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OPTIMISING THE IMPACT OF ALMOND SHELL POWDER ON THE PROPERTIES OF EFFECTIVE CERAMIC MATERIALS

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Abstract: This article explores the effects of almond shell powder (ASP) as a modifier that induces porosity in ceramic brick materials. In this study, ASP was added to a mixture of clay and brick waste in four different ratios: 10% (N1), 15% (N2), 20% (N3), and 30% (N4). The ceramic brick samples were fired at 850°C, 950°C, and 1050°C for 2 hours, after which their physical and mechanical properties were analysed experimentally. The results indicated that as the ASP content increased, porosity also increased, leading to a decrease in density values. The compressive strength of the samples varied, with a maximum of 25 MPa and a minimum of 5.2 MPa. Additionally, the thermal conductivity ranged from 0.46 to 0.17 W/(m·K). According to the research findings, the samples N1 and N2, which contained 10% and 15% ASP and were fired at 850°C, met the requirements of relevant standards (ASTM C 62-13 (2013) and TS EN 771-1).

Keywords: cyanobacteria, Spirulina, Cladophora, antioxidants, anticancer properties.

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Introduction:

The increasing demand for ceramic bricks is driven by their lightweight nature and superior thermal insulation properties compared to porous concrete blocks. Since both a porous structure and high mechanical strength are required, research on the development of innovative ceramic materials has focused on utilizing renewable waste as a modifier. In general, the modifiers used in the ceramic brick industry are classified into two main groups: powders derived from natural minerals and powders obtained from renewable sources, such as agricultural plant residues and industrial waste (Georgiev, 2018; Viruthagiri, 2013)

In this study, almond shell powder was used as a porosity-inducing modifier. As is well known, Azerbaijan, being one of the largest almond producers in the region, generates tons of almond shells as waste each year through its almond processing factories. It should be noted that almond shells constitute approximately 35–75% of the almond fruit, meaning that from an annual production of ~1.5 thousand tons of almonds, around 0.5–1.1 thousand tons of shells are discarded as waste.

In the brick industry, brick fragments separated as waste are traditionally used as a common diluent modifier (additive). In this study, the purpose of using brick fragments was to achieve the required mechanical strength in the samples (Eroğlu, 2017; Demir, 2003).

Therefore, one of the alternative approaches to waste valorization is the incorporation of renewable waste materials into the production of construction materials.

Materials and Methods:

Samples were prepared from clay and brick fragments, ground to a fine powder, and mixed

with almond shell powder at ratios of 10% (N1), 15% (N2), 20% (N3), and 30% (N4). The mixture was blended in a planetary mill and shaped into cylindrical moulds of 24x50 mm size, then dried at 50°C for 48 hours and fired at 850°C, 950°C, and 1050°C for 2 hours.

Porosity and water absorption were determined by measuring the mass of the samples after drying, boiling in water, and saturating with water. Bulk density was calculated based on these measurements.

Compressive strength was evaluated by applying hydraulic pressure and measuring the maximum force. Thermal conductivity was tested using a "TA Instruments FOX 314 Thermal Conductivity Analyzer."

Results and discussions:

1. Raw Materials Used in the Research

Clay - In this study, the raw clay material was sourced from the Binə clay deposit, located in the territory of the settlement of the same name. The deposit consists of Upper Pliocene-aged clay rocks, exhibiting an open brown and blue colouration. The clay layer, with a thickness ranging from 19.1 to 19.7 meters, is covered by a soil-vegetation layer of 0.1–0.6 meters. Based on its mineralogical composition, the clay belongs to the illite group. The chemical composition of the clay is presented in Table 1.

Oxides	SiO_2	Al_2O_3	Fe_2O_3	CaO	MgO	SO_3	Na_2O +K_2O
Mass, %	54.82-57.84	13.62-14.44	5.59-5.67	6.57-8.33	1.97-2.02	0.63-1.26	3.89-5.12

Table 1. Chemical composition of the clay (%).

The physical properties of the clay are as follows: water retention capacity of 18.38–20.28%, shrinkage after drying of 6.30–7.10%, water absorption of 15.75–17.31%, and bulk density ranging from 1.81 to 1.87 g/cm³. The compressive strength of the samples derived from the clay is 27.7–35.5 MPa, while the flexural strength is 14.3–18 MPa. The presence of CaO in the raw clay material plays a crucial role in enhancing the mechanical strength of the material (Fətəliyev, 2000; Martırena, 2006).

Brick Fragments - Brick fragments were used as a modifying agent to reduce the plasticity and shrinkage that may occur in ceramic materials (Eroğlu, 2017).

Almond Shell Powder (ASP) - Observations using an electron microscope have shown that almond shells contain large pores with diameters of 300–500 µm and small pores with diameters of 40–60 µm. The elemental composition of almond shells consists of C (72.27%), O (22.88%), N (3.87%), and Si (0.87%). Their chemical components include cellulose (38.48%), hemicellulose (28.82%), lignin (29.54%), alkali extract (14.03%), and benzyl alcohol extract (8.00%). The benzyl alcohol extracts from almond shells contain 17 types of organic compounds. Thermal stability analysis indicates that almond shells primarily reduce their volume at 260°C and 335°C. These characteristics suggest that almond shells are suitable for use in composite and porous materials (Li, 2018).

2. Experimental Section

2.1. Preparation of Samples

For the preparation of the ceramic mass, clay fragments and brick particles were first ground to a powder with a particle size of up to 1 mm. Almond shells were cleaned and dried at 50°C. The dried shells were then pulverized in an electric mill to a particle size of 400 µm. Almond shell powder was added to the mixture in proportions of 10% (N1), 15% (N2), 20% (N3), and 30% (N4). The mixture was subsequently homogenized in a ball mill under controlled moisture conditions.

Cylindrical samples with dimensions of 24 × 50 mm were prepared using moulds from the ceramic mass and dried at 50°C for 48 hours. The dried samples were then subjected to firing at three different temperatures (850°C, 950°C, and 1050°C) for 2 hours (Figure 1).

Fig. 1. Stages of sample preparation.

2.2. Conducted Tests

For each composition, two different samples were prepared, and the properties of the samples were studied using various testing methods. Determination of Porosity and Water Absorption - The mass of the samples dried at 105°C for 12 hours (W_1), the mass in water after boiling for 5 hours using the Archimedes method (W_2), and the mass of the water-saturated samples in the air (W_3) were determined. Porosity is calculated using the following formula:

$$\text{Porosity (\%)} = \frac{W_3 - W_1}{W_3 - W_2} \cdot 100 \quad (1)$$

$$\text{Water Absorption (\%)} = \frac{W_3 - W_1}{W_1} \cdot 100 \quad (2)$$

Determination of Bulk Density - The bulk density is determined using the results from the porosity test:

$$\text{Bulk Density} = \frac{W_1}{W_3 - W_2}, \quad (3)$$

Determination of Compressive Strength - After the samples were dried, they were subjected to compression under the load of a hydraulic press. The compressive strength is determined by the ratio of the maximum destructive load to the surface area of the sample:

$$\sigma = \frac{F_d}{S} \quad (4)$$

Determination of Thermal Conductivity - This test was conducted using the "TA Instruments FOX 314 Thermal Conductivity Analyzer (TCA)." The surface of the material is brought into contact with the sensor of the device, and the heat transfer from the material's surface to the interior is used to determine thermal conductivity.

3. Results of the Tests

The results of the tests conducted on the samples were compared with the standards set by ASTM C 62-13(2013) and TS-EN 771-1 [11] [12]. Significant results were obtained for the physical-mechanical properties depending on the mineralogical composition of the clay raw material, the amount of almond shell powder (ASP), and the firing temperature. No fine or large cracks were observed in the samples fired at 850°C, 950°C, and 1050°C. Normally, the shrinkage for fired clay bricks is considered to be 8%, while the results obtained in this study varied between 4% and 12%.

The density of the samples is influenced by several factors, particularly porosity and firing temperature. As shown in Figure 2, the density changes inversely with porosity. When the porosity was 20%, the density was 1.32 g/cm³, and when the porosity was 54%, the density decreased to 0.92 g/cm³. The highest porosity (54%) was observed in the samples fired at 1050°C with 30% ASP.

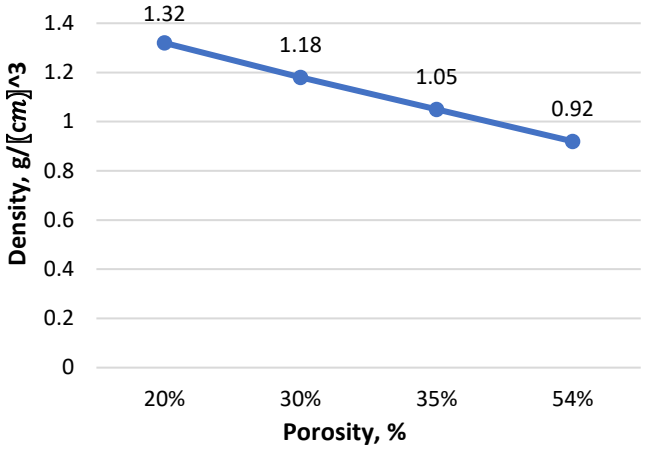

Fig. 2. Graph of the relationship between density and porosity.

One of the key factors determining the durability of clay bricks is water absorption. The water absorption values for almond shell powder (ASP) at 10%, 15%, 20%, and 30% content varied between 15.60% and 52.80% (Figure 3.). According to ASTM C62-13 (2013) standard, the water absorption should be 20.0%. Among the results obtained, only the N1 sample met the standard's requirement. As the amount of ASP increased, porosity also increased, leading to higher water absorption values.

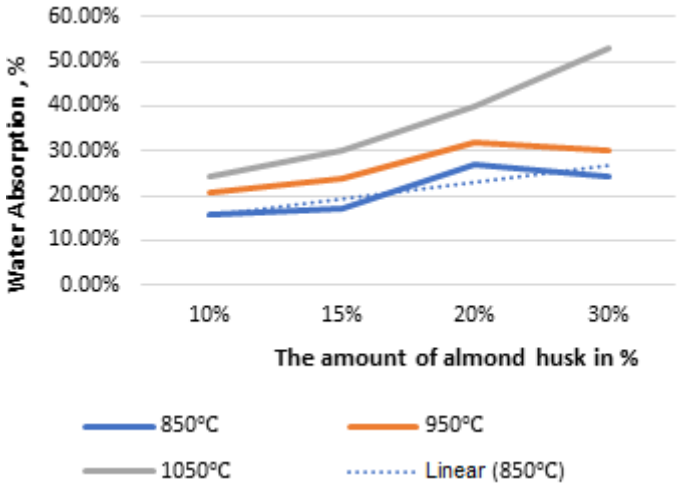

Fig. 3. Variation in water absorption of the samples depending on the firing temperature and the amount of almond shell powder (ASP), in percentage.

