



Japan Bilingual Publishing Co.

Food and Drug Safety
<https://ojs.bilpub.com/index.php/fds>

ARTICLE

Impact on Hormone Disruption and Other Endocrine Abnormalities by Genetically Modified Foods and Other Contaminants in the Food Industry

Perla D. Serrano-López ¹ , Frida B. Ocampo-Juárez ¹, Alexandra Magaña-Rodríguez ¹,
 José A. Téllez-Morales ^{1,2*}

¹ Departamento de Ciencias de la Salud, Universidad ICEL/Campus La Villa, Mexico 07060, Mexico

² Departamento de Ingeniería Bioquímica, Escuela Nacional de Ciencias Biológicas/Instituto Politécnico Nacional, Av. Wilfrido Massieu, Esq. Manuel L. Stampa s/n, Gustavo A. Madero, Mexico 07738, Mexico

ABSTRACT

The use of chemicals in the food industry and their manipulation has become a common occurrence. This raises concerns about the potential risks associated with prenatal exposure to these substances. It is important to understand how they can affect the fetus without the mother's knowledge. The objective of this review was to examine the relationship between hormone disruption and other endocrine abnormalities in the context of the constant consumption of genetically modified foods and overexposure to chemicals used directly and indirectly in the agri-food industry. Conversely, the various classes of endocrine-disrupting chemicals (EDCs) have been demonstrated to disrupt endocrine homeostasis and are directly linked to the functioning of the different body glands, including the testicles, thyroid, ovaries, and so forth. However, there is also a possibility that they may be associated with an increased risk of gastrointestinal tract cancers, although this is not yet well-established. It can be hypothesised that the changes caused by genetically modified organisms (GMOs) may have metabolic effects in the medium and long term, such as the proliferation of adipocyte tissue. It can also be deduced that

*CORRESPONDING AUTHOR:

José A. Téllez-Morales, Departamento de Ciencias de la Salud, Universidad ICEL/Campus La Villa, Mexico 07060, Mexico; Departamento de Ingeniería Bioquímica, Escuela Nacional de Ciencias Biológicas/Instituto Politécnico Nacional, Av. Wilfrido Massieu, Esq. Manuel L. Stampa s/n, Gustavo A. Madero, Mexico 07738, Mexico; Email: josetellez@icel.edu.mx

ARTICLE INFO

Received: 26 December 2025 | Revised: 10 January 2025 | Accepted: 12 January 2025 | Published Online: 15 January 2025

DOI: <https://doi.org/10.55121/fds.v2i1.223>

CITATION

Serrano-López, P.D., Ocampo-Juárez, F.B., Magaña-Rodríguez, A., et al., 2025. Impact on Hormone Disruption and Other Endocrine Abnormalities by Genetically Modified Foods and Other Contaminants in the Food Industry. Food and Drug Safety. 2(1): 15–24.
 DOI: <https://doi.org/10.55121/fds.v2i1.223>

COPYRIGHT

Copyright © 2025 by the author(s). Published by Japan Bilingual Publishing Co. This is an open access article under the Creative Commons Attribution-NonCommercial 4.0 International (CC BY-NC 4.0) License (<https://creativecommons.org/licenses/by-nc/4.0/>).

EDCs cause chronic pathologies when they are consumed frequently or when they are present in the environment. It is of the utmost importance that the general population is aware of the existence of EDCs, as this will enable them to ascertain whether they have been exposed and, if so, to make a timely diagnosis and, consequently, an appropriate treatment.

Keywords: Food; Endocrine Disruption; Chemicals; Pathologies

1. Introduction

Endocrine-disrupting chemicals (EDCs) are exogenous chemicals or mixtures of these that alter the function of the endocrine system, resulting in a range of adverse effects^[1]. Despite the fact that genetically modified foods and EDCs were not introduced for that purpose, it is well documented that one of the most significant challenges facing humanity is to meet the global food needs of a growing population. This is precisely why a significant public health issue, given the discrepancy between global food production and demand, undernutrition is linked to three million child deaths annually^[2]. However, it is questionable whether providing food alone is sufficient to safeguard health. It is also unclear what constitutes “nourishment” for the body. It is important to note that dietary habits are among the most influential factors in determining whether an individual will age healthily and experience a good quality of life. This is distinct from merely living a long life. Although the majority of clinical trials and epidemiological studies yield inconclusive results, this review collates data pertinent to endocrine health and its correlation with dietary intake and the chemical substances that come into contact with food, including pesticides. The term “pesticide” is used to describe a group of chemical compounds that are used for the protection of crops from external agents, including insects, fungi, rodents, viruses, bacteria, and any microorganisms that may affect the correct development of crops. These can be divided into different groups, each with a specific purpose. Examples of these include organophosphates, carbamates, and pyrethroids. It is crucial to identify these risks, as approximately 150,000 individuals die annually from pesticide poisoning, particularly in low- and middle-income countries, for various reasons, including climatic conditions and inadequate use of personal protective equipment^[3]. It is also important to note that, in addition to the interactions with exogenous chemicals to which all crops are subjected, crops are sometimes modified in different ways, resulting in genetically modified organisms (GMOs).

