on the impact of the pesticide DDT on maternal and child health, specifically in relation to obesity and breast cancer. The objective is to investigate the interdependence of obesity and breast cancer resulting from DDT exposure on a hormonal level, particularly estrogen, and to understand the association between DDT exposure and maternal and child health. The methodology employs a meta-analysis approach, analyzing independent studies on DDT's impacts on obesity and breast cancer, and examining the correlation between maternal DDT exposure and obesity, as well as the impact of obesity on breast cancer gene mutations. The study population primarily consists of mothers and their newborn children from the United States, with a focus on the persistence of DDT in regions such as South America, Africa, and Asia. Additionally, rodent studies were analyzed to see the impacts of DDT on generational development and reproduction. The data analysis is drawn from primary sources of similar research studies published in journals or scholarly websites, with a focus on the reliability and validity of the data. The results indicate a significant association between DDT exposure and obesity, as well as an increased risk of breast cancer, particularly estrogen receptor-positive cancer, in both maternal and child health. The report suggests a correlation between DDT exposure and heightened susceptibility to transgenerational obesity and breast cancer. However, it emphasizes the imperative for additional research endeavors and regulatory initiatives aimed at exploring alternative solutions to DDT and comprehending its multigenerational ramifications regarding disease.

❖ Introduction:

Dichloro-diphenyl-trichloroethane (DDT) was the first modern synthetic insecticides, developed to combat insect-borne diseases like malaria and typhus (EPA) (NPIC) (CDC). Though its effectiveness in insect control for agriculture, in 1972, mounting evidence of its adverse environmental and human health risks led to its ban in the US. In South America, Africa, and Asia, where malaria remains a major health issue, DDT is continually utilized as the World Health Organization declared that the benefits of the pesticide outweigh the environmental and health risks. Studies have shown that exposure to DDT causes adverse health risks regarding carcinogenicity, the nervous system, obesity, and more. For mothers, DDT poses an increased risk as noticeable levels have been found to be present in a mother’s milk, blood, and neonatal blood (Thuy, 2015).

Regarding obesity, the risk of being obese is increased during prenatal exposure to DDT, supported through in vivo evidence (Cano-Sancho, 2017). One study found that a grandmother's exposure to DDT increased the presence of obesity in their granddaughters (Cirillo, 2021). Current limitations include lack of concrete predictions between DDT exposure in utero and breast cancer, although in utero exposure to diethylstilbestrol, another xenoestrogen like DDT, is linked to an increased breast cancer risk (Cohn, 2015). In fact, these studies ultimately claim that DDT has been examined as a contributor to environmental issues and health concerns, exploring the potential role and concern of harm toward obesity and breast cancer risk as previously stated. The mechanisms through which DDT might influence obesity are not fully understood but may involve disruptions in hormonal regulation and metabolism.

In relation to generational breast cancer risk, the field of epigenetics investigates how environmental factors can influence gene expression without altering the underlying DNA sequence (Kabasenche, 2019). Limitations in research examining the extent to which obesity influenced by DDT impacts the risk of generational breast cancer include the multifactorial combination of genetics and lifestyle that isolate the specific impact of DDT on obesity and breast cancer and the lack of engagement and data collection over time concerning DDT. Therefore, we aim to explore specifically the pesticide DDT and its impact on maternal and child health by focusing on DDT's association to two diseases and their interdependence: obesity and breast cancer.

❖ Methods:

Design: To investigate the interdependence of obesity and generational breast cancer, several independent studies on DDT's impacts on obesity and breast cancer will be analyzed and compared. These independent studies show how maternal DDT exposure is associated with obesity and how obesity increases the risks of breast cancer gene mutations. Subsequently, through cross-analysis of the data, the extent obesity acts as a driving force for generational breast cancer is revealed. In conclusion, a meta-analysis will be produced through the comparison and cross-analysis of independent studies on obesity and breast cancer.

Area of Study and Why?

Several factors contribute to why DDT is still present in areas such as South America, Africa, and Asia. We decided to investigate these regions in our meta-analysis. In these regions, mosquito-borne diseases such as malaria are endemic, and DDT has historically been successful in effectiveness and affordability when combating mosquito populations. Not only that but DDT is easily transitable and remains in natural environments for long periods of time, which leads to accumulation of pesticide within the food chain. Therefore, since it has long latency in environments, we also studied populations in the United States, although DDT was banned in 1972. Toxicology reports have highlighted the environmental persistence of the pesticide's bio

accumulative nature, and associated risks to human health and ecosystems. Regulatory efforts aim to balance the need for disease control, but challenges remain in addressing DDT alternatives.