Another property that determines the quality of the material is the determination of the compressive strength limit (CSL) of the samples.

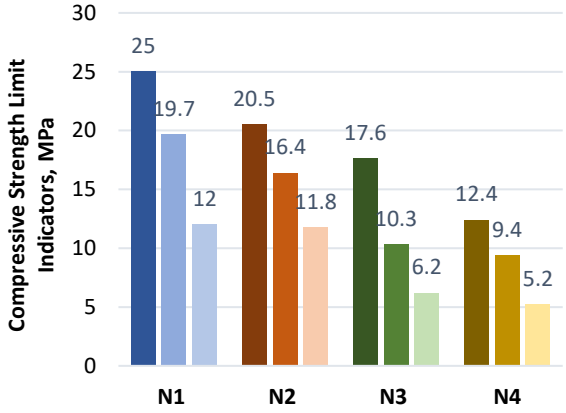

Fig. 4. Graph of the dependence of the compressive strength limits (CSL) of N1-N2-N3-N4 samples on the amount of almond shell powder (ASP).

As the amount of almond shell powder (ASP) increases, the porosity of the samples also increases, resulting in a decrease in the compressive strength limit. According to the TS-EN 771-1 standard, the compressive strength limit should be >7 MPa. The CSL values obtained from the N1 and N2 samples (25 MPa÷12 MPa; 20.5 MPa÷11.8 MPa) after firing at three different temperatures show high results, thus meeting the requirements of the standard.

Thermal conductivity is one of the most important criteria required for porous materials. According to the standard, the thermal conductivity of ceramic bricks should be below 0.60 W/(m·K). The thermal conductivity values obtained in the study ranged from 0.46 to 0.17 W/(m·K).

Conclusıon:

This research aimed to investigate the optimizing effect of almond shells, a renewable waste material, as a modifier on the properties of fired clay bricks. The behaviour of samples to physical and mechanical influences at varying raw material compositions was determined through experimental results, compared with international standards, and the results were found to be acceptable. Depending on the amount of almond shell powder (ASP), firing temperature, and porosity, the density of the samples ranged from 0.92 to 1.32 g/cm³. As porosity increased, the water absorption values also increased correspondingly. The compressive strength and thermal conductivity values for all samples were within acceptable limits. Based on the results, the physical and mechanical properties of the N1 and N2 samples met the requirements for porous ceramic materials. Therefore, almond shells can be considered a beneficial modifier both economically and practically.

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ANTISENSE OLIGONUCLEOTIDES FOR HUNTINGTON'S TREATMENT

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Abstract: Huntington's disease is one of the most widespread genetic neurodegenerative diseases inherited in an autosomal dominant manner. It causes neuron loss and degeneration of some parts of the brain, leading to motor, psychological and cognitive impairments. For today, there is no disease-modifying cure for Huntington's disease, and all possible treatment options focus on symptom management only. Molecular-based therapies like using antisense oligonucleotides (ASO) are promising in this field. They are meant to work on the mRNA level, and many medicines of this type are in clinical or pre-clinical trials. Advantages and disadvantages of ASOs are reviewed in this article.
Keywords: Huntington's disease, ASO, clinical trials, molecular therapy, neurodegeneration

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Introduction:

Huntington's disease (HD), caused by a CAG trinucleotide repeat expansion in the Huntingtin (HTT) gene, is classified as a neurodegenerative disorder. The mutation is located in exon 1 of the HTT gene (Ahmad et al., 2023), which resides on chromosome 4. The normal CAG repeat length ranges from 10 to 35. Repeats in the range of 35–39 are considered to represent a low penetrance range, whereas individuals with ≥40 repeats are highly likely to develop the disease phenotype (Medina et al., 2022). Juvenile-onset Huntington's disease typically results from expansions of ≥56 repeats. The repeat length also significantly influences the rate of disease penetrance and determines the risk of HD transmission to the next generation (Tabrizi et al., 2022) (see Table 1 and Figure 1).

HD prevalence varies with geographical location and ethnic background, with the highest rates reported among individuals of Northern European descent (Fisher & Semaka, 2021). The HTT gene is one of the most evolutionarily conserved genes, and knockout studies in mice have shown that embryonic loss of this gene is lethal. The wild-type HTT protein plays vital roles in intracellular vesicle trafficking, cell motility, transcriptional regulation, autophagy, cell division and survival, and ciliogenesis. It is also involved in the regulation of the cell cycle, DNA repair, and apoptosis through its interaction with p53 (Fields et al., 2021).

When CAG expansion occurs, the resulting mutant HTT (mHTT) leads to both loss-of-function and toxic gain-of-function. These changes include synaptic and mitochondrial dysfunction, oxidative stress, proteasome system disruption, impaired autophagy, and the formation of toxic protein aggregates. mHTT aggregates accumulate in neurons and cause dysregulation of the ubiquitylation system. Experimental studies in animal models have shown that reducing mHTT levels can reverse neuropathology, improve motor performance, and reduce behavioural deficits. These findings underscore the significance of mHTT lowering as a major therapeutic strategy for HD (Fields et al., 2021).

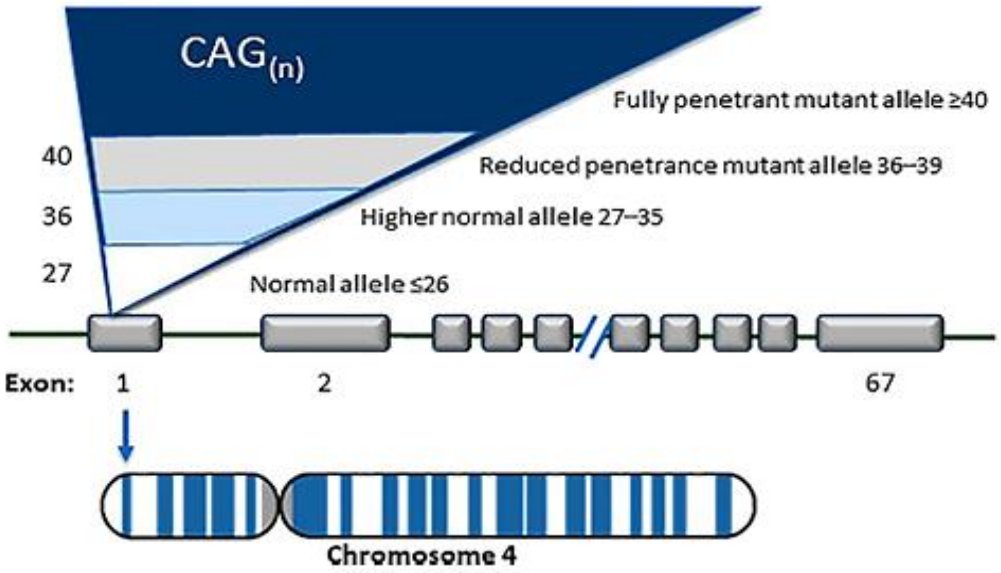

Fig. 1. CAG repeat number and allele forms.

Table 1. CAG repeat range in HD and risk assessment (Gatto et al., 2020).

Description of Gene	CAG Repeat Range	Risk of HD	Risk of HD in Next Generation
Normal	≤26	No HD	No
Higher normal	27–35	No HD	Possible
Reduced penetrance	36–39	Possible HD	Yes
Full penetrance	≥40	Definite HD	Yes

Antisense oligonucleotides (ASOs) are single-stranded synthetic DNA molecules designed to target and degrade mRNA or pre-mRNA sequences through the activation of RNase H1 (see Figure 3). By degrading mHTT transcripts, ASOs lower the levels of toxic protein expression (Ersöz & Demir-Dora, 2024). There are two types of ASOs: allele-specific and allele-nonspecific. Currently, Tominersen (allele-non-specific), WVE-120101, and WVE-120102 (allele-specific) are in different phases of clinical trials (Ferguson MW et al.). Allele-non-specific ASOs target both mHTT and wild-type HTT (wtHTT), while allele-specific ASOs are designed to selectively reduce mHTT levels (Tabrizi et al., 2022).

The effects of ASO-mediated HTT downregulation are transient, necessitating repeated administrations. Furthermore, the non-selective nature of Tominersen results in decreased levels of wtHTT, which is potentially undesirable in certain patient populations. WVE-120101 and WVE-120102 target two single-nucleotide polymorphisms (SNPs) found in approximately 40–50% of HD patients with the mutant HTT gene (Byun S, Lee M, Kim M., 2022). A novel allele-specific ASO, VO659, currently undergoing a phase 1/2a clinical trial, binds selectively to mRNA containing expanded CAG repeats. It does not activate RNase H1 but rather suppresses mHTT translation by reversibly interacting with the repeat expansion. VO659 is being evaluated not only for HD, but also for Spinocerebellar Ataxia types 1 and 3 (Cheng et al., 2024).

Materials and Methods:

NCBI, PubMed, and OMIM databases were used for collecting works on Huntington's disease, its pathogenesis and molecular basis, mHTT and wtHTT gene structures, protein structures and functions. Possible treatment options reviewed. Selection criteria for articles

included their recentness, only approved clinical trials on Antisense Oligonucleotides, clinical trials only on the adult population with HD, and clinical trials on phase 1/2a, 2 and 3. Data on clinical trials outcomes collected and checked in Clinicaltrials.gov and the World Health Organisation International Clinical Trials Registry Platform (ICTRP).

According to data collected upsides and downsides of ASO therapy for the HD patient population are compared.

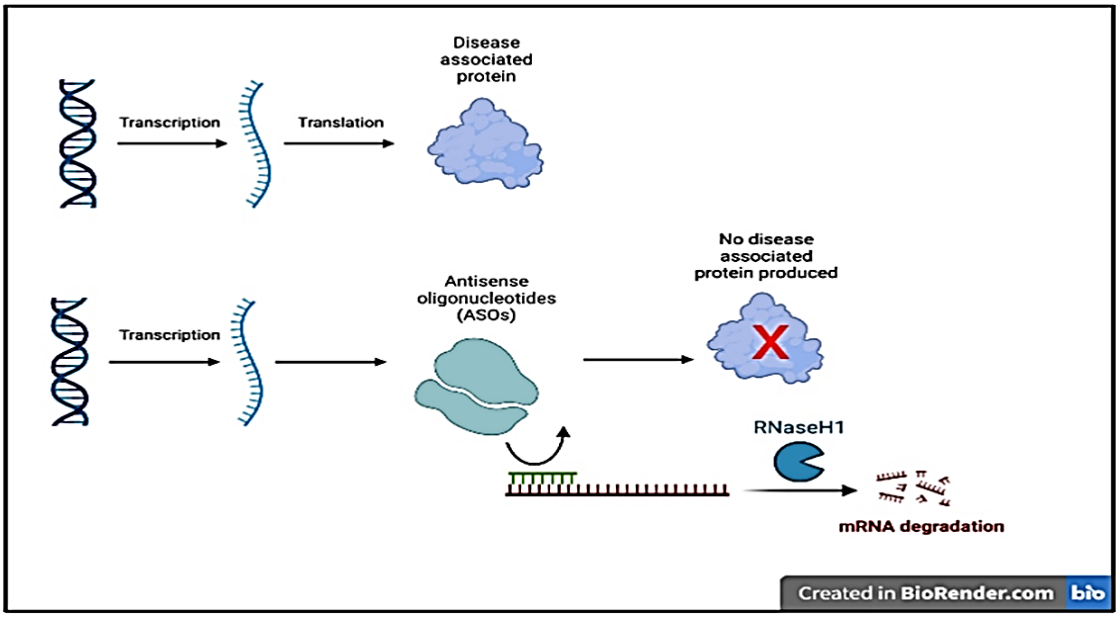

Fig. 2. Mechanism of action of ASOs. (Ersöz & Demir-Dora, 2024).