Technologically, this has been an impressive advance, made possible by the developments in biotechnology and genetic engineering. These modifications are made with the aim of conferring beneficial characteristics on crops, including visual, nutritional, or resistance to the environment^[4]. However, as they provide significant benefits, they also have an impact on the ecosystem, introduce new pests and diseases affecting humans and animals^[5], and are met with unfavorable consumer sentiment, which will be discussed in greater detail later.

2. Prenatal Exposure

Prenatal exposure can be defined as the action of exposing a pregnant person to the effects of certain agents that may or may not reach the fetus. In some cases, this exposure can have negative consequences, which will be discussed in further detail below. Endocrine-disrupting chemicals (EDCs) have the capacity to either mimic or alter the functions of the hypothalamic-pituitary system and the actions of hormones. This is achieved through their interaction with hormone receptors and enzymes that are responsible for the production of steroids^[6]. Furthermore, recent years have seen a focus on epigenetic alterations during gestation that are associated with environmental factors, including exposure to EDCs. Given that these substances have hormonal or anti-hormonal effects, endocrine homeostasis is disrupted, which can also affect maternal and fetal metabolism. This can result in a range of complications, including ectopic pregnancies, spontaneous abortions, hypertensive disorders in the mother, and neonates with sizes that do not correspond to their gestational age^[7]. Another situation that warrants consideration is the presence of plastic micro particles in animals intended for human consumption. While plastic substances are not directly ingested by the human population, research has revealed that plastics are now ubiquitous throughout the food chain, including in aquatic animals, terrestrial animals, and even birds. This raises concerns about the potential

indirect impact on human health, given that these animals are commonly consumed as food. The presence of plastic particles in the food chain could, therefore, pose a public health risk^[8]. The precise number of species affected by microplastics (MP) and nanoplastics (NP) contamination remains unknown. However, it is possible to make an estimation based on the components of these substances, taking into account the most prevalent and harmful ones, such as phthalates, bisphenol (BPA), and polybrominated diphenyl ethers (PBDE). This is supported by the findings of Talness et al.^[9] have demonstrated that these substances can disrupt the endocrine system, mimicking anti-androgenic effects. This is exemplified by BPA and PBDE, which have been shown to alter thyroid function. In their study, perinatal exposure to these chemicals in male rodents resulted in a condition that closely resembles testicular dysgenesis syndrome in humans. This suggests that exposure to these substances, even in uncontrolled and unknown quantities, can lead to the development of pathologies, even before birth. In addition to food contamination, the sources of these plastics that act as EDCs are numerous. Their use in the food industry is extensive, for example, in plastic bottles or food containers. Furthermore, the amount of waste is growing, and as time passes and these materials degrade, they can even be inhaled, ingested in water, or contaminate other foods that have been in contact with them. This can result in the leaching of these chemicals into placental tissue and amniotic fluid^[10]. The presence of MP and NP in breast milk suggests that humans may consume persistent organic pollutants (POPs) during the first 1,000 days of their lives. This exposure could potentially lead to long-lasting alterations in the human epigenome^[11]. Similarly, it is crucial to acknowledge that epigenetic alterations can only occur when exposure occurs during an early developmental stage, specifically during prenatal development. This exposure results in a permanent alteration of the germline epigenome, which can then be transmitted to subsequent generations^[12]. An alternative explanation would be that the adverse effects of these chemicals would manifest in the F3 generation. The question then arises as to why this would occur. A pregnant female represents the F0 line and is exposed to an EDC,

which indicates that the germ line that is particularly susceptible to contaminants is the F1. Consequently, the germ cells will contribute to the F2 generation, which was also directly exposed to EDCs. Therefore, the first generation that is not in contact with EDC exposure by germ cells would be the F3^[13]. Although this is a hypothetical scenario, in today's industrialized society, it is nearly impossible for a pregnant woman not to be continuously exposed to any EDC.

3. Benefits of Genetically Modified Organisms and Agri-Food Chemicals

The combination of genes from different organisms is known as recombinant DNA technology. This technology offers great promise for addressing areas of great interest in the 21st century in relation to food. As with all technologies, many purported benefits have been raised, some of which may be perceived as implausible. This has led to the emergence of potential adverse consequences. The use of chemicals in food has a long history, but it was in the 1990s that their use in food products experienced a significant increase. This was a period of experimentation with food by governments for a variety of reasons, including the desire for increased color, resistance to certain herbicides, insects, viruses, bacteria, and others. These developments were influenced by organizations such as the FAO (Food and Agriculture Organization), the FDA (Food and Drug Administration), the CFIA (Canadian Food Inspection Agency), and others around the world. However, it should be noted that some of these organizations have reports with links that are not currently accessible. **Table 1** provides a general overview of the outcomes observed in genetically modified crops. It can be seen that the anticipated results are largely comparable across different crops, with some instances of identical outcomes. This suggests that the crops and their modifications, as they were "created," have met the expected success criteria. These innovations in the agri-food industry have resulted in increased crop yields, along with the mechanization and automation of agricultural production, in order to meet the needs of a growing population^[14].

