Bioresonance diagnostics and therapy using the complete genome a person is the key to the diseases of civilization and the rejuvenation of the body V.A. Ivanchenko (Medical center "Biopharm-test", Moscow, Russia)

In bioresonance diagnostics and therapy, separate genes, DNA and RNA are used. So, in the selector of the AIC "IMEDIS-EXPERT" there are pointers to DNA abnormalities, genetic flaws, mutations, the DNA group "Medpharma". They are widely used in practice and are well appreciated by doctors, but their use cannot fully demonstrate the mechanisms of gene participation in metabolism and the influence of environmental factors (epigenetics). Only an analysis of the complete human genome can reveal this complex problem. Suffice it to say that US President Barack Obama back in 2006 introduced a law to the US Senate on genomic and personalized medicine (S-3822), which provides for multibillion-dollar public investment

research aimed at using in clinical practice, the information contained in the complete human genome. It is believed that this will take into account not only the genetic characteristics of each patient, but also individualize the influence of environmental factors and selection

therapeutic measures. Moreover, the National Institute for Research on the Complete Human Genome has been operating in the United States for more than 10 years. In Russia, this area is developing in the nanotechnology group and is considered particularly promising.

## Materials and research methods

Taking into account the urgency of the problem, we decided to use the complete human genome containing over 23,000 genes (obtained from Stanford University, USA), with the aim of using it in bioresonance diagnostics and therapy on the equipment of the IMEDIS center - APK IMEDIS-EXPERT. The human genome and frequency-resonance samples of individual genes were studied on a specially modified sequencer using a computer program developed by us to search for individual genes that individually participate in the development of the disease in each patient.

The study involved 104 patients, divided into 7 groups. The first group (18 people) - patients with breast cancer III-IV stage. The second group (14 people) - patients with type 1 diabetes mellitus taking insulin. The third group (16 people) - patients with rheumatoid arthritis. The fourth group (20 people) - patients with gastric ulcer and duodenal ulcer who have undergone repeated ineffective treatment. The fifth group - HIV-infected patients (8 people), in whom, despite standard therapy, the level of the virus did not decrease. The sixth group - old people and long-livers 85–90 years old and more (11 people). The seventh group - practically healthy people who served as control (20 people). All subjects underwent diagnostics using the ART method "IMEDIS-TEST" (biological age, adaptation reserves of the body, photon indices were determined),

disturbances, biological age with the use of the Inner Skan apparatus of the company "Tanita" (Japan), the hardware-software complex "Omega" (Russia) and other methods described by us earlier [1].

The work was divided into 6 stages:

1. Study of the functional state of patients before treatment.

2. Finding individual genes using electropunctural diagnostics in the complete human genome, responsible for the pathology in patients.

3. Identification of this gene from databases of genomes of different countries.

4. Elucidation of the biochemical mechanism of action of a given gene by genetically biochemical databases.

5. Using pathological genes to target them

homeopathic remedies, resonance frequencies, information taken from the reflexogenic zones of the patient, the use of potentiated healthy genes, etc.

