

## Harmful Effects of Agricultural Chemical Pesticides Olivia Xu

Chemical pesticides have been used by farmers to prevent insects from destroying their crops since the 1930s. Pesticides are split into three main categories: insecticides, herbicides, and fungicides [1]. Chlorinated hydrocarbon insecticides like DDT were widely applied on crops until the 1960s. Insecticides like DDT fell from favor in the 1970s once the Environmental Protection Agency (EPA) was established. Currently, farmers utilize microbial agents, synthetic organic, or synthetic inorganic chemicals to control pests; approximately one billion pounds of pesticides are applied on crops annually in America [2]. Pesticides can affect the human genome by deleting or altering certain genes, with farmers facing the highest risk of genetic mutations due to their prolonged exposure [3].

Pesticides are already known to affect human health in small ways. For example, they can cause irritation and disrupt hormones in the body [4]. Some pesticides have been observed to cause epigenetic changes in rodents [5]. In an experiment comparing the chromosomal alterations between two groups–farmers who were exposed to pesticides and individuals who were not–researchers from Colombia discovered that there were more monosomies and deletions in the population that had been exposed to pesticides. The scientists also observed that the exposed group had a chromosomal abnormality range of 19-41%, while the unexposed group had a chromosomal abnormality range of chromosome abnormalities in the study sample.

Within pesticides, the presence of chemical compound malathion can lead to DNA methylation, affecting how genes are expressed. In another study, when researchers from Mexico compared a group of bone marrow mononuclear cells (BMMCs) exposed to malathion and a group unexposed, the researchers revealed that the exposed group showed more gene deletions and alterations. Notably, out of the 3,121 genes with an altered expression, 1,747 were overexpressed and 1,374 had low expression [6]. Scientists from the United States confirmed that when testing the effects of malathion, gene mutations were observed in animals and humans. As such, the chromosome damage caused by malathion implies a risk for toxicity to human genes [7].

When researchers compared human gene samples before and after exposure to pesticides, they discovered that half of the samples showed similar alterations, with some of the samples exhibiting hypermethylation and others exhibiting hypomethylation. The methylation value directly correlates to the expression of a gene, causing proteins to produce inconsistent amounts of their specialized products. An irregular methylation value can lead to serious diseases like cancer and Parkinson's disease [8]. From another study in North Carolina,



researchers noticed that farmers who worked with pesticides had an increase of methylation compared to other workers [9]. The hypermethylation shown in the studies suggest that pesticide exposure can alter activity in the human genome.

Pesticides can also lead to negative effects for fetuses. When fetuses are exposed to pesticides in utero, there is a slightly higher chance for the babies to develop tumors after birth. Having been exposed to pesticides during pregnancy or at a very young age can increase the possibilities for children to acquire genetic mutations possibly leading to cancer. Researchers have found that even the slightest amount of exposure to flea or tick pesticides could lead to brain tumors forming in children [10].

Moreover, when studying animal and human genes, researchers in Italy noticed that certain pesticides, like ones that are considered endocrine disruptors, modified epigenetic markers [4]. Another Italian study noted that xenobiotics, such as pesticides, led to alterations in the epigenome. The disruptions in the genome could also lead to lack of stability in human cells [11]. Epigenetic changes, like histone modifications and changes in gene expression, are crucial for cell specialization, but too many epigenetic changes could lead to diseases like cancer [12]. Thus, it is important to examine the evidence in an objective manner and understand that while pesticides are likely to increase cancer risk when people are exposed to them for long periods of time, some exposure likely is not overly harmful.

Although pesticides may help with preventing pests from consuming crops or destroying farmland, it also negatively affects the health of farmers because their occupation requires them to be in constant, close contact with the chemicals in pesticides. They can lead to DNA methylation, alter expression, and increase risk of genetic mutation. In order to decrease the risk of cancer and reduce changes to the genome for this population, it may be worth developing functioning pesticides that cause less overall harm to human health.