Data type: When researching the impact of DDT on the prevalence of obesity and generational breast cancer amongst child and maternal health, the analysis conducted was drawn from primary sources of similar research studies published in journals or scholarly websites. Secondary sources used include textbook information to help better understand the history and negative consequences of DDT. The reliability and validity of the data can be attributed to the number of primary sources used in data collection as it was the principal method in which analysis was conducted regarding the interdependence of obesity and generational breast cancer. We aimed to analyze both quantitative and qualitative scientific literature.

Population of study:
Various populations were surveyed to collect data about the interconnectedness of DDT with breast cancer and obesity on a hormonal level. The primary populations sampled were mothers, and their newborn children. The primary country that they originated from is the United States, and the range of ages of women surveyed ranged from 14 years to 90, and the ages of children surveyed ranged from daughters to grandchildren. Additionally, grandmother and granddaughter mouse populations were investigated to see DDTs impact on maternal health and future generations.

❖ Results:

Data Analysis:

Based on the data collected, our collection came from different sources that were analyzed for discussion. Appropriate figures have shown that exposure to DDT can affect maternal mortality and average age mostly affected are 49–50-year-old women. For example, for the figure on the right, the distribution of methylation differences of DDT exposure indicates that prenatal DDT exposure is linked with DNA methylation that is potent to increase the likelihood of breast cancer. Consequently, prenatal DDT exposure also leads through an alteration of genes that also is associated with breast cancer, ultimately increasing the association between DDT exposure and breast cancer.

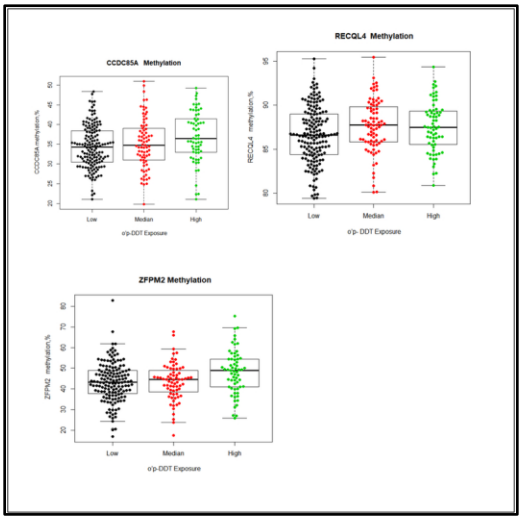


Figure 1: DNA Methylation (Three Methyl Groups Represented)

This table demonstrates this, where there is a threefold increase of breast cancer for women who were exposed to DDT before the age of 14. Specifically, DDT has been shown to have a correlation with an increase in activating estrogen receptors.

Breast cancer typically arises from disruptions or mutations on the BRCA1 and BRCA2 genes, and BRCA2 tumors are associated with ER + (estrogen receptor positive). Therefore, higher levels of estrogen from exposure to DDT can potentially be linked to maintaining the vitality of the cancer cells depending on estrogen to survive.

Table 2.

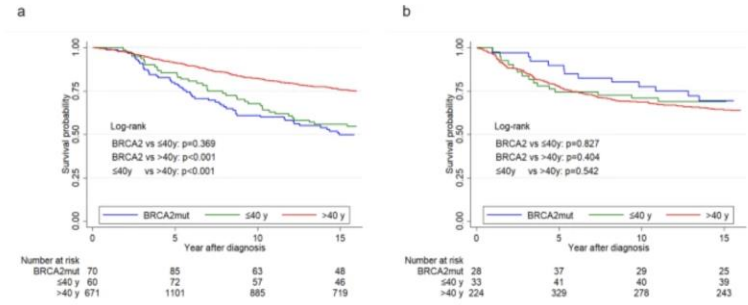
Association of *p, p'*-DDT with breast cancer stratified by age of "first exposure" in 1945, the year DDT became widely available in the United States for two prospective case-control samples nested within the Child Health and Development Studies cohort

