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Editor
Komarytskyy M.L.
Ph.D. in Economics, Associate Professor

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THE ROLE OF GENETIC MECHANISMS IN THE FORMATION AND RECOGNITION OF EMOTIONS

Mumdzhian Arsen Karenovych
Student
Yanitska Lesia Vasylivna
Ph.D, Associate Professor
Osinska Larysa Feliksivna
Ph.D
Bogomolets National Medical University, Kyiv, Ukraine

Introductions. Hereditary diseases are caused by chromosomal and gene mutations. They arise under the influence of exogenous and endogenous factors and can be lethal, sublethal and neutral. The main reason is changes in the genetic apparatus. The manifestation of these signs can be varied: from the failure to assimilate a certain component of a food product to the ability to form emotional states. Despite numerous studies and literature data on pathogenetic mechanisms, the role of mutant genes in the mechanism of Urbach-Withe syndrome development is not sufficiently defined, which does not fully reflect the range of pathogenetic mechanisms and ways to correct them.

It is known that the genotype of individuals is prone to certain pathological changes at the stage of intrauterine development. As a result of contact with the environment, nucleotide polymorphism occurs in specific genes. Their subsequent expression leads to the development of the disease, which causes a high risk of disability and mortality.

Aim. To analyze the literature data on the role of mutant genes in the mechanisms of disease development. To identify the main genetic mechanisms of the development of this pathology. To highlight the mechanisms of occurrence, course and influence of gene mutations that cause changes in the cognitive abilities of patients on the example of Urbach-Withe syndrome.

Materials and methods. The literature data were studied and analyzed to deepen the pathogenetic mechanisms and determine the role of mutant genes.

Results and discussion. Urbach-White syndrome is a rare disease (since the discovery of the disease by scientists at the University of Iowa in 1929, less than 300 cases have been known) characterized by the deposition of lipoids and proteins, collagen fibers in the form of a hyaline-like substance in the skin, mucous membranes, and other soft tissues. Most often, the upper parts of the digestive tract, brain structures and the limbic system are affected.

The risk of development is an increase in certain gene variants. The cause of the pathology is a mutation of a gene localized in the short arm of the first chromosome (1q21) that encodes an extracellular matrix glycoprotein, extracellular matrix protein 1 (ECM1). This glycoprotein is the main structural component of basement membranes and extracellular matrix. All the functions of this protein have not yet been elucidated, but some of them are known:

- Participation in endochondral osteogenesis and angiogenesis.

- Interaction with other structural proteins, which ensures the integrity of soft tissues and maintains homeostasis.

Mutation of the gene encoding ECM1 leads to a weakening of the basement membrane and infiltration of the cell with various substances. Patients develop monoblepharosis, hyperkeratosis, and increased skin scarring. The digestive system is characterized by the following symptoms: ankyloglossia, dental hypoplasia, dry mouth and frequent infections of the major salivary glands. The most important manifestations of the disease are the consequences of infiltration (calcification) of the basal structures of the nervous system - the amygdala (MT). Typical symptoms of amygdala damage include memory loss, epilepsy, depression, lack of fear, and inability to recognize emotions in the faces of others. It was found that the amygdala, among other functions, serves as a "fear center".

It has been shown that the main component in the emergence and development of fear is the protein statmin, the largest amount of which is found in the amygdala. Calcification of the MT leads to a lack of statmin protein, which leads to a weakening of synaptic connections between neurons, which is the cause of the disappearance of fear, and with it the ability to detect potential danger. The authors proved the

interdependence of MT degeneration with the loss of the ability to distinguish fear in human faces and to produce the emotion of fear.

There is a case of Urbach-Withe disease, the so-called patient named SM-046, the "woman without fear". During the controlled experiments, SM-046 showed no signs of fear, anxiety, or worry. Fear is a basic emotion that acts as a warning in the face of possible danger.

Conclusions

1. Gene mutations affect cognitive abilities and socially significant behavioral reactions.
2. Mutation of the gene encoding ECM1 leads to a weakening of the basement membrane and cell infiltration.
3. The amygdala functions as a "fear center".
4. Fear is a basic emotion that plays the role of a warning before a possible danger.

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