6. Studying the indicators of ART "IMEDIS-TEST" and other indicators after exchange rate BRT.

## Research results and discussion

It was found that the genomes of different people have significant differences in their frequency-resonance characteristics, differing in the degree of gene expression, the presence of active or repressed alleles in them, etc. In this regard, the pathology of one gene in each group was recognized only when it was involved in the pathogenesis of the disease if it was consistently repeated in all patients of each group. Indeed, a pathological gene can have several normal alleles, which in combination are capable of compensating for biochemical problems without bringing them to clinical signs. Our proposed potentiated genome and test for epigenetic disorders can serve as good indicators of genetic problems, including changes in genes due to environmental factors. These pointers are much more sensitive, and most importantly, more specific, than the generally accepted indicators of genetic flaws - Argentum C12 nitricum and DNA indices. Since clinically diagnosed health problems begin with gene dysfunction (the deepest level of the physical body), it is obvious that genomic bioresonance diagnostics should be the earliest objective method for detecting pathological changes in the body. Accordingly, in patients of all groups and even in practically healthy individuals of the control group, abnormalities in the genome were revealed. At the same time, the use of ART with Medpharma drugs did not directly reveal violations. At the same time, testing them using the genome showed violations of varying degrees. It is especially interesting that by tracing the geno-biochemical pathways of metabolism, the ART method can clearly show the entire causal chain of pathology development in the body. Hence, the very low effectiveness of homeopathic remedies used in low potencies D3, D6, D12 became clear and the instructions of unicist homeopaths to use the highest dilutions, acting most deeply. Indeed, in our studies, low dilutions only affected biochemical changes in the body, but did not affect the genome. Only with the D30 potency began to appear the reaction of genes to therapy, the most pronounced when testing

LM potencies. Now it becomes clear that the genome is not a permanent, stable formation. It changes dynamically under the influence of external and internal environmental factors (nutrition, physical, psycho-emotional and other stress). Clinically, this does not manifest itself in healthy people due to the presence of genetic reserves. We have proposed pointers to the buffer properties of the genome and to genetic reserves of various degrees. The latter are maximal at the age of 18–22 years, and subsequently gradually decrease. Indirectly, this was indicated by the CFFS indices, which were very low and significantly increased, on average by 5.4 Hz compared to the initial level, after carrying out genomic BRT. This coincides with the data [2] on the possibility of using CFMC for predicting the effectiveness of therapy. The most interesting, in our opinion, are BRT results with the targeting of individual resonant genes, homeopathic remedies, minerals at the genomic disorders of patients. It is necessary to distinguish between the concepts of "Genome pathology" and "Genetic diseases". In the first case, there may be congenital and acquired changes in individual alleles of genes, compensated by healthy alleles. Such disorders may not manifest as disease. On the other hand, environmental factors, interfering with the processes of transcription, translation, phenotypic gene expression, can cause modifications of these healthy alleles and the proteins encoded by them (enzymes, hormones, receptors, structural proteins). Even the order in which individual genes are located in the genome can change. All this affects the structural anatomy, biochemistry, physiology and mental functions of a person. This is how the genetic diversity of the body's reactions to environmental factors is determined, including the homeopathic constitution (sycosis, psora, syphilis). Currently, there has been a paradigm shift in genetics. Previously, it was believed that each gene encodes a specific protein that determines a particular function of the body. It has now been established that each gene is responsible for the synthesis of many proteins, and the functions of the body are determined by their most complex interaction. Genome changes can include gene amplification, rearrangement and loss of mobile genetic elements without causing any clinical symptoms. They develop in genetic diseases with a corresponding pathology of the genome. It should be borne in mind that the "silent" pathology of the genome under unfavorable conditions can manifest itself in the form of diseases. This is especially true for the so-called polyetiological diseases with a hereditary predisposition: diabetes, malignant tumors, atherosclerosis, pathological aging, etc. Hence it is clear why we found changes in the genome in all patients. At the same time, they were very diverse and often did not correspond to the typical genome changes described by geneticists. Indeed, in patients of the first group with breast cancer one could expect changes in the genes of tumor suppressors BRCA1 and BRCA2 [3]. However, they were registered very rarely, only in one patient out of 18. Consequently, the cancer was not of a familial nature. Almost 30% of patients had changes in the gene for caspase 8 (Casp-8), which protects against cancer development [4]. In three patients, the contribution of transforming growth factor (TGFb) was more significant, gene for estrogen receptor (ESR1), gene for fibroblast growth factor (EGFR2) [5]. However, BRT targeting one of these genes did not lead to tumor breakdown. Only

the use of a complex of genes for targeting had a noticeable effect. Indeed, in the first group, after the course BRT, the tumor size decreased on average by 42.3% (P <0.05), the general state of health, sleep and appetite improved.

