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FINDING GENETIC FACTORS ASSOCIATED WITH COGNITIVE ABILITIES

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The article provides an overview of the results of modern genetic studies of human cognitive abilities. Finding genetic factors, associated with cognitive abilities, will have far-reaching ramifications at all levels of understanding from DNA to brain and to behavior. Despite its complexity, cognitive ability is a reasonable candidate for molecular genetic research because it is one of the most heritable features of behavior. The first attempts to find genetic factors, associated with cognitive abilities, focused on genes, involved in brain development and function, but this direction proved to be unproductive, as it turned out that there are about 18.000 genes, and it was too difficult to detect among them those genes that are involved in cognitive processes. In addition, a considerable number of genetic factors of human traits are single-nucleotide polymorphisms (SNPs) which are in non-coding DNA regions rather than in traditional genes. The effect of each separate SNP is unimportant, and a clear expression of the general cognitive ability is noticeable only if all the associated SNPs are involved. Currently, over 11,000 such SNPs have been identified, which are uneven in different functional regions of the genome: over 60 % in gene introns, almost 30 % in intergenic DNA regions, about 5 % in gene exons, and about 5 % in transcribed regions (downstream, upstream) and frame regions (UTR'5, UTR'3) of genes. Also there are found 74 SNPs, associated with school achievements. These SNPs are disproportionately located in genes that regulate transcription and alternative splicing of other genes, which are expressed in nerve tissues of the brain during its prenatal development. Finding genetic factors that explain the inheritance of cognitive abilities is important for both science and society. Information about these factors can be used in other fields of human science – human genetics and medicine. It will open up new scientific horizons for education too owing to understanding of the genetic aspects of learning and memory

Keywords: *genetic factors, cognitive abilities, single-nucleotide polymorphisms, candidate genes*

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1. Introduction

The main postulate of genetics is that all the features of a living organism are determined by its genes, and are formed under the influence of environmental factors. Numerous studies have shown that only about half of the total cognitive ability is determined by genes.

Cognitive abilities are a set of mental abilities, which includes intelligence, problem solving, planning, abstract thinking, understanding of complex ideas, rapid learning and experiential learning [1]. They are studied by psychogenetics, which in Western scientific sources is mainly called behavioral genetics [2].

The search for genetic factors, related to cognitive abilities, will have far-reaching consequences at all levels of understanding of this phenomenon – DNA, brain and behavior. Despite the complexity of cognitive abilities, their molecular genetic research is a justified direction, because these abilities are among the most hereditary features of human behavior.

Understanding genetic mechanisms is relevant for the modernization of Ukrainian education and bringing it closer to international standards.

2. Literary review

There are general and specific cognitive abilities. The term "general cognitive ability" better reflects the essence of the phenomenon than the word "intelligence", because the latter has a large number of different interpretations in psychology and in general use [3]. The level of general cognitive ability is measured by the indicator *g*, which was proposed more than a century ago by the British psychologist and author of factor analysis Charles Spearman [4]. Dozens of specific cognitive abilities, the level of which is determined by special tests, are components of general cognitive ability. They are grouped into four factor groups: verbal abilities (including vocabulary and fluency), spatial abilities (visual representation of objects and their rotation in two- and three-dimensional space), speed of perception (simple calculations and comparisons of numbers) and visual memory (recognition of pictures after a short and long period of time). A separate group of specific cognitive abilities consists of learning abilities – reading, writing, mathematics, science and more. According to numerous psychogenetic studies, most traits of human behavior are subject to moderate genetic influence and little influence of the common

environment. In addition, cognitive abilities are characterized by three genetic features: their heredity increases with age; they are influenced by the assortment of the spouses; cognitive and learning abilities are determined by common genes [5].

3. Aim and tasks of the study

The aim of the article is to theoretically analyze the results of genetic research on human cognitive abilities as a guarantee of the development of science, society, as well as successful education and upbringing.

To achieve this goal, the following tasks were set:

1. To substantiate on the basis of theoretical analysis of the results of genetic research the expediency of searching for genetic factors that explain the inheritance of human cognitive abilities, which is important for the development of science and society.