Model	Current case-control sample (n = 153 cases/432 controls) for early postmenopausal breast cancer diagnoses from ages 50 to 54 y				Prior case-control sample (n = 129 cases/129controls) for premenopausal breast cancer diagnoses before age 50 y Cohn et al., 2007 (5)		
	All ages	Younger than age 3 y	Age 3 y and older	$P_{interaction\ with\ age}$[3]	All ages	Younger than age 14 y[4]	Age 14 y and older[4]
	OR (95% CI)	OR (95% CI)	OR (95% CI)		OR (95% CI)	OR (95% CI)	OR (95% CI)
Model 1: includes all DDTs[2]							
log$_2$(*p, p'*-DDT)	1.95 (1.34 to 2.83)	0.69 (0.26 to 1.79)	2.71 (1.72 to 4.27)	.01	—	—	—
Model 2: excludes DDE[2]							
log$_2$(*p, p'*-DDT)	1.99 (1.48 to 2.67)	0.56 (0.26 to 1.19)	2.83 (1.96 to 4.10)	.01	—	—	—
Model 3: DDT tertiles[2]							
Tertile 1	1.00 (Reference)	1.00 (Reference)	1.00 (Reference)	—	—	—	—
Tertile 2	0.97 (0.59 to 1.60)	0.11 (0.01 to 0.91)	1.30 (0.75 to 2.25)	.07	—	—	—
Tertile 3	1.52 (0.83 to 2.77)	0.10 (0.01 to 0.96)	2.17 (1.13 to 4.19)	.02	—	—	—
Model 4: DDT tertiles[2]							
Tertile 1	—	—	—	—	1.00 (Reference)	1.00 (Reference)	1.00 (Reference)
Tertile 2	—	—	—	—	1.92 (0.93 to 3.97)	2.77 (1.13 to 6.84)	0.68 (0.14 to 3.30)
Tertile 3	—	—	—	—	2.79 (1.15 to 6.72)	5.42 (1.71 to 17.19)	0.62 (0.12 to 3.18)

Open in a separate window

Table 1: Breast Cancer Exposure at Different Age Groups

Fig. 2: Breast cancer-specific survival according to ER status.



a ER-positive cancer. **b** ER-negative cancer. ER estrogen receptor. BRCA2 carriers of the Icelandic founder mutation 999del5.

Figure 2: Breast-Cancer Specific Survival Based on ER-Status on BRCA 1 & BRCA 2.

From the figures above, we found having ER+ breast cancer trends towards an increase in the risk in familial relatives and their risk of breast cancer and directly impacts both maternal and child health, and future generations. DDT also has significant links to increasing the risk of generational obesity, and obese postmenopausal women also had a correlation to having ER+ breast cancers. Studies have demonstrated that obesity can impact mitogen compounds in the body, impact insulin resistance, and disturb tissue through inflammation, impacting the risk of breast cancer (Zuo, 2021). This also works the other way around, where hormone receptor

positive cancer has had ties to weight gain and the risk of obesity, demonstrated by Table 1 below.

After finding these studies showing links to each other in terms of the impact of DDT on hormones and its association with diseases, specifically breast cancer and obesity, we determined that the impacts of DDT in these diseases could be interlinked. Many studies analyze the impacts of DDT on aspects like hormones and disease independently, but we aim to connect the concepts together with these findings. Females that are over 50 years of age with an overweight body mass index (BMI) have a stronger association to being diagnosed with breast cancer as there is a low p-value of 0.023, suggesting an association between both factors. Race is also shown to play a role in the association between high BMI and breast cancer diagnosis as the p-value is low at 0.003, below the cutoff of 0.05 demonstrating a link between obesity and breast cancer diagnosis regarding race. Additionally, we found that there is a strong association between fetal/neonatal complications and an increased BMI for mothers. As seen in the data, it looks that BRCA2 tumors are ER+, this type of breast cancer impacts maternal health including generational obesity, which DDT is also linked to. Postmenopausal women who are obese have a correlation to ER+ breast cancer. Lastly, we continued to analyze the generational aspect of DDT exposure in obesity in maternal and child health. There are multiple studies signifying the association between maternal health BMI and child obesity.

Table 1

Characteristics of included women at diagnosis (n = 140) from the Breast Cancer Program Longitudinal Repository (BCPLR) at Johns Hopkins University.