Table 1. Comparison of expected results with genetic modifications in various crops in the 1990–2000s and in the last 5 years. Adapted from Amanullah et al.^[15] & Kuiper et al.^[16].

Crop	Past Trait	Present Trait
Canola	High lauric acid and herbicide resistant	Herbicide resistant
Cotton	Herbicide resistant	Insect resistant
Corn	Herbicide and insect resistant	Insect and herbicide resistant
Potato	Herbicide and insect resistant	Insect resistant
Soybean	Herbicide resistant and high- oleic acid	Herbicide resistant
Squash	Virus resistant	Virus resistant
Tomato	Insect resistant	Delayed ripening and insect resistant

4. Transgenic Foods and EDCs Related to Gynecological Pathologies

The most commercially viable genetically modified (GM) crops are corn, soybeans, cotton, and canola, according to Bawa & Anilakumar^[17]. In addition to their nutritional effects, many of these crops have been linked to genetically modified organisms (GMOs), according to Zhang et al.^[18], over the past few decades, three main types of GM food have been developed. However, there is a lack of literature examining the impact of these crops on our hormonal axis. This section will focus on the impact of genetically modified organisms (GMOs) on female hormonal health. As previously discussed, women of reproductive age are particularly vulnerable to prenatal exposure to environmental contaminants, which can have adverse effects on their health and that of their offspring. This underscores the importance of understanding the potential risks associated with GMOs,

which are not only genetically modified in their DNA but are also often supplemented with growth hormones (GH) in animal feed^[4, 19]. This is important because, like all hormones, GH has peaks, plateaus, and troughs in release and absorption, so if we continue to consume growth hormone that is not accurately made and synthesized by our body, it can cause irreversible damage to our DNA, since GH blocks the autophosphorylation of ataxia telangiectasia mutated kinase (ATM), which can subsequently lead to neoplastic epithelial growth progression^[20], it is also important to emphasize the rupture of the hypothalamus-pituitary-sexual gonads axis, which leads to hormonal imbalance, resulting in dysmenorrhea, irregular menstrual cycles, hormonal/adult acne, hirsutism, among others, which in most cases can be symptoms of other important gynecological pathologies^[21]. **Table 2** shows how some of the proven EDC chemicals present in the agro-food industry are related to female endocrine disruption.

Table 2. Persistent organic pollutant chemicals, phytoestrogens, agricultural production chemicals, consumable animal and industrialized chemicals in relation to female hormone disruption, adapted from Caserta et al.^[21].

Chemical(s)	Pathway of Exposure	Mechanism of Action
Polychlorobiphenyl (PCB)	Fat-rich food, (milk and derivatives, fried food), fatty fish, etc. living environmental	Alteration of steroid hormone metabolism/transport, ability to bind with the thyroxine transport protein, interaction with thyroid hormone receptors, neuroendocrine effects
Dioxins and dioxin-like PCB's	Fat-rich food, (milk and derivatives, fried food), fatty fish, etc. living environmental	Aryl hydrocarbon receptor interaction leading to altered steroid hormone metabolism and neuroendocrine effects including thyroid
DDT and metabolites	Fat-rich food, (milk and derivatives, fried food), fatty fish, etc. living environmental and workplaces (in developing countries)	Mainly estrogenic activity but also interaction with
Triazoles, imidazoles	Agricultural and zoo technical fungicides, living environmental (agricultural areas)	Effects on hypothalamo-hypophysis-gonadal (HHG) axis
ETU (metabolite of ethylene bisdithiocarbammates, benzimidazoles)	Agricultural and zoo technical fungicides, living environmental (agricultural areas)	Thyreostatic effects

Table 2. Cont.

Chemical(s)	Pathway of Exposure	Mechanism of Action
Bisphenol A (BPA)	Plastics in contact with food	Estrogen agonist-ER alpha
Polybrominated flame retardants	Fat-rich food, (milk and derivatives, fried food), fatty fish, etc... living environment, food containers	Interaction with pregnane X receptor (PXR) leading to altered steroid and thyroid hormone homeostasis
Cadmium	Refined foods such as flour, sugar and some seafood, etc.	Estrogen agonist-estrogen receptor (ER) alpha
Isoflavones, lignans, etc.	Modified vegetables, soy-based food	SERMs, high affinity for estrogen receptor (ER) beta

In the last 5 years the topic of endocrine disruption and its relation to gynecological situations has been of increasing interest, but despite this there is little information available about what actually happens in the human body with EDCs exposure, however, low dose BPA has been found to increase the incidence of multiocytic follicles and altered fetal ovarian steroidogenic expression and microRNAs that measure gonadal differentiation and folliculogenesis in sheep, and decreased the number of primordial follicles in mice^[22], this leads us to consider bisphenol A as an ovarian and uterine toxicant, although this does not mean that there is sufficient evidence on the effects of bisphenol A on the oviduct, placenta, and pubertal development. This is not a guarantee that its effects are not clear, as Yuan et al.^[23] did an *in vitro* study with ICR mice, where 100 µg/kg/day of BPA was administered, in addition to having a control group, this to know how it can be detrimental to embryo implantation, which in the end the results suggested that BPA can downregulate the expression of SGK1 and ENaCa proteins, through estrogen receptors in Ishikawa cells.