2. To show the scientific horizons of genetic research in education.

3. To deepen the value understanding of research in genetics on genetic factors of cognitive abilities.

4. Materials and methods

Methods of structural-logical analysis and the bibliosemantic research method were used during the work. The materials of the study were international experience in studying the genetic factors of cognitive abilities. The bibliosemantic method was used to determine the state of study of the problems of genetic research of human cognitive abilities, modern information systems in Ukraine and the world, literary sources, electronic resources. The method of structural-logical analysis allowed to identify, to logically structure and to establish links between general cognitive abilities, determined by genes, as well as environmental factors.

5. Results

The first attempts to find genetic factors, related to cognitive abilities, focused on genes, involved in the development and functioning of the brain [5]. But this direction turned out to be unproductive, because it was deprived of clear hypotheses about which genes can be the real candidate genes of the actual cognitive abilities. The fact is that the human brain expresses 86 % of its identified genome, which is almost 18 thousand genes [6], among which it is too difficult to identify those genes that are involved in cognitive abilities. Moreover, the problem is complicated by the fact that a significant number of genetic factors of human traits are found in non-coding DNA fragments, rather than in traditional genes. As a result, despite significant samples of research (up to 10 thousand people), it has not yet been possible to obtain unambiguous and reliable data on the associations of genes, associated with cognitive abilities [7, 8].

Some scientists are guided by a different strategy of searching for candidate genes of cognitive abilities, which is to study the so-called endophenotypes, ie individuals with intermediate manifestations of the trait. It is believed, that endophenotypes are genetically simpler, which may allow for smaller samples to obtain very specific and reliable results [9–12].

As a result of the above problems, attempts to find genes, associated with complex traits, such as cognition, have gone beyond the search for candidate genes. Researchers are not looking for genes themselves, but for certain cognitive DNA markers in the form of single nucleotide polymorphisms (SNPs), which are scattered throughout the genome and can be found not only in coding but mainly in non-coding fragments and in the intergenic intervals of the genome. This is due to the scanning of the entire human genome by the technology of genome-wide associations study (GWAS) [3]. GWAS makes it possible to conduct simultaneous search and analysis of hundreds of thousands of SNPs. The first GWAS in terms of general cognitive ability (*g*) did not give unambiguous results [13–16].

Even a fairly large sample of children's intelligence of almost 18,000 children did not show a significant impact of individual SNPs on overall cognition, although its heredity, determined based on the impact of the total array of known SNPs, reached almost 50 % [17]. Thus, to identify the effect of individual SNPs and determine the real rate of inheritance of general cognitive ability requires much larger samples, which was confirmed by G. Davis and colleagues' meta-analysis of the results of GWAS in almost 54 thousand adults, which revealed 13 reliable general genomic SNPs, involved in general cognitive ability [18]. The most pronounced were 4 SNPs that were associated with the following genes: rs10457441 with MIR2113, rs17522122 with AKAP6, rs10119 with TOMM40 and rs13625 with HMGN1. All these genes are expressed in brain tissues during its development.

The MIR2113 gene is located on chromosome 6 (6q16.1) and produces short non-coding RNAs that are involved in the regulation of gene expression, affecting both stability and mRNA translation. SNP rs10457441 is located in a single exon of the gene. Earlier, this gene was found to be involved in academic performance and bipolar disorder.

The AKAP6 gene is located on chromosome 14 (14q12) and encodes a protein, involved in alternative splicing. SNP rs17522122 was detected in the final transcriptional region of the 3'downstream gene. The gene may be involved in up to 8 diseases, including schizophrenia, tachycardia, glioma.

The TOMM40 gene is located on chromosome 19 (19q13.32) and encodes a protein that is part of the outer membrane of mitochondria and provides them with protein. SNP rs10119 is located in the terminal framework region of the 3'UTR gene. The gene may be involved in nearly 30 diseases, including dementia, Alzheimer's disease, depression, Charcot-Marie-Tooth neuropathy and others.

The HMGN1 gene is located on chromosome 21 (21q22.2) and encodes a protein that facilitates the transcription of genes, ensuring their structural preparation for this process. The localization of SNP rs13625 in the gene is not specified. The gene may be involved in up to 16 human diseases, including Down syndrome, autism spectrum disorder, several types of anemia and leukemia, allergies and more.