## References

1. National Research Council (US) Committee on Pesticides in the Diets of Infants and Children. (1993). Pesticides in the Diets of Infants and Children. Washington (DC): National Academies Press (US). 1, Background and Approach to the Study. https://www.ncbi.nlm.nih.gov/books/NBK236265/

2. Ohio-Kentucky-Indiana Water Science Center. (2017, March 23). Pesticides. U.S. Geological Survey,

https://www.usgs.gov/centers/ohio-kentucky-indiana-water-science-center/science/pesticides

3. Cepeda, S., Forero-Castro, M., Cárdenas-Nieto, D., Martínez-Agüero, M., & Rondón-Lagos, M. (2020). Chromosomal Instability in Farmers Exposed to Pesticides: High Prevalence of

Clonal and Non-Clonal Chromosomal Alterations. *Risk management and healthcare policy*, *13*, 97-110. https://doi.org/10.2147/RMHP.S230953

4. Collotta, M., Bertazzi, P. A., & Bollati, V. (2013). Epigenetics and pesticides. *Toxicology*, 307, 35–41. https://doi.org/10.1016/j.tox.2013.01.017

5. Karwal, P., Mittal, P., Nager, G., Singh, A., Singh, I. K. (2022). Chapter 13 - Effects of pesticides on human physiology, genetics, and evolution. *Emerging Contaminants in the Environment*, 287-310. https://doi.org/10.1016/B978-0-323-85160-2.00005-6

6. Navarrete-Meneses, M. d. P., Salas-Labadía, C., Juárez-Velázquez, M. d. R., Moreno-Lorenzana, D., Gómez-Chávez, F., Olaya-Vargas, A., Pérez-Vera P. (2023). Exposure to Insecticides Modifies Gene Expression and DNA Methylation in Hematopoietic Tissues In Vitro. *International Journal of Molecular Sciences*, *24*(7), 6259. https://doi.org/10.3390/ijms24076259

7. Flessel, P., Quintana, P. J., & Hooper, K. (1993). Genetic toxicity of malathion: a review. *Environmental and molecular mutagenesis*, 22(1), 7-17. https://doi.org/10.1002/em.2850220104

8. Giambò, F., Leone, G. M., Gattuso, G., Rizzo, R., Cosentino, A., Cinà, D., Teodoro, M., Costa, C., Tsatsakis, A., Fenga, C., & Falzone, L. (2021). Genetic and Epigenetic Alterations Induced by Pesticide Exposure: Integrated Analysis of Gene Expression, microRNA Expression, and DNA Methylation Datasets. *International journal of environmental research and public health*, *18*(16), 8697. https://doi.org/10.3390/ijerph18168697

9. Howard, T. D., Hsu, F. C., Chen, H., Quandt, S. A., Talton, J. W., Summers, P., & Arcury, T. A. (2016). Changes in DNA methylation over the growing season differ between North Carolina farmworkers and non-farmworkers. *International archives of occupational and environmental health*, *89*(7), 1103–1110. https://doi.org/10.1007/s00420-016-1148-0

10. Nicolella, H. D., de Assis, S. (2022). Epigenetic Inheritance: Intergenerational Effects of Pesticides and Other Endocrine Disruptors on Cancer Development. *International Journal of Molecular Sciences*, *23*(9), 4671. https://doi.org/10.3390/ijms23094671

11. Ficociello, B., Sturchio, E., Minoia, C., Casorri, L., Imbriani, P., & Signorini, S. (2010). Epigenetica ed esposizione ambientale a xenobiotici [Epigenetics and environmental exposure to xenobiotics]. *Giornale italiano di medicina del lavoro ed ergonomia*, *32*(1), 13-22.



12. Reamon-Buettner, S. M., Mutschler, V., & Borlak, J. (2008). The next innovation cycle in toxicogenomics: environmental epigenetics. *Mutation research*, *659*(1-2), 158-165. https://doi.org/10.1016/j.mrrev.2008.01.003