5. Antibiotic Resistance

It is estimated that approximately 80% of antibiotics sold in the United States are sold for livestock use as a pro-

phylactic measure to prevent future infections in animals that are not yet sick. This practice is a cause for concern, as some researchers have concluded that by 2050, antibiotic resistance will result in an estimated 10 million deaths per year, leading to economic losses of \$100 billion^[24]. Similarly, the confirmation of the existence of the gene for resistance to tetracyclines, sulfonamides, chloramphenicols, and macrolides is related by the study of cattle farms in eastern China^[25]. This suggests that the feedlot may serve as a significant reservoir for the dissemination of antibiotic resistance genes. The data presented in **Table 3** are cause for concern, particularly given that they were collected exclusively from regions with the highest incidence rates. What, then, is the nature of this concern? It has been demonstrated that clinical, veterinary, and livestock practices represent the primary factor associated with the emergence and dissemination of so-called “superbugs”^[26]. As previously stated, food is associated with socioeconomic circumstances that directly affect consumers and producers. This suggests that it may be related to the specific regions and data points under consideration. **Figure 1** illustrates a series of recommendations for the prudent utilization of antibiotics in food production, with the objective of mitigating the emergence of antibiotic-resistant pathogens.

Table 3. Residues of antibiotic groups found in manure from different farm animals, adapted from Tian et al.^[24].

Fecal Type	Antibiotic Residues (µg/kg)		
	Tetracyclines	Sulfonamides	Quinolones
Pig	Shanghai (12.27–18.70)	Shanghai (4.88–7.56)	Beijing (0.41–1.45)
Cattle	Shanghai (12.01–21.36)	Shanghai (4.57–9.36)	Three northeastern provinces (0.61–4.17)
Chicken	Jiangsu (8.9–65.7)	Jiangsu (0.75–2.18)	Jiangsu (8.73)



Figure 1. Recommendations for the responsible use of antibiotics in food production.

6. Effects of Pesticides on Food and Human Health

The advent of plant protection products has enabled the acceleration of food production over the past century. This is due to the necessity of increasing food production to nourish an ever-growing population, while simultaneously maintaining pressure on the intensive use of pesticides and fertilizers^[27]. In response to the growth in population, significant legislative amendments have been enacted in the United States in recent years with regard to pesticides. The U.S. Environmental Protection Agency has introduced a reevaluation review program, which has authorised the implementation of pesticide standards^[28]. Bretveld et al.^[29] posited that certain pesticides have the potential to interfere with female hormonal function, resulting in hormonal alterations. However, it is notable that the population exposed to pesticides is more prevalent among men, given that men are often the primary applicators, whereas women are exposed primarily through re-entry activities. Additionally, the ovarian cycle has not been as extensively studied as spermatogenesis. There are also indications that exposure to specific pesticides may induce ovarian dysfunction. The study revealed that in 20% of subfertile couples, the problem was predominantly male, while in 38% of cases, it was predominantly female. In 27% of cases, abnormalities

were observed in both the male and female partners. In the remaining 15% of cases, no obvious cause of subfertility could be identified^[29]. A significant number of contemporary pesticides have been demonstrated to possess hormonal activity, which has led to their classification as endocrine-disrupting chemicals (EDCs) according to Cremonese et al.^[30]. These chemicals exert their effects by binding to relevant hormone receptors, altering cell signaling pathways, directly affecting the central neuroendocrine system, inhibiting hormone synthesis, or causing toxic effects on relevant organs. In numerous regions across the globe, DDT continues to be employed in agricultural practices to bolster crop resilience against insect-related challenges. However, the low molecular weight and high water solubility of DDT permit its passage into food sources. In contrast, neonicotinoids are capable of penetrating plant tissues and being stored within them. These neonicotinoids have been observed to inflict damage upon the insect central nervous system. However, they may also exert adverse effects upon the human neurological system and testosterone^[31]. The use of organochlorine and organophosphorus pesticides has been linked to adverse effects on the reproductive system. These effects include alterations in sex hormone levels, delayed menstrual cycles, ovarian dysfunction, changes in ovarian weight, alterations in follicle growth, compromised oocyte viability, and modifications in the quality of spermatogenesis^[32]. A growing body of evidence suggests that prolonged exposure to specific pesticides, such as organophosphates, may result in adverse alterations to the aroma, taste, and nutritional profile of crops. This raises concerns about the nutritional value of pesticide-treated foods^[33]. As posited by Dahiri et al.^[34], the utilization of pesticides has facilitated the sustenance of global populations; however, this has been achieved through the cultivation of foodstuffs that, in the long term, may precipitate significant health concerns. Those most vulnerable to the development of diseases are, in particular, agricultural workers, who are at an elevated risk of developing cancer and experiencing adverse effects on fertility and pregnancy. In conclusion, further research is required to ascertain whether the phenomenon in question affects only a specific social group at a particular age. What has been established, however, is that the environment in which an individual is developing plays a significant role in determining the likelihood of the occurrence of pathologies.