The second group of patients with type 1 diabetes mellitus had disorders of more than 10 genes that contribute to the development of this disease. In the first place is the main histocompatibility complex, that is, the complex of genes encoding the immune response (combination D3 / Dr4 or D4 / Ar9). Such combinations deprive this complex of the ability to bind to insulin, which leads to the impossibility of presenting it to the developing immune system in the first 6 months of life and to create immunological tolerance. Therefore, the body subsequently reacts to β-cells already as a foreign body, which leads to their damage and a decrease in insulin production. Hence - the second gene that determines the development of diabetes - the insulin gene. In our studies, 88.2% (!) Of patients had a defective insulin gene, which contributed to the development of diabetes. The third and fourth genes (PTPN22 and CTLA4) in total give the risk of developing diabetes up to 50% and are involved in the formation of killer T-lymphocytes that damage β-cells [6]. The individual contribution of all these genes to the pathogenesis of diabetes in patients of group 2 was different. The greatest contribution (over 51%) was made by the genes of the main histocompatibility complex. In second place (31%) is the insulin gene, followed by the PTPN22 and CTLA4 genes. Therefore, the targeting was made at all these genes with different potentiation coefficients. If the duration of the illness was less than 3 years, then we very often managed to remove the patient from insulin (8 people). The dose was reduced in 6 patients. These were patients with long-term diabetes, when the βcells, apparently, atrophied. Probably, the use of the stem cell genome can radically help here.

In the third group of patients with polyarthritis, we paid attention to the HLA DRB1SE gene. Its violation contributes to the development of autoimmune diseases due to the fact that unusual proteins are formed, similar to those of joint proteins. This is why the immune system attacks the joints. However, BRT targeting this gene did not produce results. In this regard, we paid attention to other parts of the genome. In particular, it was found that the CCR2 gene, which is modified during the immune response, is much more often detected, which coincides with the data [7]. The DNase II gene is also involved in the pathogenesis of polyarthritis [8]. In addition, genes encoding

anti-inflammatory cytokines (IL-1, IL-4, IL-10, Il-13), a gene for an inhibitor of metalloproteinase that destroys the joint. This coincides with the data of N. Bessis, M. Boissier [9].

In the fourth sick group (ulcerative disease stomach and duodenal intestines), very unexpected results were obtained. IN In particular, the fact of influence on the genome of resonant-frequency drugs was established: "antimicrobes", "antiviruses", "antifungals" in different potencies. Such drugs are very often used by BRT specialists for the treatment of pathogenic microorganisms. We have shown that this is accompanied by negative changes in the human genome with repression of a number of genes. So, for example, the appointment of patients of the fourth group of Helicobacter nosodes in individual potencies, although it inhibits the development of the ulcerative process, but in parallel repress the genes encoding intestinal enzymes. ByApparently, it is not by chance that Helicobacteria colonize the intestinal tract of more than half of the population. Probably, they perform certain physiological functions in the body, for example, the cleavage of urea, since they have urease activity, etc., therefore, Helicobacteria, like E. coli, apparently, can be attributed to opportunistic bacteria. They can only under certain conditions, excessive reproduction and extreme weakening of the body, harm it. Hence, it is clear that "killing microbes" by nosodes or resonant frequency therapy not only leads to the development of resistant forms, but also disrupts the human genome, which is in symbiosis with microorganisms. It is much more effective to strengthen

natural protection of the gastroduodenal mucosa [10]; therefore, we used BRT with genes encoding its protection. In particular, the calcitonin gene-related peptide, the annexin 1 gene and survivin in potency even allowed to achieve epithelialization of the ulcer in an average of 12 days, which significantly exceeds the standard of scarring (21 days).