Results and discussions:

ASOs represent a promising disease-modifying treatment for HD patients, offering advantages over conventional drugs that primarily address symptoms. By directly targeting the underlying genetic mutation, ASOs enable selective mHTT reduction, with clinical benefits observed in motor and cognitive functions. In contrast to symptom-managing agents, ASO therapy can be administered less frequently - once, twice, or even more sparsely in some cases - depending on the pharmacokinetics of the agent.

The use of SNP data enables allele-specific ASOs to selectively target mHTT transcripts, minimising adverse effects related to wtHTT depletion. However, this approach has limitations. The presence of the two SNPs recognised by WVE-120101 and WVE-120102 is restricted to approximately 40–50% of the HD population, indicating that these ASOs may not be universally applicable. Expanding treatment access will require additional SNP discovery and development of new allele-specific ASOs tailored to genetically diverse populations.

Another major concern is the method of ASO delivery. Currently, ASOs are administered intrathecally (IT) or intracerebroventricularly (ICV), routes that facilitate wide drug distribution across the central nervous system. However, these delivery methods carry risks such as infection, inflammation, and tissue damage (Tong et al., 2024).

Targeting the CAG expansion itself also carries a risk of off-target effects, as other transcripts containing similar repeats could be inadvertently affected. Furthermore, exon 1 of the HTT gene may undergo incomplete splicing, leading to the production of mHTT that evades degradation by traditional ASOs. This highlights the need for complementary strategies to ensure full therapeutic coverage.

Nevertheless, the ASO VO659 represents a significant advancement in the field. Unlike RNase H1–H1-mediated degradation, VO659 binds expanded CAG repeats and inhibits mHTT translation without triggering transcript cleavage. This mechanism may reduce the risk of off-target degradation and offers a potential pathway for treating HD and other trinucleotide repeat disorders with similar pathology.

In conclusion, ASOs are emerging as a major class of therapeutics in the field of neurogenetics, specifically for HD. While challenges such as delivery, off-target effects, and genetic variability remain, ongoing research and clinical trials continue to refine and optimise their use. Future directions should focus on improving ASO specificity, expanding the range of applicable patient populations, and exploring novel mechanisms of action such as those utilised by VO659.

Conclusıon:

Huntington's disease is a severe neurodegenerative disorder with no cure. CAG repeat expansions in the HTT gene play a key role in disease progression. Antisense oligonucleotide (ASO) therapy targets mutant HTT transcripts to reduce toxic protein levels and shows promise as a disease-modifying treatment.

Clinical trials of allele-specific and non-specific ASOs report improvements in motor and cognitive symptoms. Challenges include risky delivery methods, the need for repeated dosing, limited patient applicability due to genetic differences, and possible off-target effects. New ASOs like VO659, which block mutant HTT translation without degrading RNA, may address some issues. Continued research is needed to optimise therapies and improve patient outcomes.

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MOLECULAR VARIATION AND BIOTECHNOLOGICAL APPLICATIONS OF ALGAE

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Abstract: Food-significant algae (red, green, blue-green, and brown algae) are rich in vitamins, minerals, antioxidants, and other bioactive compounds, and they play an important role in both nutrition and medicine. In recent years, studies on molecular variation have allowed for a deeper understanding of the biological properties and beneficial components of algae by examining their genetic diversity. Through molecular approaches, the genetic variation of different species and populations can be identified, enabling the selection of more suitable and effective algae species for food and medical applications.

Keywords: Food-significant algae, molecular variation, genetic diversity, bioactive compounds, genetic markers, bioinformatics, food and medical applications.

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Introduction:

Algae are organisms with significant biotechnological potential in the fields of food, medicine, energy, and ecology. They possess diverse species and characteristics, and their biological diversity is extensively studied. Molecular variation is a key approach for studying the genetic diversity of algae and utilising this diversity in biotechnological applications. These studies help improve our understanding of the biological properties of algae and allow for more efficient use of their beneficial traits.

Food-significant algae offer numerous beneficial components for the human body due to their rich composition. Depending on the species, algae are rich in various nutrients. They contain valuable substances such as proteins, vitamins, minerals, omega-3 fatty acids, antioxidants, amino acids, and dietary fibre. These compounds make algae an ideal

choice as a nutritional supplement and for therapeutic purposes.

Algae, particularly microalgae, have high protein content. Algal species such as Spirulina and Chlorella contain between 60% and 70% protein. The proteins found in algae include all the essential amino acids required by the human body, making them a high-quality source of protein. With these properties, algae are considered an indispensable food source in vegetarian and vegan diets.

Algae are also rich in many essential vitamins. In particular, they contain B-group vitamins, vitamin A (beta-carotene), vitamin C, and vitamin E. The vitamin B12 content of algae is especially important, as B12 is typically found only in animal products, yet it naturally occurs in some algal species. This is especially beneficial for individuals who follow vegan or vegetarian diets.

Algae are also rich in minerals. They offer essential minerals such as potassium, calcium, magnesium, phosphorus, iron, and iodine. These minerals are crucial for human

health, particularly for bone strength, immune system function, and the transport of oxygen in the blood. Algae, especially marine algae, contain high levels of iodine, which is essential for the proper functioning of the thyroid gland.

Algae are a valuable source of omega-3 fatty acids as well. These fatty acids, especially EPA (eicosapentaenoic acid) and DHA (docosahexaenoic acid), contribute to cardiovascular health, reduce inflammation, and support cognitive function. Marine algae are considered an irreplaceable plant-based source of omega-3 fatty acids (Guliyev & Ismayilov, 2017)

Algae are also abundant in antioxidants. They contain compounds such as carotenoids and polyphenols, which help neutralise free radicals and prevent cellular damage. These antioxidants may slow the ageing process and reduce the risk of chronic diseases such as cancer (Alizadeh & Rzayev, 2018).

Lastly, algae are rich in dietary fibre. The fibres found in algae support digestive health, improve gut function, and help prevent constipation. They also aid in the elimination of toxins from the body, thereby promoting overall wellness.

Due to their nutrient-rich composition, food-significant algae are considered highly valuable for use as dietary supplements, health products, and functional foods. Their richness in diverse nutrients offers numerous health benefits and makes them an indispensable component of modern diets.

Molecular variation refers to changes that occur in an organism's genetic material, including DNA, RNA, or proteins. Algae possess a high degree of genetic diversity due to their ability to thrive in various environmental conditions. Studies on molecular variation help identify these genetic differences and examine their biological effects. In order to explore the genetic diversity of algae, researchers utilize genetic markers, DNA barcoding, gene expression profiling, and other molecular biology techniques.

Such investigations enable a better understanding of the adaptation capabilities, biological activities, and ecological compatibility of different algal species within their respective ecosystems. Molecular variation studies are also critical for the conservation and sustainable development of algal genetic resources.

Algae have a wide range of biotechnological applications, particularly in food production, pharmaceuticals, cosmetics, bioenergy, and environmental protection. Understanding these applications on a molecular level is essential, as such research supports the selection of more beneficial and effective algal strains for industrial purposes.

Due to their richness in proteins, vitamins (especially B12), minerals, omega-3 fatty acids, and other nutrients, algae are extensively used in the food industry. Molecular variation studies allow for the identification of more nutritious and health-promoting algal species suitable for food production. For instance, algae such as Spirulina and Chlorella are widely used as dietary supplements due to their unique nutritional profiles, and exploring their genetic diversity can help enhance their productivity and quality. (Huseynova & Mammadova, 2020).

Algae are used in the field of medicine, particularly as a source of bioactive compounds with anticancer, antibacterial, anti-inflammatory, and antiviral properties. Molecular variation facilitates the more efficient production of these potential pharmaceutical components. For example, some algal species produce substances that may contribute to the development of novel treatments for cancer. Genetic variation studies provide essential data for the genetic improvement and optimisation of such species. (Huseynova & Mammadova, 2020).

Algae also play an important role in the cosmetics industry, as they offer beneficial minerals, vitamins, and antioxidants for skin health. Products derived from algae are used to protect and rejuvenate the skin. Molecular variation research enables the identification of more effective and skin-compatible algal strains for cosmetic applications (Nasirov & Veliyev, 2019). Algae can also be utilised in bioenergy production. It is possible to produce biodiesel and bioethanol from various algal species. Mo-

lecular variation aids in selecting species with enhanced energy production potential. Furthermore, algae absorb carbon dioxide from the atmosphere, helping to mitigate environmental pollution. Their genetic modification and adaptability allow for more efficient and scalable bioenergy processes.

Algae are also employed in environmental remediation and ecosystem restoration. They play a key role in water purification, prevention of soil degradation, and maintaining ecological balance. Molecular variation contributes to the identification of algae involved in these processes and supports the enhancement of their bioremediation capabilities (Rustamov & Shukurova, 2016).

Variation studies are essential for determining the genetic potential of algae and for using this potential more effectively in biotechnological applications. Algae are widely used in the food, pharmaceutical, cosmetic, bioenergy, and environmental sectors, and molecular variation accelerates the development of these fields. Genetic research facilitates the creation of more robust and beneficial algal strains, helping to shape the future of biotechnology (Rustamov & Shukurova, 2016).

Food-significant algae are also used for medical purposes. They are rich in vitamins, minerals, amino acids, and polysaccharides. However, the lack of comprehensive knowledge about their genetic diversity and biological properties limits their optimal use. Molecular variation research is a vital tool for overcoming these limitations (Ahmadov & Safarov, M., 2021).

These algae have a wide range of applications in the food, pharmaceutical, and cosmetic industries. Their genetic diversity determines the variety of their metabolic and bioactive properties, which in turn broadens the scope of their practical uses. Studies on molecular variation are essential for understanding the evolution and adaptation of the biological and chemical characteristics of algae.