7. Carcinogenic Effects and Tumor Development

As has been previously established, the various categories of EDCs have been demonstrated to disrupt endocrine homeostasis. Consequently, it is reasonable to hypothesize that they may exert a significant influence on the diverse spectrum of cancers that exist. The answer is affirmative, particularly in the case of hormone-dependent cancers, which are directly linked to the functioning of various body glands, including the testicles, breasts, thyroid, uterus, and so forth. It is also postulated that they may have a significant relationship with digestive tract cancers, although this remains unclear and understudied. It may be surmised that the changes induced by GMOs may have metabolic effects in the medium and long term, such as the proliferation of adipose tissue, which in turn releases adipocytokines, which have been demonstrated to promote tumor progression, given that numerous EDCs utilized in the agri-food industry are not metabolizable^[35]. The data collected above allows us to conclude that EDCs are a significant contributing factor to the development of endocrine pathologies in females. This is due to the fact that numerous EDCs act as agonists in estrogen receptors (ER), antagonists in androgen receptors (AR), and also interact with progesterone receptors (PR). One receptor (PR), in turn, triggers binding to the cytoplasmic aryl hydrocarbon receptor (AhR), which is of great relevance as it interacts with nuclear ER and AR receptors, which may or may not be activated at the time^[21]. Oogenesis posits that oocytes are the cells with the longest lifespan within our organism. They are born and die, yet never regenerate. Consequently, the environmental factors to which they are exposed are more challenging to quantify. Additionally, they are susceptible to significant damage, which further complicates the determination of an exact amount. The relationship between damage and complex pathologies such as cancer is a complex one. However, the epigenetic theory posits that breast cancer may be influenced by the deregulated and toxic growth of cells, which are themselves affected by daily exposure to exogenous substances^[36]. Despite the discrepancies in the studies examining the relationship between estrogen-dependent cancers and EDCs, arising from differences in sample size, population type, genetic variables, and life stage, a noteworthy finding emerges from a study conducted by Cohn et al.^[37]

where they realized all this and focused on studying the relationship between uterine and breast cancer with the use of dichlorodiphenyltrichloroethane (DDT). This study measured the incidence of cancer in daughters of women who had been exposed to DDT from a very early age and yielded significant findings, including the confirmation that DDT is an endocrine disruptor. The study identified cancer predictor and high-risk marker characteristics in women exposed to DDT before the age of 14, with a fivefold increase in cancer risk. This risk was even higher in women exposed before the age of four. Additionally, the study highlighted the lack of research on the incidence of estrogen-dependent cancers in humans exposed to endocrine-disrupting chemicals, including DDT, persistent organic pollutants (POPs), and BPA. In the 1970s, polybrominated biphenyls (PBBs) were utilized extensively in various industrial sectors as flame retardants, specifically to diminish the propensity of a given product to ignite. However, they were subsequently banned in the same decade following the discovery that PBBs had been mixed with certain foods of animal origin, including eggs, meat, and their derivatives. PBBs contaminated vast quantities of food and exposed 9 million individuals to contaminated foodstuffs. PBDEs, which were introduced as replacements, demonstrated minimal efficacy as EDCs. Consequently, numerous PBDEs have already been banned in certain regions of the USA^[38]. This is a significant finding, as research has demonstrated that various flame retardants can affect thyroid function^[39]. This is of significant importance, given that thyroid cancer is the fifth most prevalent cancer in women in the USA. Additionally, data from 2015 indicate that 62,000 diagnoses were recognized in both men and women^[40]. The question thus arises as to how EDCs may affect the thyroid to the extent of causing carcinogenesis. As elucidated by Marotta et al.^[41], numerous POPs and phenol derivatives can physiologically interact with the thyroid and its associated hormones in a manner that is analogous to the blocking of intracellular iodine reception and subsequent inhibition of iodine-tyrosinase transport, ultimately preventing its release into the bloodstream. Conversely, the absence of release into the bloodstream impedes the uptake of thyroid hormones in various bodily tissues. Additionally, it precludes the binding of thyroid hormones to active intracellular transport proteins, thyroid receptors, and their cofactors. This ultimately results in a detrimental impact on basal endocrine feedback loops,

which could potentially evolve into malignant processes. According to Bräuner et al.^[1], testicular cancer is the most common malignant neoplasm in males aged 15–44 years, and this is because the meta-analysis that was done concluded that organochlorines that led to post- and prenatal exposure in males were associated with a higher incidence of testicular cancer, and what is a dietary organochlorine? DDT and the results in this meta-analysis are quite similar to the review we had on female cancers associated with the same type of EDC. The etiology of testicular neoplasia is suspected to be of fetal origin, which would be one of the many reasons for banning DDT, and not only that, another organochlorine of concern would be dichlorodiphenyldichloroethylene (DDE), which also has a strong association with germ cell cancers such as seminoma^[42], and fortunately neither is currently used in the food industry, but EDCs have epigenetic effects^[12]. That is, these substances are able to alter our DNA and RNA sequence, with transgenerational effects.