Characteristic	Total	Healthy/Underweight (BMI < 25)	Overweight (25 ≤ BMI < 30)	Obesity (BMI ≥ 30)	*p*-Value
	N (%)	*N* (%)	*N* (%)	*N* (%)	
BMI Group	140 (100)	40 (28.6)	53 (37.9)	47 (33.6)	
Age at Diagnosis (years)					
Mean (SD)	57.2 (12.4)	53.6 (13.1)	58.9 (12.6)	58.5 (11.3)	0.094
≥50 years	97 (69.3)	21 (52.5)	41 (77.4)	35 (74.5)	0.023
Sex					
Female	140 (100)	40 (100)	53 (100)	47 (100)	
Marital Status					0.676
Divorced	12 (8.6)	3 (7.5)	4 (7.5)	5 (10.6)	
Married	97 (69.3)	28 (70)	40 (75.5)	29 (61.7)	
Single	31 (22.1)	9 (22.5)	9 (17.0)	13 (27.7)	
Race					0.003
White	115 (82.1)	31 (77.5)	47 (88.7)	37 (78.7)	
Black	18 (12.9)	3 (7.5)	5 (9.4)	10 (21.3)	
Asian	7 (5)	6 (15)	1 (1.9)	0 (0)	
Ethnicity					
Hispanic	1 (0.7)	0 (0)	0 (0)	1 (2.1)	0.373
Family History					
Ovarian Cancer	5 (3.6)	0 (0)	2 (3.8)	3 (6.4)	0.277
Breast Cancer	29 (20.7)	7 (17.5)	15 (28.3)	7 (14.9)	0.214

Table 2: Characteristics of BMI ranges for women characterized in different groups.

Fetal/neonatal complications		
Fetal macrosomia (EFW ≥ 4500 g)	2.2 (1.6–3.1)	< .001
Shoulder dystocia	3.6 (2.1–6.3)	< .001
Birth weight < 4000 g	1.7 (1.4–2.0)	.0006
Birth weight < 4500 g	2.0 (1.4–3.0)	< .0001
Childhood obesity	2.3 (2.0-2.6)	< .05

Open in a separate window

95% CI, 95% confidence interval; EFW, estimated fetal weight; IVF, in vitro fertilization; OR, odds ratio; VBAC, vaginal birth after cesarean.

Table 3: Fetal and Neonatal Complications due to Obesity Complications

For example, the table above analyzes fetal/neonatal complications for mothers with a high body mass index, yielding a p-value of <0.05 in childhood obesity, which is significantly

low and can point to an association between maternal and child obesity. Additionally, the same goes for the increased risk of breast cancer with ER+ coming from exposure to DDT.

❖ Subsection 1: Breast Cancer Focus:

Estrogen disruptors, when they bind to and activate estrogen receptors, mimic the effects of natural estrogen even when natural estrogen is absent. This can lead to conditions like early puberty or cancer. Estrogen-like disruptors such as DDT are widespread in our environment and can significantly affect processes like maturation and cellular responses similar to natural estrogens (Calaf, 2020). Studies, in *vivo* and in *vitro*, have shown that DDT can interfere with estrogen signaling by affecting the parts of estrogen receptors that bind with the hormone. High levels of DDT have been associated with breast cancer in women who were exposed to it either during fetal development or before puberty. For instance, in breast cancer tissues and white blood cells, there have been observations of methylation silencing the BRCA1 gene (Calaf, 2020). Furthermore, environmental factors can cause epigenetic changes, altering DNA methylation levels without changing the DNA structure directly. Wu et al found three regions in DNA (known as DDT DMRs) affected by DDT exposure, which are related to growth, development, and susceptibility to breast cancer. In areas where DDT is used to combat malaria, such as Bangladesh, the isomer p, p'-DDT is transferred to breast milk when mothers breastfeed. This leads to elevated levels of DDT in breast milk and subsequently in the blood serum of infants. Research indicates a correlation between increased DDT levels in breast milk and higher rates of breast cancer (Subah, 2024). However, there is some inconsistency in the findings of population-based case-control studies retrieved from databases like PubMed and EMBASE. A meta-analysis of 35 case studies found that only 5 of them showed a significant increase in breast cancer risk associated with DDT exposure (Park, 2014).