Main research. In recent years, advances in molecular biotechnology have opened new opportunities for investigating the genetic structure of algae. These studies reveal the presence of genetic variation across different algal populations and species, and how this variation correlates with their biological activity. Molecular markers, such as SSR (Simple Sequence Repeat), AFLP (Amplified Fragment Length Polymorphism), and RFLP (Restriction Fragment Length Polymorphism), are widely used to assess the genetic diversity of algae. Through these markers, genetic variation at the species and population levels is examined (Tahmazov, R., & Hasanov, N., 2015).

Bioinformatics tools are also used in the study of algal genetic diversity. These tools enhance the speed and accuracy of analyzing algal genetic data. As a result, researchers can identify the variety and quantity of bioactive compounds in algae, such as polyphenols, polysaccharides, fatty acids, vitamins, and minerals. These analyses also provide insight into how different algal species adapt to specific environmental conditions and ecosystems (Alizadeh & Rzayev, 2018).

One of the most significant aspects of these studies is the ability to efficiently identify bioactive compounds associated with molecular diversity and to select genetically superior algal strains. This enables improvements in the quality of algae used in food and medical industries, leading to the development of more targeted and effective products. For example, certain algae with high levels of antioxidants and vitamins can be used as nutritional supplements.

Genetic diversity demonstrates how algae adapt to diverse ecosystems and environmental conditions and how they develop specific beneficial traits. This makes it possible to select more effective and application-specific algal species, particularly for use in the food and pharmaceutical sectors. The use of genetic markers to analyse various species and populations of algae enhances their classification and suitability for specific purposes (Guliyev & Ismayilov, 2017).

Environmental factors also influence the genetic structures and biochemical composition of algae. Conditions such as temperature, salinity, and light intensity can alter the production and content of bioactive compounds, which in turn determine how algae may be used in medical and food-related applications. Developments in biotechnology, especially in genetic

engineering and selection methods, allow for the enhancement of desirable traits in algae and the cultivation of more suitable species.

Climate change also affects this process. Rising global temperatures and other environmental changes may influence the diversity of algae and their impact on ecosystems. Therefore, the conservation and sustainable use of algal genetic resources is of particular importance (Nasirov & Veliyev, 2019).

In conclusion, the study of molecular variation in food-significant algae enables a deeper understanding of their biological characteristics and allows for more efficient use of these species in food and medical applications. Investigating the genetic diversity of algae and adapting them to environmental conditions will facilitate the selection of more productive and beneficial strains in the future. These studies are also crucial for ensuring food security, addressing health-related challenges, and promoting ecological sustainability.

Results and discussions:

Molecular variation studies of food-significant algae allow for a deeper understanding of their genetic structure and biological characteristics. Research in this field provides essential information for the more effective and targeted use of algae. By using molecular markers, scientists can identify both the genetic diversity and the range of bioactive compounds present in algal species, enabling more efficient selection for nutritional and medicinal purposes.

As a result, molecular variation studies expand the application of food-significant algae in biotechnology and pharmaceuticals. The outcomes of such studies pave the way for the development of healthier and more effective nutritional supplements and pharmaceutical products. Selecting algal strains with genetically superior traits is also important for ecological sustainability, as these strains are more adaptable to various ecosystems and can be cultivated in a more resilient and sustainable manner. A better understanding of the genetic diversity and bioactive potential of algae will help researchers discover new commercially valuable species in the future. These species will not only enhance the quality of food products but also open new avenues in the biomedical and cosmetic industries. In conclusion, molecular variation research holds great potential both economically and ecologically.

Research on molecular variation and the biotechnological applications of algae enhances our understanding of the genetic potential of these organisms and promotes the efficient use of their beneficial properties. Algae are widely applied in the fields of nutrition, medicine, cosmetics, bioenergy, and environmental protection. By studying their genetic diversity through molecular variation, more productive, healthier, and effective strains can be selected. This approach accelerates the development of algal biotechnological applications, supports the creation of new bio-based products, and contributes to the advancement of existing technologies. Ultimately, molecular variation studies not only strengthen the ecological contributions of algae but also help unlock their full biotechnological potential.

Conclusıon:

Molecular variation studies of food-significant algae provide critical insights into their genetic diversity, biological traits, and bioactive potential. These studies enable the identification and selection of genetically superior strains with enhanced nutritional and medicinal value, contributing to the development of effective biotechnological and pharmaceutical applications. By utilizing molecular markers, researchers can improve strain selection for ecological sustainability and adaptivity, promoting environmentally resilient cultivation systems.

Moreover, understanding genetic variation helps uncover new algal species with commercial potential in industries such as food, medicine, cosmetics, and bioenergy. This knowledge facilitates the creation of healthier nutritional supplements, novel therapeutic agents, and bio-based products, while also accelerating innovation in environmental protection technologies.

Overall, molecular variation research significantly enhances the economic and ecological utility of algae, making it a key driver for

sustainable development and technological advancement in multiple sectors.

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THEORETICAL AND CONCEPTUAL FOUNDATIONS OF CHRONIC MYELOID LEUKEMIA

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Abstract: Chronic myeloid leukemia (CML) is a myeloproliferative neoplasm characterised by the uncontrolled proliferation and accumulation of myeloid lineage cells. As a malignant disorder, CML involves abnormal hematopoiesis and associated clinical manifestations. Over the past decades, significant advancements have been made in understanding and treating CML; however, further research is still required to elucidate its fundamental mechanisms and pathophysiology. The hallmark of CML is the presence of the Philadelphia chromosome, a genetic abnormality resulting from a translocation between chromosomes 9 and 22. This translocation leads to the formation of the BCR-ABL oncogene, which encodes a protein with constitutive tyrosine kinase activity, promoting uncontrolled cell proliferation and survival by evading apoptosis. CML progresses through three distinct phases: the chronic phase, the accelerated phase, and the blast crisis phase. In the chronic phase, symptoms are often mild and may be detected incidentally through routine blood tests. The accelerated phase is marked by worsening symptoms and an increased risk of progression to blast crisis, the most severe and life-threatening stage, which necessitates urgent intervention. Tyrosine kinase inhibitors (TKIs) represent the cornerstone of modern CML treatment. Drugs such as Imatinib have significantly altered the disease course, improving survival rates and patients' quality of life. However, drug resistance and adverse effects remain challenges, necessitating the exploration of alternative therapeutic strategies.

Keywords: Myeloid leukemia, Disease, Genetic, Chronic, Stem Cell.

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Introduction:

Chronic myeloid leukemia (CML) is a malignant hematologic disorder that arises from the clonal expansion of myeloid cells. Over the past few decades, significant advances in molecular biology and genetics have contributed to an enhanced understanding of this disease. The role of molecular and genetic testing in the diagnosis and monitoring of CML has become increasingly important. Precision in the detection and quantification of key genetic markers, such as the Philadelphia chromosome and the BCR-ABL fusion gene, is crucial in managing the disease. These biomarkers provide not only a means for diagnosing CML but also a valuable tool for monitoring disease progression and evaluating the effectiveness of therapeutic interventions. The ability to detect BCR-ABL at varying levels allows clinicians to track the evolution of the disease over time and identify patients at risk for progression to more advanced stages.

In addition to monitoring disease progression, genetic tests can help identify

early signs of resistance to treatment. Resistance to tyrosine kinase inhibitors (TKIs), such as imatinib, is a major challenge in the management of CML, and molecular testing plays a vital role in identifying mutations in the BCR-ABL gene that may confer resistance. These mutations may be present in a small fraction of leukemic cells, making early detection essential for adjusting therapeutic strategies. Furthermore, the use of genetic testing allows for the personalization of treatment regimens, ensuring that patients receive the most appropriate therapy based on their unique genetic profile. This approach improves the chances of successful treatment and reduces the likelihood of unnecessary side effects caused by ineffective therapies.

In recent years, ongoing research has led to the discovery of additional genetic alterations and novel biomarkers in the genomes of CML patients. These genetic changes, such as mutations in signaling pathways and epigenetic regulators, have opened up new avenues for targeted therapies. By focusing on these additional genetic factors, researchers are identifying potential new therapeutic targets, which may allow for more effective and individualized treatment strategies. These findings highlight the complexity of CML and the need for continuous innovation in treatment approaches. The discovery of new biomarkers also holds promise for improving early diagnosis and better stratifying patients based on their prognosis and potential response to therapy.

The theoretical and conceptual foundations of CML have evolved significantly due to the rapid advancements in biotechnology and genetic research. The development of high-throughput sequencing technologies, which allow for comprehensive genomic analysis, has revolutionized our understanding of the molecular landscape of CML. These technologies have enabled the identification of novel mutations, gene expression profiles, and epigenetic changes that play a role in the pathogenesis of CML. Moreover, recent advances in gene editing tools, such as CRISPR-Cas9, offer the potential for more precise interventions at the genetic level, potentially leading to more effective treatments. These technological innovations, along with ongoing clinical trials, are driving the development of novel therapeutic agents and treatment strategies, which are expected to improve outcomes for patients with CML.

As the understanding of CML deepens, the development of new and more effective treatments continues to progress. These advancements aim to not only improve survival rates but also enhance the quality of life for patients living with the disease. Through ongoing research and collaboration between scientists, clinicians, and patients, the future of CML treatment looks promising. The continued evolution of molecular and genetic testing, along with novel therapeutic approaches, will pave the way for a more personalized and effective management of CML.

Materials and Methods:

The gene panel for chronic myeloid leukemia (CML) was developed by analyzing genetic data from patients diagnosed with CML. Bioinformatics tools were used to identify genetic variants and mutations associated with CML, focusing on genes linked to disease progression, treatment response, and drug resistance. The gene panel was designed to include both known and novel genetic variants for a comprehensive profile of each patient. Blood samples were collected from CML patients across multiple clinical centers, with informed consent obtained from all participants. Control samples were also included to establish baseline genetic profiles for comparison.

DNA was extracted from the blood samples using a commercial kit and assessed for quality and quantity. Next-generation sequencing (NGS) using Illumina platforms was employed to detect genetic variants, mutations, and polymorphisms. The sequencing data were aligned to the reference genome using the Burrows-Wheeler Aligner (BWA), and variant calling was conducted using the Genome Analysis Toolkit (GATK). Variants were annotated with databases such as dbSNP and ClinVar to identify known CML-related

mutations, and novel variants were classified based on in-silico tools to predict pathogenicity.

To validate the identified genetic markers, quantitative PCR (qPCR) and Sanger sequencing were used. These techniques ensured the accuracy of the variants found in both patient and control samples. Clinical data, including age, gender, disease stage, and treatment history, were gathered and correlated with the genetic findings. Statistical analyses, including Chi-square tests and logistic regression, were performed using SPSS and R software to assess the significance of the correlations between genetic markers and clinical outcomes.