In the 5th group of patients with HIV infection, a new thing is that it has now been established [11] that some people, in principle, cannot be infected with the AIDS virus. This is due to the fact that they lack the gene encoding the CCR5 coreceptor ( $\Delta$ 32), which causes innate immunity against AIDS. The fact is that the virus enters the cell through the activation of the CCR5 receptor ( $\Delta$ 32), which, as it were, opens the "door" for it. We found that all patients of group 5 have this gene, so we decided to repress it in order to prevent reinfection. Another gene that determines the level of viruses in

blood - HCP5. It encodes an enzyme that converts RNA into the in DNA, chromosome of the host cell. The presence of a particular verse of the this gene is a kind of "genetic condom" giving for the lucky ones the impossibility of contracting AIDS. Apparently, the activity of the gene determines the incorporation of the virus into the human genome, its replication, viremia, and the clinical development of the disease. According to our data, activation of this gene in all patients reduced the degree of viremia by 20–45%. Of course, long-term observation is necessary in order to completely resolve the issue of the diseappearance of the virus.

In the 6th group of patients, the genes responsible for the induction or acceleration of aging were studied. It was found that all patients had different violations of the Foxo gene, which is responsible for providing a response to stress. BRT with the Foxo gene optimizes its expression, eliminates stress, reduces the biological age of patients, and increases the reserves of adaptation. However, this does not provide anti-aging, but only pushes it back. Earlier [1], we used resonance preparations of genes encoding sirtuins. This made it possible to reduce the biological age of various organs and systems. Since the genome contains different alleles of the SIRT gene family, we used their potentiated copies to fight aging. It was found that different alleles of these genes reduce biological age in different ways, which determines the individual rate of aging.

Thus, diagnostics using the complete human genome made it possible to take a fresh look at the etiology and pathogenesis of civilization diseases, and genomic BRT showed significant efficiency, in some cases by 80–100

%, exceeding all other methods of multiresonance therapy. New data have been obtained on the role of certain genes in the development of aging and the possibility of antiaging. Bioresonance genomic diagnostics and therapy can be recommended for use, reflecting the thesis repeatedly put forward by Professor Yu.V. Gotovsky at the lectures: "The best drug for a person is a human drug. "

## Bibliography

1. Ivanchenko V.A. Bioresonance approachto the use of markers //biological age in the fight against agingAbstracts and reports. XVInternationalconference"Theoreticalapplication of bioresonance andmultiresonance therapy ". Part II. - M .:IMEDIS, 2009. - pp. 66–74.Image: Conference

2. Bizyaev P.D., Bobrov I.A. Determining the critical merge frequency flashes as an effective way to predict the long-term effects of therapeutic effects // Homeopathic Yearbook. Materials of the XXI Moscow International Homeopathic Conference "Development

homeopathic method in modern medicine ". - M .: Tekhpoligraftsentr, 2010. - pp. 103–116.

3. Fackenthal JD, Olopade OI Breast cancer risk associated with BRCA1 and BRCA2 in diverse populations, Nature Reviews Cancer, 2007, 7, 937-948.

4. Cox A. et al., A common coding variant in CASP8 is associated with breast cancer risk // Nature Genetics, 2007, 39, 352-358.

5. Walsh T., King MC Ten genes for inherited breast cancer // Cancer Cell, 2007, 11, 103-105.

6. Jahromi MM, Eisenbarth GS Genetic determinants of type 1 diabetes across populations // Annals of the New York Academy of Science, 2006, 1079, 289-299.

7. Fujii Hiroshi et al., Ablation of the CCR2 gene exacerbates polyartritis in inter genes A receptor antagonict-Deficient mice // Artritis and Rheumatism, 2011, vol 63, issue 1, 96–106.

8. Kawane K. et al., Chronic polyartritis caused by mammalian DNA that escape from degradation in macrophages // Nature, 2006, 443, 998–1002.

9. Bessis N., Boissier M., Gene therapy for patients with rheumathoid arthritis // Joint Bone Spine, 2006, 73, 2, 169-176.

10. Zhu A., Kaunith J.Gastroduodenalmucosaldefense//Curr.Gastroenterol.Rep 2008, 10, 6, 548–554.

11. Sabeti PC et al., The case for selection at CCR5- Delta32 // PLoS Biology, 2005, 3, 378.