❖ Discussion:

Our data shows that prenatal DDT exposure is linked with DNA methylation. DNA methylation is when methyl groups are added to DNA molecules, possibly changing the activity of the DNA segment. This alteration of genes by prenatal DDT exposure could cause mutations in breast cancer genes BRCA1 and BRCA2. Moreover, BRCA2 tumors are associated with estrogen receptor-positive breast cancers. However, exposure to DDT increases the activation of estrogen receptors. As the activation of estrogen receptors increases, levels of estrogen rise, and these high levels of estrogen allow the further development and growth of cancer cells. Hence, DDT exposure, by correlating with increased estrogen receptors, increases the vitality of cancer cells of BRCA2 tumors. As previously mentioned in the results, having estrogen receptor-positive cancer increases the risk in familial relatives. This means that women who have ER+ because of DDT exposure are more likely to pass the gene to their daughters, impacting both maternal and child health along with future generations. As described by Subah and Ryu, DDT

was found in breast milk, leading to an increased risk of breast cancer in Bangladeshi women. Children rely on breast milk as a main source of food, and their blood serum showed levels of DDT, increasing their own risk for cancer. This points to the idea of generational impacts of DDT, particularly in the realm of disease and breast cancer. However, there is variability in findings regarding the association between DDT exposure and breast cancer risk, as highlighted by the meta-analysis conducted by Park et al, 2014, which raises important questions and considerations within the scientific community. While some studies suggest a significant increase in breast cancer risk linked to DDT exposure, others do not find such a strong correlation.

❖ Subsection 2: Obesity Focus

The link between DNA methylation and obesity has been established. Through research on rodents, it has been discovered that Endocrine disruptors (EEDs) can potentially increase histone acetylation through the activation of acetylases and the oxidation of DNA bases, crucial for the mediation of transcription regulation of genes related to obesity and metabolic complications by ID3, a target of estrogenic receptors. Additionally, there is evidence suggesting that EEDs may imprint epigenetic changes on adipocyte progenitor or fat stem cells through ID3, contributing to the maternal programming of obesity in offspring exposed to these disruptors. It is hypothesized that EEDs induce ROS-mediated ID3 phosphorylation and acetylation, as well as histone acetylation and DNA base oxidation collectively, thereby controlling the expression of ID3 target genes involved in obesity and metabolic regulation. In a separate rodent study, it was observed that the F1 generation offspring directly exposed to gestating female rats from the F0 generation did not develop obesity, but rather exhibited various diseases in adulthood (Egusquiza, 2020). However, the F3 generation showed a significant increase in obesity rates, this transgenerational effect was seen to be induced by DDT exposure: the authors note over 50% of F3 and/or F4 offspring developing obesity following ancestral prenatal or perinatal exposure to obesogens. Despite these findings, however, the precise mechanisms behind these transgenerational effects remain unclear.

❖ Discussion:

Obesity is a chronic disease that has many consequences. Obesity has been shown to impact insulin resistance, cause inflamed tissue, and impact mitogen compounds in the body, especially in postmenopausal women. The condition is defined by an increase of excess fat deposits in the body (Glassman 2023). The research conducted by researchers Egusquiza and Blumberg provides valuable insights into the complex interplay between endocrine disruptors, such as estrogen-mimicking compounds and DDT, and their impact on obesity through epigenetic mechanisms and transgenerational effects. Specifically, they identified how EEDs can modulate the activity of ID3, a molecule targeted by estrogenic receptors, leading to epigenetic changes in adipocyte progenitor or fat stem cells. This mechanism suggests that environmental exposures to EEDs may contribute to the maternal programming of obesity in offspring, highlighting the intricate relationship

between environmental factors, epigenetics, and metabolic disorders. Parallelly, Egusquiza and Blumberg described the transgenerational effects of prenatal or perinatal exposure to obesogens, with a particular focus on DDT. Their findings revealed a noteworthy difference in the impact of DDT on generations: while the F1 generation offspring did not exhibit obesity but showed various diseases in adulthood, the F3 generation displayed a significant increase in obesity rates. This suggests a potential inheritance of metabolic traits across generations due to ancestral exposure to obesogens like DDT, rather than direct impact on the sister generation. However, it is important to note that the precise mechanisms driving these transgenerational effects remain unclear. The convergence of these studies underscores the intricate relationship between environmental exposures, epigenetic modifications, and obesity. Both endocrine disruptors and obesogens like DDT have been implicated in disrupting normal metabolic regulation through epigenetic mechanisms, potentially leading to transgenerational effects on obesity predisposition. Understanding the molecular pathways underlying these phenomena is crucial for developing targeted interventions to mitigate the adverse effects of environmental exposures on metabolic health and combat the rising prevalence of obesity-related diseases.