The study was approved by institutional review boards (IRB), and informed consent was obtained from all patients. Data confidentiality and privacy were maintained by anonymizing patient information. Additionally, a clinical trial was designed to evaluate the effectiveness of personalized treatment strategies based on the gene panel's findings. Patients were stratified based on their genetic profiles, and personalized treatments were administered, with outcomes monitored to assess improvements in disease progression, treatment response, and survival.

Results and discussions:

XML research is part of our expanding knowledge base in cancer biology and will lead to greater advancements in managing the disease in the future. Furthermore, studies on chronic myeloid leukemia (XML) have increasingly started to incorporate scientific and computer technologies. The application of large data sets and complex statistical modeling enables deeper analysis of the genetic and molecular profiles of the disease. This approach allows for more accurate prognostic indicators and personalized treatment protocols, considering the specific characteristics of cancer in each patient. For instance, the application of artificial intelligence and machine learning technologies can predict the progression of the disease and response to treatment. These technologies assist in rapidly processing large volumes of variables associated with the disease, helping to identify differences between patients who respond to treatment and those who do not.

Chronic myeloid leukemia (XML) is a blood cancer that mainly develops due to genetic changes. The most significant cause of this disease is a genetic anomaly called the Philadelphia chromosome. This anomaly occurs as a result of a reciprocal translocation between chromosomes 9 and 22, leading to the formation of a hybrid gene known as BCR-ABL. The BCR-ABL gene codes for a continuously active tyrosine kinase protein, which promotes the uncontrolled growth and division of blood cells, disrupting the normal development and function of blood cells. Healthy blood cells are gradually replaced by cancerous ones. Other factors that can lead to the development of chronic myeloid leukemia include radiation exposure, such as during atomic bomb explosions or radiation accidents. However, such incidents rarely cause the disease, and genetic factors are still considered the primary cause.

The disease typically occurs in middle-aged and older adults, but exceptions can occur. It can even develop in children, although the highest-risk group consists of individuals aged 50-60. Moreover, innovations in clinical trial design and execution also promise significant advancements in XML treatment. Adaptive trial designs ensure real-time evaluation of data and optimization of treatment protocols.

Chronic myeloid leukemia (XML), though one of the rarer types of blood cancer worldwide, is influenced by the unique genetic characteristics of each ethnic and geographic group, playing a crucial role in its spread and treatment effectiveness. Although data on the genetic characteristics and prevalence of XML in the Azerbaijani population is currently limited, research in this field could improve the understanding of genetic predisposition and aid in personalizing treatment approaches. Investigating the genetic traits of XML in Azerbaijan, particularly the analysis of various BCR-ABL oncogene variants and mutations, is important to understand how the disease spreads in the local population and the response to treatment. Such research may also suggest

potential uses for various genetic markers in the early diagnosis of the disease (Куликов, 2014).

Genetic studies on chronic myeloid leukemia (XML) allow health institutions in countries like Azerbaijan to gain a deeper understanding of the local characteristics of cancer diseases and tailor treatment protocols accordingly. Expanding genetic research not only improves the effectiveness of treatment methods but also provides a foundation for reducing potential side effects and increasing patient survival rates. In this regard, genetic research conducted in Azerbaijan can assist in the identification of new biomarkers and targets for more accurate diagnostics, treatment personalization, and improved survival rates. For example, identifying genetic variants that cause resistance in some patients could play a crucial role in selecting alternative drugs or therapy combinations for these patients (Бутенко, 2001). Furthermore, such research allows for the real-time monitoring of biomarkers to evaluate the response to treatment. This helps in closely monitoring the progression of the disease and adjusting the treatment to suit the patient's current health status. Local genetic research contributes to optimizing XML treatment in Azerbaijan and achieving better outcomes for patients. This process benefits both patients and national health systems, as more effective treatment methods can reduce costs and improve patients' health and quality of life. Thus, local genetic research is critical in the fight against chronic myeloid leukemia, and investments in this area can offer substantial returns for the development of the healthcare sector (Кресова, 2014).

One of the most important treatment methods, Tyrosine Kinase Inhibitors (TKIs), has been a turning point in the treatment of XML. These drugs provide the possibility to slow the progression of the disease and improve patients' quality of life. However, some patients develop resistance to these drugs over time, which necessitates a reconsideration of treatment strategies (Куклев, 2016).

Immunotherapy and Stem Cell Transplantation are treatment options that offer alternative approaches, especially for advanced stages of the disease. Immunotherapy stimulates the patient's immune system to fight cancer. Stem Cell Transplantation, on the other hand, is a more radical treatment, often applied when other therapies fail, particularly in severe cases. Next-generation therapies include innovative approaches such as gene editing and targeted molecular interventions. These treatments offer promising alternatives, particularly for patients who have developed resistance to TKIs or have not benefited sufficiently from standard therapies. The collection and analysis of local data on the genetic characteristics and prevalence of chronic myeloid leukemia plays an important role in shaping healthcare policies in countries like Azerbaijan and the fight against the disease. This could also be an important step in providing more effective and accessible treatment options for patients (Туркина, 2017).

Bioinformatics analysis of chronic myeloid leukemia (CML) is a crucial tool for the in-depth study of the genes responsible for this disease. CML is a type of blood cancer mainly associated with the formation of the Philadelphia chromosome. This chromosome results from the fusion of the BCR and ABL1 genes. Bioinformatics plays a significant role in the analysis of these genetic alterations, in identifying gene expression profiles, and in predicting responses to treatment. With the development of genetic sequencing technologies, bioinformatics methods have opened new horizons in understanding the genetics and pathophysiology of CML (Жуков, 2000). Molecular-level analyses allow for a more precise identification of the biological foundations of the disease and its resistance tendencies to treatment. The improvement of the effectiveness of drugs, such as tyrosine kinase inhibitors (TKIs), which inhibit BCR-ABL kinase activity, and the reduction of their side effects, is based on the results of these analyses. Bioinformatics analyses provide essential information in identifying genetic variants and polymorphisms, as well as in tracking changes in gene expression and signaling pathways. As a result of these analyses, the relationship between the different biomarker profiles of CML patients and their

response to treatment becomes clearer. This enables doctors to design more targeted and individualized treatment strategies for each patient. The bioinformatics analysis of the genes responsible for CML ensures a better understanding of this serious disease and helps achieve progress in its treatment. These analyses play an important role in the treatment of genetic diseases and the development of large-scale research and clinical practices, opening new ways to increase patients' survival rates and improve their quality of life.

With the expansion of these genetic and molecular analyses, new biomarkers and potential therapeutic targets in the treatment of CML may also be discovered (Кресова, 2014). Through the bioinformatics analysis of CML genes, the molecular mechanisms of the disease are identified more accurately, providing a foundation for the improvement of treatment. For example, certain genetic variants or segments may exhibit resistance to treatment, in which case alternative drugs or therapeutic strategies could be applied. Progress in bioinformatics and molecular biology allows for a better understanding of various aspects of the disease, including genetic predispositions, disease progression, and response to treatment. This knowledge also plays a crucial role in the design and implementation of clinical trials, as these trials can target more precise patient groups and evaluate the effectiveness and safety of specific treatment types (Андросова, 1996). Bioinformatics analyses also encourage the development of innovative approaches in the treatment of CML, such as gene therapies and targeted treatments. For instance, gene editing technologies can be used to correct disease-related genetic defects and restore healthy cell functions.

The bioinformatics analysis of genes responsible for chronic myeloid leukemia (CML) continues to create revolutionary changes in medical research and clinical practices. These advancements provide significant benefits in improving patients' survival rates and quality of life. At the same time, they contribute to the expansion of scientific understanding and the enhancement of healthcare service effectiveness. It enables doctors and researchers to assess the relative importance of each gene in the biological context of CML. The comparison of gene expression levels provides valuable information in the management of the disease and the development of new treatment strategies. These analyses play a crucial role in evaluating the effects of drugs such as tyrosine kinase inhibitors (TKIs) in the treatment of chronic myeloid leukemia. For example, the "BCR-ABL" gene is recognized as the primary target for TKIs because its hyperactivity plays a critical role in the development of CML.

The accurate measurement of the expression levels of this gene forms the foundation for assessing the effectiveness of these drugs on the patient and evaluating the disease's progression. Additionally, the investigation of other genes such as "TET2," "ASXL1," and "DNMT3A" enables the creation of a more complex genetic portrait of CML (Fig. 1). These genes are associated with epigenetic modifications and cell differentiation. Their activity can influence treatment resistance and affect the course of the disease. Understanding the expression levels of these genes helps to further personalize treatment strategies and potentially discover new ways to overcome resistance. Moreover, this genetic data provides a foundation for uncovering the deeper biological mechanisms of cancer and improving treatment methods (Львов, 1989).

For example, specific genetic variants and mutations identified through bioinformatics analyses can reveal differences between various types and subtypes of the disease. Understanding these differences enables the offering of more suitable treatment options for patients and the development of personalized therapy strategies based on each patient’s needs. The creation of a new gene panel for chronic myeloid leukemia (CML) represents a significant advancement in the diagnosis, treatment, and management of this disease. CML is a type of blood cancer in which genetic factors play a crucial role, and the key genetic event that causes this disease is the BCR-ABL gene fusion (Fig. 2). A new gene panel, which includes this and other genetic markers, can ensure faster and more accurate detection of the disease and facilitate the development of

personalized treatment approaches (Куликов, 2014). This new gene panel will cover genes related to CML, including resistance variants and other genes potentially involved in treatment response. It will provide a deeper insight into the biological and molecular profile of the disease and help make treatments more targeted and effective. For example, based on the specific genetic profile identified by the gene panel, stronger tyrosine kinase inhibitors may be prescribed for some patients, while gentler treatment methods may suffice for others. The application of this gene panel will also be valuable for clinical research because it will allow a better understanding of various aspects of the disease. The discovery of new biomarkers and therapeutic targets in the fight against the disease may be faster and more effective due to this panel. This innovation will play a significant role in improving survival rates and quality of life for CML patients. The implementation of the new gene panel will also assist in the early detection and monitoring of chronic myeloid leukemia (CML). Early diagnosis offers the opportunity to slow the disease's progression and achieve better treatment responses. This panel can provide the necessary genetic data to clarify the stages of the disease and make treatment options more targeted. For instance, the genetic variants identified by the gene panel play a key role in personalizing the treatment regimen (Кресова, 2014).