Gene localization is given by [19], the location of SNPs by [20], gene involvement in diseases by [21].

The results of this study confirmed that the effect of each individual SNP is negligible and that a clear

manifestation of the general cognitive ability is noticeable only if the whole set of SNPs involved.

Recently, the same group of authors completed an even larger meta-analysis of genetic mechanisms of general cognitive ability, a sample of which exceeded 300 thousand people aged 16 to 102 years [22]. At the same time, more than 11,000 SNPs were found to be significantly involved in general cognitive ability, and almost 22,000 were probably involved. These SNPs are localized in different functional structures of the genome unevenly. Most of them are in the introns of genes – more than 60 %, and almost 30 % in the intergenic regions of DNA. Oly about 5 %. SNP - in the coding fragments of genes The rest of them are located in the transcribing regions (downstream, upstream) and framework regions (UTR'5, UTR'3) of genes. The average density of SNPs in DNA is almost the same throughout the genome [23], so their number in a single gene is directly proportional to its size (see the description of the GATAD2B, SLC39A1 and AUTS2 genes below). Among all these SNPs, 434 independent valid SNPs were identified, distributed within 148 loci on all autosomal chromosomes, of which 58 loci were shown for the first time. These SNPs were also involved in many other human characteristics - physical (eg, body mass index, height, weight), medical (eg, lung cancer, Crohn's disease, blood pressure) and psychiatric (eg, bipolar disorder, schizophrenia, autism, Alzheimer's disease).

Based on this meta-analysis, 709 genes were identified that are significantly associated with general cognition. Of these, 418 genes have already appeared in previous studies of general cognitive ability and academic performance [19, 24–26], and 291 genes were discovered for the first time. All these genes are grouped into seven functional sets, associated with processes that provide general cognitive ability: neurogenesis, regulation of nervous system development, neuronal process formation, neuronal differentiation, and cell development regulation. These genes are expressed in all structures of the brain. In addition, the level of their expression in the cortex and cerebellum was mostly related to general cognition.

Here is a brief description of three of these genes, which were also found in previous studies – GATAD2B, SLC39A1 and AUTS2 [25, 26]. The localization of genes is given by [19], and their involvement in diseases by [21].

The GATAD2B gene (size ~ 134 Kb) is located on chromosome 1 (1q21.3) and contains 396 SNPs. The protein, encoded by this gene, is involved in suppressing gene expression. The gene is involved in 17 diseases, including autism spectrum disorder, intellectual disability, mental retardation, lung cancer and more.

The SLC39A1 gene (size ~ 9 Kb) is located on chromosome 1 (1q21.3) and contains 64 SNPs. It encodes a protein that is part of cell membranes and is involved in zinc transport. The gene is involved in 7 diseases, including Alzheimer's disease, mental retardation, prostate and breast cancer.

The AUTS2 gene (size ~ 1195 Kb) is located on chromosome 7 (7q11.22) and contains 4061 SNPs. Its molecular functions in humans are still unknown. The gene is involved in more than 30 diseases, including mental retardation, autism spectrum disorder, attention deficit hyperactivity disorder, schizophrenia, reading disorders, alcoholism, leukemia and others.

A. Okbay and colleagues [27] conducted a meta-analysis of the GWAS results of a sample of 329 thousand adults, which revealed 74 SNPs, related to school achievements. These SNPs are disproportionately located in genes that regulate the transcription and alternative splicing of other genes that are expressed in the nervous tissues of the brain during its prenatal development.

Of the several hundred loci of the genome, only 15 candidate genes of school achievements were selected. For example, we give a brief description of the 7 most significant of them. Gene localization is given by [19], the location of SNPs by [20], gene involvement in diseases by [21].

The TBR1 gene is located on chromosome 2 (2q24.2), contains SNP rs4500960, the location of which is not indicated in the source. It is also involved in 27 human diseases, including intellectual disability, autism spectrum disorder, microcephaly and more.

The MEF2C gene is located on chromosome 5 (5q14.3), contains SNP rs7277187, the location of which is not specified in the source. It is also involved in more than 100 human diseases, including intellectual disability, autism spectrum disorder, speech disorder, attention deficit hyperactivity disorder, microcephaly, Alzheimer's disease and others.