8. Conclusions and Suggestions for Further Research

In summary, chemical substances added for decades have had an impact on the cultivation of food, where food was experimented with for various reasons, improving color, size, resistance to certain herbicides, insects, viruses, bacteria, etc., although there are currently organic crops, but in a smaller proportion. These chemicals have been shown to cause harm in hormone disruption and other endocrine abnormalities as detailed, however, some of these chemicals such as DDT are currently banned for use in agriculture. On the other hand, the increase in bacterial resistance is becoming more and more serious, and although measures have been taken for years, they are still not enough. Finally, future research should focus on how to eliminate microplastics, since they not only cause the death of animals, but also damage the biological functions of the human body.

Author Contributions

Conceptualization, P.D.S.-L. and J.A.T.-M.; methodology, P.D.S.-L.; software, P.D.S.-L.; validation, J.A.T.-M.; formal analysis, J.A.T.-M.; investigation, F.B.O.-J. and A.M.-R.; resources, P.D.S.-L.; data curation, P.D.S.-L.; writing—origi-

nal draft preparation, P.D.S.-L.; writing—review and editing, J.A.T.-M.; supervision, J.A.T.-M.; project administration, J.A.T.-M. All authors have read and agreed to the published version of the manuscript.

Funding

This work received no external funding.

Institutional Review Board Statement

Not applicable.

Informed Consent Statement

Not applicable.

Data Availability Statement

Not applicable.

Conflicts of Interest

The authors declare no conflict of interest.

References

- [1] Bräuner, E.V., Lim, Y.H., Koch, T., et al., 2021. Endocrine disrupting chemicals and risk of testicular cancer: A systematic review and meta-analysis. *The Journal of Clinical Endocrinology & Metabolism*. 106(12), e4834–e4860. DOI: <https://doi.org/10.1210/clinem/dgab523>
- [2] Myers, S.S., Smith, M.R., Guth, S., et al., 2017. Climate change and global food systems: potential impacts on food security and undernutrition. *Annual Review of Public Health*. 38(1), 259–277. DOI: <https://doi.org/10.1146/annurev-publhealth-031816-044356>
- [3] Eddleston, M., 2020. Poisoning by pesticides. *Medicine*. 48(3), 214–217. DOI: <https://doi.org/10.1016/j.mpmed.2019.12.019>
- [4] Shaheen, N., Shahzaib, M., Khan, U.M., et al., 2024. Genetically modified organisms for crop biofortification. In: Azhar, M.T., Ahmad, M.Q., Rana, I.A. (eds.). *Biofortification of Grain and Vegetable Crops*. Academic Press: Cambridge, MA, USA. pp. 19–37. DOI: <https://doi.org/10.1016/B978-0-323-91735-3.00002-9>
- [5] Naveen, A.K., Sontakke, M., 2024. A review on regulatory aspects, challenges and public perception in acceptance of genetically modified foods. *Food Sci-*