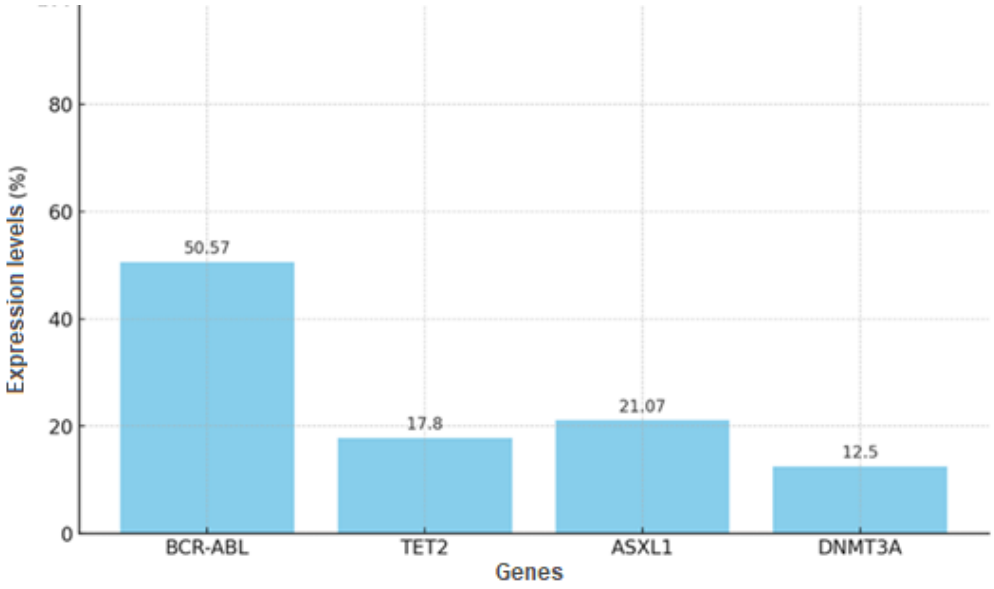

Fig. 1. Expression levels of genes responsible for chronic myeloid leukemia (Туркина, 2017).

Doctors can use this data to develop specific treatment protocols based on each patient's genetic profile. This approach is a crucial step in increasing the effectiveness of treatment and reducing side effects. Furthermore, the new gene panel will allow for a better understanding of cases resistant to treatment. Identifying resistant variants enables doctors to apply alternative treatment pathways promptly, which increases the patient's chances of survival. Additionally, studying resistance mechanisms lays the foundation for the development of new drugs and treatment strategies. The development and application of the gene panel will allow for a deeper understanding of the complex genetic and molecular dynamics of the disease. The comparison of gene expressions can help identify potential treatment targets and biomarkers, which can be used in the management of CML patients and further personalize treatment plans. The implementation of the new gene panel will allow for the development of more targeted approaches in the management and treatment of chronic myeloid leukemia (CML). This panel aims to increase the effectiveness of treatment,

reduce resistance, and minimize side effects. Based on gene expression data, doctors can tailor treatment plans more accurately, particularly by offering personalized therapies according to each patient's genetic characteristics. (Туркина, 2017).

Furthermore, the new gene panel will play a crucial role in the early diagnosis of CML. Accurate identification of genetic markers can ensure the detection of the disease in its early stages, which leads to more effective treatment at the outset. Early diagnosis not only increases the patient's chances of survival but also has the potential to reduce treatment costs. The new gene panel will also contribute to clinical research. Through this panel, the effects of various genetic variants on treatment can be studied comparatively. This information can help in better understanding the different variants of the disease and potentially lead to the development of new treatment methods. For example, if certain genetic profiles respond better to treatment, treatment strategies can be developed and tailored to those profiles. The new gene panel will lead to revolutionary changes in CML research and treatment. These advancements will offer significant innovations to improve survival rates, enhance treatment effectiveness, and improve the quality of life for patients. By providing researchers and doctors with new insights and treatment options, this development will open a new chapter in the battle against CML (Туркина, 2017).

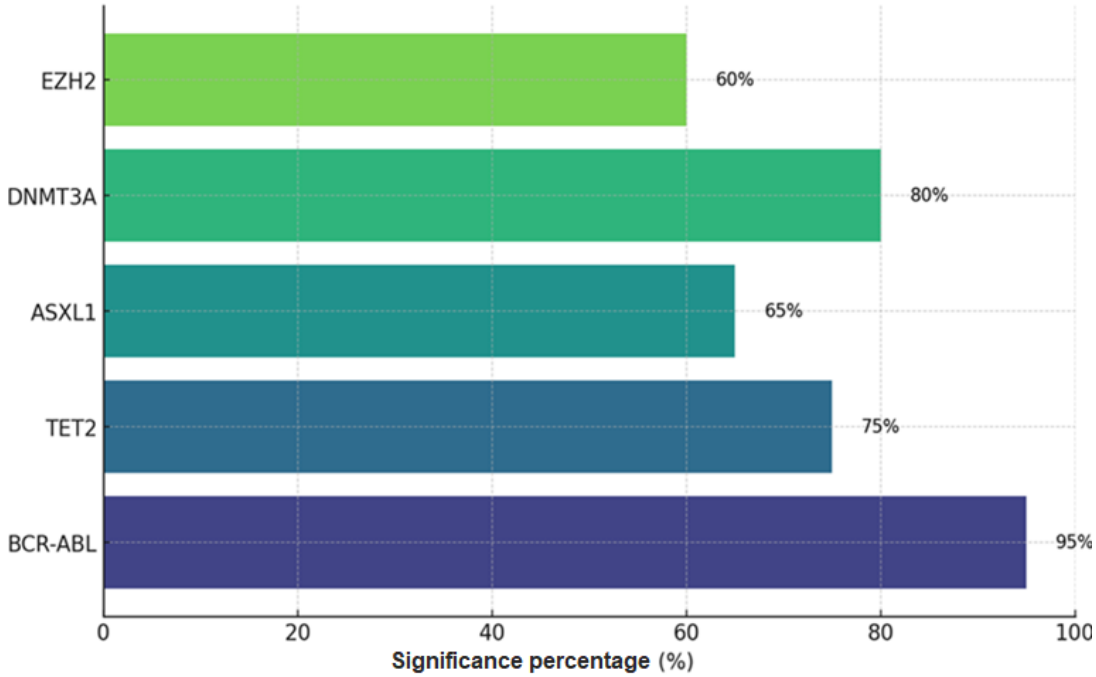

Fig. 2. Significance values of genes in the new gene panel for chronic myeloid leukemia (Куликов, 2014).

Conclusıon:

The newly created gene panel marks the beginning of a new era in research and treatment of chronic myeloid leukemia (CML). This panel provides a deep view of the disease at the genetic level and allows for the development of more effective treatment methods tailored to the genetic characteristics of each patient. Specifically, the precise identification of genes associated with CML is crucial for early diagnosis, improving treatment response, and managing resistance cases. This gene panel also helps in more targeted planning and implementation of clinical trials, providing a foundation for discovering new therapeutic targets. As a result, the application of this panel

creates a pathway for the development of effective treatment approaches that can increase survival chances and improve the quality of life for CML patients. Overall, the creation of this new gene panel is a significant advancement in the fight against chronic myeloid leukemia. Its widespread application in future research and clinical practice promises better outcomes for patients. These advancements will contribute to further improvement in genetic and molecular research in the medical field and increase the effectiveness of large-scale healthcare services. Innovations also enable patients and their families to have a deeper understanding of the disease, allowing them to be more informed and actively participate in the treatment process. A better understanding of patients' genetic profiles ensures that disease-related decisions are made more transparently and personalized, thereby increasing patient satisfaction and treatment adherence. At the same time, the new gene panel provides healthcare policymakers with the opportunity to develop more informed strategies for managing chronic myeloid leukemia. This information helps healthcare systems manage their financial and resources more efficiently, as treatment methods can be applied more purposefully and effectively. In the future, further development and expansion of this gene panel will lead to greater progress in the fight against CML. The gene panel may expand to cover more genetic markers and biomarkers, allowing for a more precise biomolecular profile of the disease. These advancements will ensure a deeper understanding of diseases and the discovery of more effective treatment options. In conclusion, the creation of a new gene panel for chronic myeloid leukemia is considered a turning point in the management and treatment of the disease. This innovation marks a new phase in medical research and opens wide possibilities for improving patients' health outcomes. This is a valuable and significant advancement for both the scientific community and the general public.

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THE EFFECTS OF EROSION ON THE SOILS IN THE OGUZ REGION

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Abstract: In modern times, many natural ecosystems have suffered significant degradation due to global climate change, particularly soils formed in vertical belts, which are of special interest. This article offers detailed information about the soils in the Oguz region and discusses how their typical diagnostic indicators have changed due to the erosion process.

Keywords: ridge, climate, air temperature, soil erosion, genetic section, soil type

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Introduction:

The Main Caucasian Range is a system of mountain folds that are not crossed by rivers. It is characterised by high-mountain relief, with a watershed that generally takes the form of a narrow ridge where the highest peaks, Tufan and Bazarduzi, are located. The main features of the relief in this geomorphological region indicate that local mountain ranges and elevations are classified as anticlines while the depressions are classified as synclines (Budagov, 1969).

In the Oguz region, both at depth and at the surface where parent rocks are exposed, the sedimentary materials are composed of clays, limestones, loams, and conglomerates. These materials are susceptible to the destructive effects of water, significantly contributing to gully erosion in the area. Climate is an active natural factor that plays a crucial role in soil formation. The climatic characteristics largely determine the occurrence and development of erosion processes and the intensity of these processes (Museyibov, 1998).

To understand the impact of climate on erosion, we must first describe the climatic conditions of the region. Azerbaijan has a diverse range of physical and geographical conditions across its various natural zones, leading to the formation of different climatic types. There are eight climate types in Azerbaijan, ranging from humid subtropical to mountain tundra. Three of these types are typical of the Oguz region:

1. Moderately warm steppe climate with dry summers.
2. Moderately warm steppe climate with dry winters.
3. Moderately warm climate with dry winters.

The first two climates cover the southern part of the region, while the third type is found in the central and northern areas. The air temperature is affected by the area's altitude above sea level. Between the lowest point and the watershed, the temperature difference can reach -20°C in winter and +30°C in summer.

The significant changes in temperature, both throughout the day and by season, contribute to the degradation of the soil surface, which in turn hinders vegetation growth and increases soil erosion during heavy rainfall. In winter, when temperatures drop below 0°C (especially in the elevated parts of the region), the soil surface can freeze quickly. However, it is important to note that the average monthly soil

temperature during the coldest months of winter remains positive.

Research methods:

The study utilized both ground-based measurement data and digital maps created through the interpretation of satellite images. This approach ensures high accuracy in geographical analysis and modelling. Ground-based measurements reflect data obtained in field conditions and are integrated with information derived from satellite imagery to enhance the precision and scalability of the research findings. The interpretation of satellite images enables the study of terrain structure, landscape changes, and other critical geographical elements. This method serves as an effective tool for analyzing and modelling the environment according to the objectives of the research.

Results and discussions:

In the Oguz district, the annual precipitation totals 500 mm, with the majority occurring during the spring and autumn months. The highest levels of soil erosion are typically recorded in the spring, coinciding with the peak precipitation. This rapid onset of erosion in spring is largely due to the intense rainfall, which overwhelms the soil's capacity to absorb water, resulting in surface runoff and accelerated erosion (Fig. 1).