❖ Subsection 3: Results of Intersection of Obesity and Breast Cancer

It's well-established that having a higher Body Mass Index (BMI), whether one is classified as overweight or obese, increases the risk of developing breast cancer in women after menopause. Exposure to environmental chemicals from previous generations, even if they've been banned for decades, might affect the onset of puberty and obesity. These factors are known to contribute to the risk of breast cancer and other cardiometabolic diseases. Further supporting this, experiments with mice showed that exposure to a mixture of p,p'-DDT and o,p'-DDT during pregnancy led to obese offspring. Previous CHDS research has also linked maternal DDT exposure during pregnancy or shortly after birth with an increased risk of breast cancer in daughters, along with a higher prevalence of obesity among them. (Mirrell, 2020).

The focus of current research is on o,p’-DDT, which has previously been associated with breast cancer, obesity, and other health issues in daughters. It's believed to be the most sensitive biomarker for exposures before and after birth. Since exposure to o,p’-DDT can occur through a mother in utero egg cell development, levels of this chemical may predict exposure outcomes in future generations. Studies have also shown a significant increase in the risk of postmenopausal breast cancers, particularly in estrogen and progesterone receptor-positive cases. The relationship between BMI and breast cancer risk, as well as breast density, is complex. BMI is associated with higher breast fat and lower breast density, which impacts cancer risk. Examining the susceptibility window during mammary tissue development is crucial for understanding breast cancer risk. Surprisingly, higher BMI during pre-menopause and adolescence has been associated with a reduced risk of all breast cancer types. Research has consistently shown a

positive association between prenatal exposure to DDT pesticide metabolites and childhood obesity (Mirrell 2020). Studies also indicate a positive association between recent exposure to DDTs and weight-related issues in young children. The CHDS cohort's research further confirmed a link between o,p'-DDT and higher BMI and waist circumference in middle age.

❖ Discussion:

Elevated Body Mass Index (BMI) has been firmly established as a modifiable risk factor for breast cancer, particularly among postmenopausal women. The American Cancer Society reports a substantial number of breast cancer cases annually attributable to excessive body weight. Thus, the association between DDT exposure-induced obesity and breast cancer risk suggests a potential link between environmental exposures, obesity, and cancer development. Research from the Child Health and Development Studies (CHDS) cohort suggests that perinatal exposure to DDT may contribute to obesity later in life. This finding underscores a potential pathway through which environmental exposures, like DDT, can lead to obesity, a well-known risk factor for cancer. The mechanisms underlying the association between obesity and cancer risk provide insight into how DDT exposure-induced obesity may influence breast cancer development.

Hormonal factors, such as increased estrogen production in adipose tissue, chronic inflammation, insulin resistance, alterations in adipokine levels, and impaired immune function, all contribute to the pro-carcinogenic environment associated with obesity. These mechanisms create conditions favorable for cancer initiation, growth, and progression. Experimental studies involving mice suggest a transgenerational effect of DDT exposure on obesity risk. This implies that early-life exposures to environmental chemicals, such as DDT, may not only impact the exposed individuals but also affect future generations, potentially increasing the overall risk of obesity-related cancers, including breast cancer. The findings from the CHDS cohort highlight the potential link between DDT exposure-induced obesity and breast cancer risk. Understanding the mechanisms underlying this association provides insights into how environmental exposures and obesity interact to influence cancer development. Addressing modifiable risk factors like obesity through lifestyle interventions may help mitigate the risk of obesity-related cancers, including breast cancer, particularly in populations exposed to environmental chemicals like DDT. However, it is also highlighted that the current understanding of obesity as a risk factor is complex and has yet to be fully explored.