- ence and Biotechnology. 33(4), 791–804. DOI: <https://doi.org/10.1007/s10068-023-01481-0>
- [6] Topper, V.Y., Reilly, M.P., Wagner, L.M., et al., 2019. Social and neuromolecular phenotypes are programmed by prenatal exposures to endocrine-disrupting chemicals. *Molecular and Cellular Endocrinology*. 479, 133–146. DOI: <https://doi.org/10.1016/j.mce.2018.09.010>
- [7] Sakali, A.K., Papagianni, M., Bargiota, A., et al., 2023. Environmental factors affecting pregnancy outcomes. *Endocrine*. 80(3), 459–469. DOI: <https://doi.org/10.1007/s12020-023-03307-9>
- [8] Jâms, I.B., Windsor, F.M., Poudevigne-Durance, T., et al., 2020. Estimating the size distribution of plastics ingested by animals. *Nature Communications*. 11(1), 1594. DOI: <https://doi.org/10.1038/s41467-020-15406-6>
- [9] Talsness, C.E., Andrade, A.J., Kuriyama, S.N., et al., 2009. Components of plastic: experimental studies in animals and relevance for human health. *Philosophical Transactions of the Royal Society B: Biological Sciences*. 364(1526), 2079–2096. DOI: <https://doi.org/10.1098/rstb.2008.0281>
- [10] Dagher, J.B., Hahn-Townsend, C.K., Kaimal, A., et al., 2021. Independent and combined effects of Bisphenol A and Diethylhexyl Phthalate on gestational outcomes and offspring development in Sprague-Dawley rats. *Chemosphere*. 263, 128307. DOI: <https://doi.org/10.1016/j.chemosphere.2020.128307>
- [11] Caba-Flores, M.D., Martínez-Valenzuela, C., Cárdenas-Tueme, M., et al., 2023. Micro problems with macro consequences: accumulation of persistent organic pollutants and microplastics in human breast milk and in human milk substitutes. *Environmental Science and Pollution Research*. 30(42), 95139–95154. DOI: <https://doi.org/10.1007/s11356-023-29182-5>
- [12] Gore, A.C., Chappell, V.A., Fenton, S.E., et al., 2015. EDC - 2: The endocrine society's second scientific statement on endocrine-disrupting chemicals. *Endocrine Reviews*. 36(6), E1–E150. DOI: <https://doi.org/10.1210/er.2015-1010>
- [13] Skinner, M.K., 2008. What is an epigenetic transgenerational phenotype?: F3 or F2. *Reproductive Toxicology*. 25(1), 2–6. DOI: <https://doi.org/10.1016/j.reprotox.2007.09.001>
- [14] Vega Rodríguez, A., Rodríguez-Oramas, C., Sanjuán Velázquez, E., et al., 2022. Myths and realities about genetically modified food: A risk-benefit analysis. *Applied Sciences*. 12(6), 2861. DOI: <https://doi.org/10.3390/app12062861>
- [15] Amanullah, M., Nahid, M., Hosen, S.Z., 2024. Global overview of genetically modified foods and its benefits: A review. *Health Dynamics*. 1(7), 236–243. DOI: <https://doi.org/10.33846/hd10703>
- [16] Kuiper, H.A., Kleter, G.A., Noteborn, H.P., et al., 2001. Assessment of the food safety issues related to genetically modified foods. *The Plant Journal*. 27(6), 503–528. DOI: <https://doi.org/10.1046/j.1365-313X.2001.01119.x>
- [17] Bawa, A.S., Anilakumar, K.R., 2013. Genetically modified foods: safety, risks and public concerns—a review. *Journal of Food Science and Technology*. 50(6), 1035–1046. DOI: <https://doi.org/10.1007/s13197-012-0899-1>
- [18] Zhang, C., Wohlueter, R., Zhang, H., 2016. Genetically modified foods: A critical review of their promise and problems. *Food Science and Human Wellness*. 5(3), 116–123. DOI: <https://doi.org/10.1016/j.fshw.2016.04.002>
- [19] Uusi-Heikkilä, S., Perälä, T., Kuparinen, A., 2018. Species' ecological functionality alters the outcome of fish stocking success predicted by a food-web model. *Royal Society Open Science*. 5(8), 180465. DOI: <https://doi.org/10.1098/rsos.180465>
- [20] Chesnokova, V., Zonis, S., Barrett, R., et al., 2019. Excess growth hormone suppresses DNA damage repair in epithelial cells. *JCI insight*. 4(3), e125762. DOI: <https://doi.org/10.1172/jci.insight.125762>
- [21] Caserta, D., Maranghi, L., Mantovani, A., et al., 2008. Impact of endocrine disruptor chemicals in gynaecology. *Human Reproduction Update*. 14(1), 59–72. DOI: <https://doi.org/10.1093/humupd/dmm025>
- [22] Peretz, J., Vrooman, L., Rieke, W.A., et al., 2014. Bisphenol A and reproductive health: update of experimental and human evidence, 2007–2013. *Environmental Health Perspectives*. 122(8), 775–786. DOI: <https://doi.org/10.1289/ehp.1307728>
- [23] Yuan, M., Hu, M., Lou, Y., et al., 2018. Environmentally relevant levels of bisphenol A affect uterine decidualization and embryo implantation through the estrogen receptor/serum and glucocorticoid-regulated kinase 1/epithelial sodium ion channel α - subunit pathway in a mouse model. *Fertility and Sterility*. 109(4), 735–744. DOI: <https://doi.org/10.1016/j.fertnstert.2017.12.003>
- [24] Tian, M., He, X., Feng, Y., et al., 2021. Pollution by antibiotics and antimicrobial resistance in livestock and poultry manure in china, and countermeasures. *Antibiotics*. 10(5), 539. DOI: <https://doi.org/10.3390/antibiotics10050539>
- [25] He, L., Ying, G., Liu, Y., et al., 2016. Discharge of swine wastes risks water quality and food safety: Antibiotics and antibiotic resistance genes from swine sources to the receiving environments. *Environment International*. 92–93, 210–219. DOI: <https://doi.org/10.1016/j.envint.2016.03.023>
- [26] Pallecchi, L., Bartoloni, A., Paradisi, F., et al., 2008. Antibiotic resistance in the absence of antimicrobial use: mechanisms and implications. *Expert Review of Anti-Infective Therapy*. 6(5), 725–732. DOI: <https://doi.org/10.1586/14787210.6.5.725>