During the winter months, although little snow falls, some soil erosion still occurs. The winter is relatively mild, preventing the formation of a permanent snow cover. Snow is only present when temperatures drop, with coverage ranging from 8 to 14 cm thick. This snow typically lasts from 15 to 60 days, appearing in late November or early December and melting by March.

Figure 1. Space image of the Oguz district

The peripheral zone that borders the mountain ranges is characterized by steppe soil formation. This area features both dark grey-brown and light grey-brown (chestnut) soils, as shown in Table 1 and illustrated in Picture 2 (Aliyev, 1978).

Table 1. Water-physical properties of steppe mountain-brown soils of the Oguz district.

Section	Genetic horizon.	Depth in sm.	Field moisture %	Bulk density g/sm^3	Specific gravity g/sm^3	Total porosity %
1 Unwashed	A	0-36	19,85	1,06	2,45	55,61
	B	36-59	18,49	1,12	2,49	57,3
	C	59-88	16,19	1,14	2,52	57,9
2 medium washed out	A	0-27	18,94	1,04	2,54	58,2
	B	27-44	18,35	1,09	2,54	58,7
	C	44-67	16,79	1,11	2,51	58,9

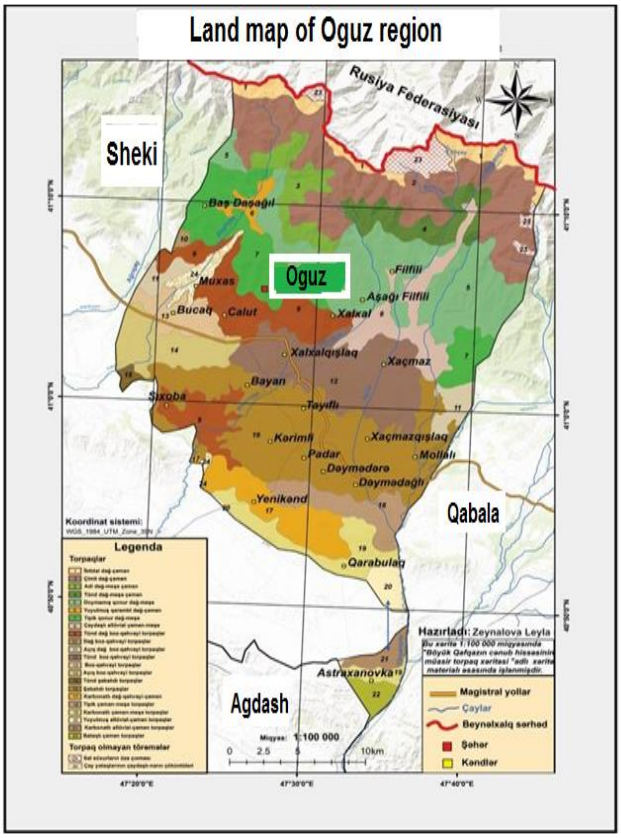

Figure 2. Soil map of Oguz district

The research was conducted in the foothills of the Oguz district, where soil sections were examined in both unwashed and moderately washed areas.

Section No. 1: Unwashed Soil

- A (0-36 cm): Dark brown with a granular-nutty structure, containing live roots; the soil is heavily loamy, and wet, and shows a clear transition.

- B (36-59 cm): Dark brown with roots; this layer is also heavily loamy, wet, and has a clear makeup.

- C (59-88 cm): Light brown, with an indistinct structure; fragments of slightly weathered rocks are present, and it effervesces when treated with HCl.

Section No. 2: Moderately Washed Soil

- A (0-27 cm): Brown with a lumpy-granular structure; this soil is heavy loamy and wet with a clear transition.

- B (27-44 cm): Light brown with a lumpy structure; heavy loamy and moderately wet, with a clear transition.

- C (44-67 cm): Light clayey with no expressed structure; contains fragments of parent rock, is dense, and boils when treated with HCl.

Table 2 presents the water-physical properties of mountain-brown steppe soils in the Oguz region. The total porosity, specific weight, and volumetric weight of unwashed soils in the upper horizon are 55.6%, 2.45 g/cm³, and 1.06 g/cm³, respectively. In moderately washed samples, these indicators increased, suggesting an ongoing erosion process.

Table 2. Particle size distribution of mountain brown soils of Oguz district

Section	Genetic horizon.	Depth in sm.	Factions %						
			1-0,25	0,25-0,05	0,05-0,01	0,01-0,005	0,005-0,001	<0,001	<0,01
3 Unwashed	A	0-25	6,95	0,92	20,56	9,84	18,00	42,00	69,84
	B	25-57	4,15	1,70	19,42	7,85	16,12	36,22	61,82
	C	57-86	5,35	11,20	21,42	7,33	19,10	33,45	64,52
4 medium washed out	A	0-11	5,74	2,45	24,85	11,15	19,34	34,65	51,75
	B	11-39	2,35	10,55	13,14	8,09	17,55	29,15	47,65
	C	39-67	0,75	6,74	11,35	7,91	15,64	22,41	44,99

Regarding the mechanical composition, the soils are classified as heavy loamy with a physical clay content of 69.84%. However, in moderately washed samples, the physical clay content is noticeably lighter (refer to Table 3). According to Table 4, the structural-aggregate composition shows that the content of aggregates larger than 1 mm during dry sifting was 60.85% in unwashed samples and 33.80% in moderately washed samples. In the upper 0-27 cm horizon of moderately washed samples, these values were recorded at 52.68% and 21.20%, respectively.

In terms of agrochemical indicators, the mountain-brown steppe soils of the Oguz district contain a total nitrogen content of 0.344% and a humus of 5.2% in unwashed samples. The sum of exchangeable cations was 38.23 m.eq. per 100 g of soil. In moderately washed samples from the upper 0-27 cm horizon, total nitrogen is 0.279%, while humus content is 3.1%, indicating a downward trend as the depth increases.

Table 3. Structural and aggregate composition of mountain-brown steppe soils of the Oguz district.

Section	Genetic horizon.	Depth in sm.	Factions мм							
			>7	7-5	5-3	3-1	1-0,5	0,5-0,25	<0,25	>1
1 Unwashed	A	0-36	35,42 8,80	6,31 7,00	9,42 11,60	9,70 16,40	1,01 14,60	0,94 6,40	2,14 27,00	60,85 33,80
	B	36-59	31,11 3,40	9,04 8,60	7,25 9,50	8,38 14,60	0,73 15,00	0,63 6,20	1,96 32,40	45,88 36,10
	C	59-88	20,44 -	3,40 5,60	6,79 11,80	12,34 9,60	22,10 14,70	82,40 10,10	32,15 39,80	42,97 27,11
2 medium washed out	A	0-27	25,67 -	9,91 1,00	9,76 7,00	7,17 13,60	0,74 15,30	0,58 12,70	1,59 42,90	52,68 21,20
	B	27-44	15,65 -	1,58 1,40	10,75 3,20	8,17 10,05	0,98 14,60	0,46 9,10	13,41 49,70	44,32 14,65
	C	44-67	11,31 0,91	7,32 2,51	5,68 5,49	4,14 3,75	15,77 24,75	28,95 32,84	26,83 29,75	28,45 12,66

Table 4. Some agrochemical indicators of mountain-brown steppe soils of the Oguz district.

Section	Genetic horizon	Depth in sm.	Ca+Mg/eq. 100g. soil			Total Nitrogen%	humus, %	C:N
			Ca	Mg	Ca+Mg			
1 Unwashed	A	0-36	32,11	6,12	38,23	0,344	5,2	8.77
	B	36-59	30,52	5,8	36,32	0,336	4,8	8.28
	C	59-88	27,41	4,7	32,11	0,265	3,4	7.44
2 medium washed out	A	0-27	23,15	4,31	27,46	0,279	3,1	6.44
	B	27-44	19,49	3,09	22,58	0,235	2,6	6.41
	C	44-67	14,19	2,8	16,99	0,174	1,9	6.30

Conclusıon:

In both samples provided, we observe differences in the shapes and sizes of morphological and diagnostic indicators throughout the soil profile. The gradation of transitions along the profile is illustrated in the soil profile example. Additionally, erosion and degradation have resulted in the reduction of the soil profile, leading to a decrease in absorbed cations and humus in the productive sowing layer.

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GENETIC POLYMORPHISM TNF-α GENE OF GUM DISEASE IN PATIENTS WITH DIABETES MELLITUS (TYPE 2)

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Abstract: Approximately 400 million people worldwide suffer from diabetes, and this number will increase by about 50% by the year 2030. Gingivitis, is a gum disease of the teeth, which is part of the periodontium, supports the teeth. It is estimated that severe periodontal disease affects about 19% of the world's adult population and represents more than 1 billion cases worldwide. In the description WHO Oral Health Status Report (2022) poor oral hygiene and tobacco use are the most common factors for periodontal disease. There is evidence that there is an association between these two chronic conditions. Although studies have been conducted on the immune system and its components, mechanisms have not been fully understood. This article will discuss associations between diabetes and oral health, focusing on periodontal diseases, find relation genetic polymorphism in TNF-alpha gene.

Keywords: Diabetes, gum disease, diabetus mellitus type 2, TNF-α gene, genetics of diabetes.

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Introduction:

Gingivitis is an inflammatory disease of the gum tissue, which is a component of the periodont that surrounds the tooth. Although the etiology includes the microflora of the oral cavity, changes in the body's metabolism and external environmental factors increase the risk of disease. We can see the most prominent effect of metabolic dysfunctions in the case of diabetes. Diabetes is a disease that is common against the background of hyperglycemia due to lack or lack of insulin hormone or inability to use it. In 1998, 4 types of forms were approved by the American Diabetes Asossation. Of these, although idiopathic and gestational diabetes are less common, type 1 and Type 2 have become a kind of epidemic [1]. It should also be noted that, in 1935 Hinsworth identified 2 types of diabetes, for almost 2000 years it has been identified as just 1 disease. Both common types of diabetes are characterized by a persistent increase in its glucose in the blood plasma, but type 1 diabetes (T1D) is an autoimmune disease that causes the complete loss of insulin-producing cells in the pancreatic islets, type 2 diabetes (T2D) occurs due to increased resistance to insulin circulating in target tissues (especially muscle, liver and fat) despite insulin secretion from the islets [2].