❖ Conclusion:

Through our comprehensive analysis of data on DDT exposure and its association with breast cancer and obesity, we have elucidated interconnected pathways that underscore the significance of early-life exposures and hormonal disruptions in disease development. Our research demonstrates the interplay between DDT exposure, hormonal changes, and disease outcomes, particularly breast cancer and obesity, across generations. By synthesizing findings from diverse studies, we have found evidence linking DDT exposure to alterations in hormone receptors, epigenetic modifications, and disease susceptibility, and some differences which could lead to new research. Furthermore, our meta-analysis has revealed the critical role of obesity in mediating the relationship between DDT exposure and breast cancer risk, highlighting the importance of considering both environmental and genetic factors in disease prevention and intervention strategies. Overall, our research contributes to a deeper understanding of the health implications of environmental pollutants like DDT and underscores the need for continued efforts to mitigate exposures and protect maternal and child health.

Works Cited:

Calaf, G., Cusi, R., Aguayo, F., Munoz, J., & Bleak, T. (n.d.). *Endocrine disruptors from the Environment Affecting Breast Cancer*. Oncology letters. https://pubmed.ncbi.nlm.nih.gov/32565930/

Cano-Sancho, G., Salmon, A., & Merrill, M. (n.d.). *Association between exposure to P,p'-ddt and its metabolite P,p'-dde with obesity: Integrated systematic review and meta-analysis*. Environmental health perspectives. https://pubmed.ncbi.nlm.nih.gov/28934091/

Cirillo, P., Mirrell, M., Cohn, B., & Krigbaum, N. (n.d.). *Grandmaternal perinatal serum DDT in relation to granddaughter early menarche and adult obesity: Three generations in the Child Health and Development Studies cohort*. Cancer epidemiology, biomarkers & prevention : a publication of the American Association for Cancer Research, cosponsored by the American Society of Preventive Oncology. https://pubmed.ncbi.nlm.nih.gov/33853850/

Egusquiza, R., & Blumberg, B. (n.d.). *Environmental obesogens and their impact on susceptibility to obesity: New mechanisms and chemicals*. Endocrinology. https://pubmed.ncbi.nlm.nih.gov/32067051/

Glassman, I., Le, N., Asif, A., Goulding, A., Alcantara, C. A., Vu, A., Chorbajian, A., Mirhosseini, M., Singh, M., & Venketaraman, V. (2023, August 14). *The role of obesity in breast cancer pathogenesis*. Cells. https://www.ncbi.nlm.nih.gov/pmc/articles/PMC10453206/

Kabansenche, W., & Skinner, M. (n.d.). *DDT, epigenetic harm, and transgenerational environmental justice*. Environmental health : a global access science source. https://pubmed.ncbi.nlm.nih.gov/25086599/

Keleher, M. R., Zaidi, R., Hicks, L., Shah, S., Xing, X., Li, D., Wang, T., & Cheverud, J. M. (2018, December 7). *A high-fat diet alters genome-wide DNA methylation and gene expression in SM/j mice - BMC genomics*. BioMed Central. https://bmcgenomics.biomedcentral.com/articles/10.1186/s12864-018-5327-0

Park, E., Min, S., Kim , Z., Yoon, C., Jung , K., & Nam , S. (n.d.). *Basic facts of breast cancer in Korea in 2014: The 10-year overall Survival Progress*. Journal of breast cancer. https://pubmed.ncbi.nlm.nih.gov/28382089/

Subah, Z., & Ryu, J. H. (2024a, January 19). *Impact of DDT on Women's Health in Bangladesh: Escalating Breast Cancer Risk and disturbing menstrual cycle*. Frontiers. https://www.frontiersin.org/journals/public-health/articles/10.3389/fpubh.2024.1309499/full

Thuy, T. (n.d.). Effects of DDT on environment and human ... https://www.jesoc.com/wp-content/uploads/2015/11/SS-42.pdf

Wu, H.-C., Cohn, B. A., Cirillo, P. M., Santella, R. M., & Terry, M. B. (2020, March). *DDT exposure during pregnancy and DNA methylation alterations in female offspring in the Child Health and Development Study*. Reproductive toxicology (Elmsford, N.Y.). https://www.ncbi.nlm.nih.gov/pmc/articles/PMC6710160/

Zuo, Q., Band, S., Kesavadas, M., & Madak Erdogan, Z. (2021, September 14). *Obesity and postmenopausal hormone receptor-positive breast cancer: Epidemiology and mechanisms*. OUP Academic. https://academic.oup.com/endo/article/162/12/bqab195/6370080