The ZSWIM6 gene is located on chromosome 5 (5q12.1), contains SNP rs61160187, the location of which is not specified in the source. It is also involved in 18 human diseases, among which brain disorders predominate.

The BCL11A gene is located on chromosome 2 (2p16.1), contains SNP rs2457660 in the intron. It is also involved in more than 80 human diseases, including autism spectrum disorder, intellectual disability, dyslexia, microcephaly, leukemia and more.

The CELSR3 gene is located on chromosome 3 (3p21.31), contains SNP rs11712056, the location of which is not indicated in the source. It is also involved in more than 90 human diseases, including schizophrenia, epilepsy, microcephaly and more.

The MAPT gene is located on chromosome 17 (17q21.31) and contains SNP rs192818565 in the intron. It is also involved in about 200 diseases, including intellectual disabilities, inability to learn, schizophrenia, autism, depression and more.

The SBNO1 gene is located on chromosome 12 (12q24.31) and contains SNP rs7306755 in the intron. It is also involved in schizophrenia and prostate cancer.

In this study, the polygenic rate, ie the cumulative effect of all genetic factors involved, significantly reached 4 % of the variance of the trait. A similar polygenic index in previous, smaller analyzes also correlated with school achievements [28] and cognitive abilities [29, 30].

GWAS of specific cognitive abilities – to reading [31, 32], mathematics [33], memory [34, 35] and information analysis [16, 31, 34] were also carried out. They reaffirmed the idea of the polygenic nature of any cognitive ability. As in other similar cases, there is also a need for fairly large samples of research, which can only be achieved through meta-analysis.

There is also a search for SNPs, involved in the size of certain brain structures that are related to cognitive abilities. The results of a meta-analysis of almost 20,000 people in 17 studies, which found one SNP, associated with hippocampal volume and another SNP with intracranial volume [36], are known. Although these SNPs account for only 0.3 % of the variance of the trait, they also correlate significantly with general cognitive ability.

The hippocampus is a key structure of the brain that is involved in the mechanisms of emotions, memory and learning [37–39]. In addition, intracranial volume is significantly correlated with overall cognitive ability [40–42]. The volume of the hippocampus, brain and intracranial space are significantly inherited in all primates. The coefficient of heredity of these traits in humans averages 70 %, 80 % and 80 %, respectively [36].

Intergenic SNP rs7294919, located on chromosome 12 (12q24,22), is associated with hippocampal volume and TESC gene expression levels in brain tissues. The product of this gene is involved in the regulation of cell pH. The TESC gene is involved in 11 diseases, including depression, mood disorders, neuroticism, and psychotic disorders.

SNP rs10784502, located on the same chromosome within the HMGA2 gene (12q14.3), is associated with intracranial volume. The HMGA2 gene encodes a non-histone protein that is part of chromosomes and is involved in gene transcription. It is involved in more than 200 diseases, among which oncopathology predominates.

There was also a tendency to correlate the SNP rs10494373, located on chromosome 1 within the DDR2 gene (1q23.3), with the total brain volume. The protein, encoded by this gene, is involved in cell proliferation and adhesion, as well as in the reconstruction of intercellular substance. The DDR2 gene is involved in up to 70 diseases, among which oncopathology predominates.

Gene functions are presented according to [19], gene involvement in diseases according to [43].

All the findings of genetic factors of cognitive abilities relate only to general, common SNP. As for rare SNPs, so far they have been found to be associated only with monogenic mental disorders that are accompanied by impaired cognitive abilities [44]. It is assumed, that rare SNPs do not have a significant impact on cognitive abilities [44–48].

6. Conclusions

Thus, the search for genetic factors that explain the inheritance of cognitive abilities is important for both science and society, because:

1) information about these factors can be used in other fields of human science - in anthropogenetics and medicine;

2) new scientific horizons in education are opened due to the understanding of the genetic aspects of learning and memorization;

3) the value understanding that it is necessary to prepare all children for the requirements of real society, which, the further away, the more technological, rather than invest all educational resources in raising the most talented children, which can be determined genetically, is deepened.

It is important to be as careful as possible, accurately consider the consequences of activities and strictly adhere to ethical standards.

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