Etiopathogenesis of gingivitis:

Quantitatively hundreds of thousands of bacteria colonize the oral cavity in lifetime [3]. The quantitative balance, which changes due to the existing status of the organism, does affect the soft tissues of the oral cavity including the gingiva. Mainly gram negative and gram positive bacteria are responsible for inflammation of the gum tissue. These bacteria secrete lipopolysaccharide endodoxins, stimulating magrophages that cause gingival destruction, synthesizing interleukin-1 beta (IL-1β), tumor necrosis factor-alpha (TNF-α), prostaglandin E2 (PGE2) [4]. Hyperglycemia in diabetes has

been shown as an important risk factor for the manifestation of vascular complications. The five classic complications associated with T2D include retinopathy, neuropathy, nephropathy, cardiovascular complications (coronary arterial disease, stroke, and peripheral vascular disease), and chronic wound healing. Periodontal disease has recently been recognized as the *"sixth complication"* of type 2 diabetes [5] . As a manifestation of this complication, we can see accumulation non-enzymatic glycolysis products (AGE-advanced glycation end-products) and activation of sensitive receptors (RAGE – receptor advanced glycation end-products). These receptors are located on neutrophils and macrophages, which provide stimulation of phagocytic activity during inflammation. Under the influence of diabetes, proteins such as collagen, lipids, nucleic acids ,entering into non-enzymatic glycolysis and oxidation with aldose sugars, configuring irreversible molecular structure, which accelerates vascular wall permeability, immune response IL-1 and TNF-α hyperactivity, synthesis of IgA and IgG, leading to intensive disintegration of gums and surrounding tissues [6].

II. The role of TNF-α

TNF-α is mainly produced by macrophages. It is an effective immuno-inflammatory mediator and can promote bone resorption by activating the maturation of osteoclasts (bone-destroying cells). Stimulates the production of related cytokines, increases the adhesion expression of molecules, promotes the activation of neutrophils and T cells. It plays a key role in the pathogenesis of some serious chronic inflammatory and autoimmune diseases [7]. A Meta-analysis showed that TNF-alpha can be used as an additional criterion for a more accurate diagnosis of periodontal inflammation. Single nucleotide polymorphisms SNPs are the most common type of genetic variation in humans.. Studies have shown that single nucleotide polymorphisms, specifically the transition from G to A at position 308, increase TNF-a production fivefold in vitro [8]. Tumor necrosis factor alpha (TNF-α) is a proinflammatory cytokine that has an important role in the pathogenesis of a several diseases. The gene encoded by is located on the short arm of chromosome 6p21.3 (Figure 1), in the region of the main histocompatibility complex (MHC)Class III. Most TNF-α gene polymorphisms are located in its promoter region and are believed to affect the susceptibility or severity of various human diseases. This review summarizes data on the relationship between the TNF-α gene and its receptor polymorphisms and the development of autoimmune diseases. Genetic changes in the promoter region can regulate TNF-α production, transcription, and affect susceptibility to inflammation-related diseases. Some studies have investigated single nucleotide polymorphisms in the promoter region of the TNF gene, such as 238G/A, 308G/A, 857C/T, and 1031T/C in humans. Although several studies have focused on the relationship between the TNF-α-308g/a polymorphism and T2DM, their results remain uncertain, leading to the need for further research. It plays a central role in the development of T2DM, according to research by Feng, Y.Li, and others [9]. Swetha Chikoti, Umme Najiya and others was determined polymorphism of the TNF-alpha gene -308 G/A according to a study of 400 patients in the southern Indian population. As a result among 200 T2DM and 200 control patients, gender, age, sugar, cholestorol values were studied and statistical analysis was carried out. Genotyping was studied at these loci −238G/A; rs361525 and −308G/A; rs1800629. gel-agarose results of the rs1800629 locus GG / 129 n.c. GA / 149 n.c. (Figure 1) are monitored. Allele and genotype frequencies for all SNPs were calculated using the Chi-square criterion [$\chi 2$] to estimate intergroup significance.

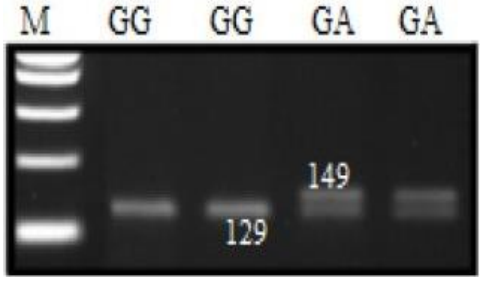

Figure1. Agarose-gel polymorphic result.

The risk of disease by genotype was determined by determining the odds ratio (OR) with

a confidence interval (CI) of 95%, respectively (Figure 2). The G / A genotype increased more than 2 times (1.70-3.85) in relation to others [10].

SNP	Genotypes				HWE	Alleles				Odds ratio	
rs1800629 (−308G/A)	AA (%)	GA (%)	GG (%)	χ^2 (p-value)	χ^2 (p-value)	G	A	χ^2 (p-value)	Group comparison	OR 95 % CI	p value
HC (200)	84 (42)	93 (47)	23 (11)	18.21 (0.0001)	0.13 (0.71)	0.35	0.65	0.005 (0.94)	AA vs. others	0.50 (0.33–0.76)	0.001
T2DM (200)	53 (26)	135 (68)	12 (6)		33.5 (0.000)	0.40	0.60		GA vs. others	2.56 (1.70–3.85)	0.00001
									GG vs. others	0.49 (0.23–1.02)	0.09

Figure 2. Genotype and allele frequency distribution of TNF-*α*.

It has been widely researched that Periodontal disease is one of the main causes of tooth loss in people with diabetes. Individually, many mechanisms have been proposed that explain the increased susceptibility to periodontal disease in patients with unchecked T2D, including changes in collagen metabolism and vascular wall. In addition, poorly controlled T2D patients show an excessive inflammatory reaction to the bacterial hazard of periodontitis. Such hypersensitive reactions lead to a delay in the regeneration of intra-oral tissues, the completeness of which is impaired against the background of increased inflammation, as well as to the degeneration of periodontal tissues [11]. A small percentage of non-autoimmune diabetes (5% or less) is caused by monogenic causes and is classified as juvenile or MODY(monogenic diabetes of the young) monogenic diabetes. These changes are caused by individual high penetrance genes, in which mutations in the nuclear factor-1a (HNF-1a) and Glucokinase (GCK) gene of hepatocytes are most common. These forms of diabetes are sometimes mistaken for T2D, but they are different diseases in terms of their clinical course. Decommunization is important, considering that the boundaries between polygenic and monogenic forms are not always clearly defined at the genetic level [12]. Poulami, Keheibamding and their colleagues found that single nucleotide polymorphism of this gene (TNF-afla) is found in the aggressive course of gum disease. So, as a result of research on 397 people, SNPs were detected. In this study, 40 people were identified as patients with aggressive periodontitis, 157 as patients with chronic periodontitis, and 200 as healthy controls. The study, conducted among both women and men of different age groups (Table 1), characterizes the population of the East-India region. Five SNPs of the promoter site of the TNF-alpha gene, (rs361525, rs1800629, rs1799724, rs1800630 and rs1799964) were genotyped by PCR sequences in patients with periodontitis and control group. The aim is to find out the relationship of polymorphisms of the TNF-alpha gene with both chronic and aggressive periodontal diseases in the Indian population and to analyze the combination and distribution of haplotypes in acute and chronic periodontitis in both populations of patients.

As a method, the current state of the oral cavity of individuals in this population including gingiva and bone melting was evaluated and indexed by evaluating gingival pockets on all teeth, (Table 2) and on the gene-238G/A (rs361525) polymorphism F-5` CAGTGGGGTCTGTGAATTCC3` R-5`TCCCTCTTAGCTGGTCCTCT3`,-308G/A(rs1800629) F-5` CAGTGGGGTCTGTGAATTCC3` ; R-5`GGGCGGGGAAAGAATCATTC3`, -857C/T (rs1799724) F-5` CTGCTTGTGTGTGTGTGTCT 3` R-5` CCGGAGACTCATAATGCTGGT3` -863C/a (rs1800630) and - 1031T/C (rs1799964) respectively F-5` GTGTGTGTCTGGGAGTGAGA3`; R-5` GCAGGCCTTCTTCTTCATTCT3`, F-5` GAGAGAAAGAAGTAGGCATGAGG3` R-5` TCTTAAACGTCCCCTGTATTCCA3` amplified by PCR method using primer sets.

Table 1. Age, gender parameters of patients in the study (AP-agressive periodontitis, CP-chronic periodontitis, P<0,05).

Parameters	***AP***	***CP***	***Kontrol***	***AP vs Control (Pvalue)***	***CPvsControl (Pvalue)***
Age (year)	17-44	22-69	24-65		
Average	30.23± 6.81	41.59± 11.12	38.41± 9.48	0.0001	0.0038
Man(%)	60	65,33	47,5	0,1515	0,0016
Woman(%)	40	34,67	52,5	ref.	ref.

Table 2. Gingiva rates in patients (GP-gingival pocket, BR-bone resorbtion, TP-tooth plaque index, Gİ-gingival index).

Parameters	**Aggressive periodontitis**	**Chronic periodontitis**	**Control**
GP *(all teeth,ave. ±mm)*	6.01± 1.94	6.36 ±1.62	0.34 ±0.66
BR *(ave. ±mm)*	8.3±2.21	8.79±1.94	0.03±0.21
DI	2.83±1.08	2.95±0.81	0.05 ±0.2
GI	3.05 ±0.85	2.61±1.01	0.01±0.08
P value <0.05			

Amplified solutions were electrophoresized in 2-3% agarose gel. The PCR products were sequenced (by the Sanger method) by the Prism 3100 DNA Genetic Analyzer (biosystem, Carlsbad, CA, USA).

The difference between clinical parameters was assessed using a single - factor analysis-ANOVA. Age, gender, ethnicity, smoking, chewing tobacco, and the habit of drinking tea have been used as independent variables for their multiple analysis. All statistical analyses were performed with commercially available SPSS software (version 16.0, SPSS Inc., Chicago, Illinois, USA).

Genotypes – 238 G/A (rs361252) and –308G/A in the AP population as well as polymorphisms-308G/A and-1031T/C (rs1799964) in the CP community are interrelated and are likely to be passed down from generation to generation. The genotype level of TNF-alpha-308 G/A (rs1800629) was significantly higher in patients with both AP and CP compared to healthy control groups [13]. Several studies have been conducted to assess the relationship between TNF-a promoter polymorphisms and periodontitis in different populations, but this is still a matter of controversial discussion [14,15]. There are several contradictions regarding the TNF-a gene as a candidate for genetic studies related to gingivitis and complications. There is reason to believe that the TNF-a gene plays an important role in the pathogenesis of periodontitis, since it is a powerful immunological mediator with anti-inflammatory properties [16].

Conclusıon:

Consequently, the TNF-alpha gene, which controls inflammatory processes and plays a leading role in the formation of immune response reactions, manifests itself with complications when exposed to polymorphism of the promoter region -308 G/A in both patients with gingivitis and patients diabetes mellitus with poor sugar control.

Abbreviation:

T2DM - type2 diabetus mellitus, TNF - tumor necrosis factor, HWE - Hardy-Weinberg Equilibrium

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