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MAIZE MUTATIONS INDUCED BY FUNGAL DISEASES

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Maize is one of the most widespread cereal crops in the world, possessing high economic value and being used as a food, feed, and industrial crop [1]. However, the yield and grain quality largely depend on the phytosanitary condition of the crops. The most harmful diseases of maize are fungal infections caused by representatives of the genera *Fusarium*, *Aspergillus*, *Ustilago*, and *Helminthosporium* [2]. Phytopathogenic fungi produce a wide range of toxic substances – mycotoxins – which affect metabolic processes in cells and are capable of inducing mutational changes [3]. Studying mutational changes in the maize genome under the influence of fungal pathogens is important both for understanding plant adaptation processes to stress factors and for developing new approaches to breeding disease-resistant varieties.

The aim of the study is to investigate the nature and mechanisms of mutational changes in the maize genome under the influence of fungal pathogens and their metabolites.

The main objectives of the research are:

1. To analyze the main types of fungal pathogens of maize and their toxigenic properties.
2. To determine the biochemical mechanisms of DNA damage induced by mycotoxins.
3. To examine the morphological and molecular characteristics of mutations in infected plants.
4. To assess the potential use of mutations in breeding disease-resistant maize forms.

The study was conducted on samples of maize varieties of domestic breeding infected by fungi of the genera *Fusarium, Aspergillus,* and *Ustilago*. The identification of pathogen species composition was carried out based on morphological and cultural characteristics according to methodological recommendations. To analyze mutations, PCR diagnostics, agarose gel electrophoresis, and biochemical indicators of oxidative stress were used. It was established that maize infection by *Fusarium graminearum* and *F. verticillioides* is accompanied by intensive accumulation of fumonisins, trichothecenes, and zearalenone – secondary metabolites with pronounced mutagenic and cytotoxic effects. These compounds cause disruption of cell membrane integrity, alteration of plasma membrane permeability, and destabilization of the cell nucleus. As a result, the formation of reactive oxygen species (ROS) increases, inducing oxidative stress, DNA and protein damage, and disturbances in the replication of genetic material.

Molecular-genetic analysis of infected maize samples revealed an increased frequency of point mutations in genes encoding enzymes involved in pigment biosynthesis (including anthocyanins and carotenoids), as well as in genes responsible for the synthesis of structural proteins of the cell wall. This leads to alterations in kernel coloration, the formation of atypical phenotypes, and reduced mechanical stability of cell walls, which in turn increases the plants' susceptibility to secondary infections.

Under the influence of mycotoxins, activation of transposons – mobile genetic elements capable of changing their position within the genome – was also observed, causing insertions, deletions, or DNA rearrangements. This phenomenon is particularly pronounced in regions containing repetitive sequences. The movement of transposons can lead to disruptions in gene expression regulation, the formation of non-functional proteins, or, conversely, the emergence of new adaptive traits. Thus, fungal toxins act not only as damaging agents but also as inducers of genetic variability, providing a certain evolutionary potential for the species.

The obtained results are consistent with the findings of other researchers, which confirm the ability of fungal pathogens to act as natural mutagens. According to field and laboratory studies, fumonisins and trichothecenes are capable of inducing chromosomal aberrations, disrupting mitotic division, and altering DNA methylation, which may affect genome stability and its epigenetic regulation.

The effect of *Fusarium* toxins is dual in nature: on the one hand, they inhibit plant growth and development through the destruction of cellular structures; on the other hand, they stimulate the formation of new genetic combinations that may confer resistance to subsequent stress factors. This process can be regarded as an induced mutagenesis of natural origin, which, although accompanied by a temporary decrease in productivity, contributes in the long term to the enhancement of the adaptive potential of populations.

In a number of cases, infected maize plants develop adaptive mutations that increase their resistance to reinfection. These changes may result either from the selection of cells with modified alleles of resistance-related genes (such as ZmPR1, ZmLOX10, and ZmNPR1), or from the stabilization of epigenetic rearrangements associated with cytosine methylation and chromatin remodeling. Such processes are likely to underlie long-term induced tolerance, in which subsequent generations of plants exhibit enhanced resistance to fungal pathogens even in the absence of direct contact with the infection.

Thus, fungal diseases of maize can be regarded not only as a destructive factor but also as an evolutionarily significant mechanism that contributes to the formation of genetic diversity, the natural selection of more resistant forms, and the expansion of the species' adaptive potential. Understanding these processes opens up prospects for developing new maize varieties through the use of naturally induced mutations in breeding programs.

References

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