- [27] Carvalho, F.P., 2017. Pesticides, environment, and food safety. *Food and Energy Security*. 6(2), 48–60. DOI: <https://doi.org/10.1002/fes3.108>
- [28] Handford, C.E., Elliott, C.T., Campbell, K., 2015. A review of the global pesticide legislation and the scale of challenge in reaching the global harmonization of food safety standards. *Integrated Environmental Assessment and Management*. 11(4), 525–536. DOI: <https://doi.org/10.1002/ieam.1635>
- [29] Bretveld, R.W., Thomas, C.M., Scheepers, P.T., et al., 2006. Pesticide exposure: the hormonal function of the female reproductive system disrupted?. *Reproductive Biology and Endocrinology*. 4, 1–14. DOI: <https://doi.org/10.1186/1477-7827-4-30>
- [30] Cremonese, C., Piccoli, C., Pasqualotto, F., et al., 2017. Occupational exposure to pesticides, reproductive hormone levels and sperm quality in young Brazilian men. *Reproductive Toxicology*. 67, 174–185. DOI: <https://doi.org/10.1016/j.reprotox.2017.01.001>
- [31] Colopi, A., Guida, E., Cacciotti, S., et al., 2024. Dietary exposure to pesticide and veterinary drug residues and their effects on human fertility and embryo development: A global overview. *International Journal of Molecular Sciences*. 25(16), 9116. DOI: <https://doi.org/10.3390/ijms25169116>
- [32] Venkidasamy, B., Subramanian, U., Samynathan, R., et al., 2021. Organopesticides and fertility: where does the link lead to? *Environmental Science and Pollution Research*. 28, 6289–6301. DOI: <https://doi.org/10.1007/s11356-020-12155-3>
- [33] Leng, B., 2023. Impact of pesticides on food quality and human health. *Highlights in Science, Engineering and Technology*. 74, 1285–1289. DOI: <https://doi.org/10.54097/7wc57g15>
- [34] Dahiri, B., Martín-Reina, J., Carbonero-Aguilar, P., et al., 2021. Impact of pesticide exposure among rural and urban female population. An overview. *International Journal of Environmental Research and Public Health*. 18(18), 9907. DOI: <https://doi.org/10.3390/ijerph18189907>
- [35] Bokobza, E., Hinault, C., Tiroille, V., et al., 2021. The adipose tissue at the crosstalk between EDCs and cancer development. *Frontiers in Endocrinology*. 12, 691658. DOI: <https://doi.org/10.3389/fendo.2021.691658>
- [36] Crain, D.A., Janssen, S.J., Edwards, T.M., et al., 2008. Female reproductive disorders: the roles of endocrine-disrupting compounds and developmental timing. *Fertility and Sterility*. 90(4), 911–940. DOI: <https://doi.org/10.1016/j.fertnstert.2008.08.067>
- [37] Cohn, B.A., La Merrill, M., Krigbaum, N.Y., et al., 2015. DDT exposure in utero and breast cancer. *The Journal of Clinical Endocrinology & Metabolism*. 100(8), 2865–2872. DOI: <https://doi.org/10.1210/jc.2015-1841>
- [38] United States Environmental Protection Agency (EPA), 2008. Emerging contaminants - polybrominated diphenyl ethers (PBDE) and polybrominated biphenyls (PBB). Available from: <https://nepis.epa.gov/Exe/tiff2png.cgi/P1000L3S.PNG?-r+75+-g+7+D%3A%5CZYFILES%5CINDEX%20DATA%5C06T%5CHRU10%5CTIFF%5C00000138%5CP1000L3S.TIF> (cited 7 June 2024).
- [39] Zoeller, R.T., Bergman, Å., Becher, G., et al., 2014. A path forward in the debate over health impacts of endocrine disrupting chemicals. *Environmental Health*. 13, 1–11. DOI: <https://doi.org/10.1186/1476-069X-13-118>
- [40] Cabanillas, M.E., McFadden, D.G., Durante, C., 2016. Thyroid cancer. *The Lancet*. 388(10061), 2783–2795. DOI: [https://doi.org/10.1016/S0140-6736\(16\)30172-6](https://doi.org/10.1016/S0140-6736(16)30172-6)
- [41] de Oliveira, P.E., Gomes, A.C., de Oliveira, A.C., et al., 2018. Pesticides and thyroid function: A systematic review. *Chemosphere*. 213, 1023–1034. DOI: <https://doi.org/10.1016/j.chemosphere.2018.11.084>
- [42] Ahsan, H., Chen, C., Vahakangas, K.H., et al., 2008. Association between prenatal DDT exposure and breast cancer risk among young women in Bangladesh. *Journal of the National Cancer Institute*. 100(7), 502–508. DOI: <https://doi.org/10.1093/jnci/djn101>