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# NEW ASPECTS OF METABOLISM POST-COVID-19

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**Abstract**
The paper presents the results of studies on the impact of COVID-19 on metabolism and in particular lipids, as well as the genes regulator of metabolism, which reflects the antioxidant potential of the body. The possibility of using body mass index with control to increase the activity of the immune system and its resistance against coronavirus infection, both in preventive measures and as an additional element in medical procedures during the SARS-CoV-2 pandemic has been shown.

**Keywords:** COVID-19, post-COVID-19, physiology, metabolism, blood, body mass index.

## Introduction

The coronavirus disease, the disease caused by the severe acute respiratory syndrome virus 2 (SARS-CoV-2), had a devastating effect on the population of the world, causing more than 6.64 million deaths worldwide. The World Health Organization (WHO) declared it a global pandemic on March 11, 2020. The virus continues to cause disease, with many countries experiencing multiple waves of outbreaks. The Coronaviridae family includes 2 subfamilies and 46 species. The first subfamily - Letovirinae was separated in 2018 and includes 1 family (Alphaletovirus), 1 subfamily (Milecovirus), 1 species (Microhyla letovirus1). The second subfamily - Orthocoronavirinae family is divided into 4 families [13].

1) Alphacoronavirus family - 14 subfamilies and 19 species;
2) Betacoronavirus family - 5 subfamilies and 14 species (including SARS-CoV-2 also belongs to this family);
*a) Embecovirus*
*b) Hibecovirus*

*c) Merbecovirus*
*Middle East respiratory syndrome-related coronavirus (MERS-CoV)*
*d) Nobecovirus*
*e) Sarbecovirus*
*SARS-CoV*
*SARS-CoV-2*
3) Gammacoronavirus family consists of 3 subfamilies and 6 species;
4) Deltacoronavirus family includes 3 subfamilies and 7 species.
To date, 7 types of coronaviruses (HCoV-229E, HCoV-OC43, HCoV-NL63, HCoV-HKU1, SARS-CoV, MERS-CoV and SARS-CoV-2) have been found to infect humans. [3]. The new coronavirus disease was named SARS-CoV-2 (severe acute respiratory syndrome coronavirus-2) due to its high homology (~80%) to SARS-CoV, which caused acute respiratory distress syndrome (ARDS) and many deaths in 2002-2003 [5,7]. Coronaviruses are RNA-storing viruses, 80-220 nm in size. The outer shell of the virus is covered with villi. Through these villi, the virus attaches to the cell (that's why this family of viruses is called "Coronoviridae" - "coronavirus").
Initially, most of the cases of direct transmission of the disease among people were identified among medical workers who were in contact with patients. The main source of infection is an infected person, and they are dangerous during the latent periods of the disease and when it manifests clinically. In the case of coronavirus disease, the susceptibility to infection is extremely high among all groups of the population, and the risk groups include those over 60 years old, those with chronic (respiratory, cardiovascular, diabetes, oncological diseases), in which the death rate is from 2 to 4%. [2,4,6].
The entrance gate of the pathogen is the epithelium of the upper respiratory tract and epithelial cells of the stomach and intestines. The initial stage of infection is the entry of SARS-CoV-2 into target cells by binding to angiotensin converting enzyme II (ACE2), a functional receptor. ACE2 receptors are found in cells of the respiratory tract, kidneys, esophagus, bladder, small intestine, heart, and central nervous system. However, the main and most frequently contacted target cell is alveolocyte type II (AT2) cells of the lung, which determines the development of pneumonia. The role of CD-147 in invading cells during the invasion of SARS-CoV-2 is also discussed [3, 4].

## Materials and Methods

General and biochemical analysis of blood was conducted mainly in the 16th family clinic of the Almazar district of Tashkent, in the multidisciplinary clinic of the Tashkent Medical Academy. Analyzes were performed on a biochemical analyzer BA-

88A Mindray Co.Ltd (KNR). HUMAN (GmbH) reagents (Germany) were used. The study was conducted in a room with a moderate temperature (26°C) [10]. For the correct organization of bioimpedance analysis, it was ensured that the study participants did not consume liquids and food, did not perform physical activity 2-3 hours before the study. Measurements were taken in the morning, on an empty stomach, as the ideal time for bioimpedance analysis. Tanita BC-730 (TANITA, Japan) was used for bioimpedancemetry[12].

The participation of study participants was voluntary and not funded. The study was conducted in accordance with the rules of scientific ethics, while maintaining the anonymity of the participants [11]. The results were processed using the Excel and OriginPro6.2017 programs (OriginLab Corporation, USA). The results were processed using Fisher's test, Student's t-factor. The arithmetic mean (M), mean deviation (±m), and statistical significance index (p) were determined. At $p \leq 0.05$, the results were considered 95% statistically significant.

The research participants were divided into 4 experimental and 2 control groups in the post-COVID-19 period. For the 1st group, on the basis of voluntary consent, individuals with a severe course of COVID-19, under the age of 40 years, without chronic diseases were selected (n = 25). The 2nd group, on the basis of voluntary consent, were selected with moderate and mild forms of COVID-19, 2-3 months after recovery, not older than 40 years and without chronic diseases (n = 25). The second control group consisted of healthy people aged 41-55 years who were not infected with COVID-19 (n=12). The 3rd group, on the basis of voluntary consent, were selected with a severe course of COVID-19, 2-3 months after recovery, no older than 41-55 years, without chronic diseases (n=25). The 4th group, on the basis of voluntary consent, were selected with moderate and mild forms of COVID-19, 2-3 months after recovery, not older than 41-55 years, without chronic diseases (n = 25).

## Results and Discussion

The content of total cholesterol was 5.01±0.87 mmol/l ($P<0.05$) and 4.12±0.13 mmol/l ($P<0.001$) in the first and second groups during the disease. After the disease 5.32±0.0.21 mmol/l ($P<0.001$) ($P<0.05$) and 3.26±0.34 mmol/l ($P<0.001$). It was found that in those who had a mild form, the indicator decreased to normal, and in those who had a severe form it was 1.6 times higher than in the control.

The corresponding lipoproteins were also analyzed. High-density lipoproteins (HDLC), a useful plastic element for the body, amounted to 1.12±0.018 mmol/l ($P<0.01$) during the period of the disease in the first group and 1.03±0.032 mmol/l ($P<0.01$ ) after illness. It was found that useful lipoproteins decreased to a critical level

by 1.5 times compared with the control. In the second group, the reverse trend was observed, before and after the disease it was 1.31±0.021 mmol/l (P<0.05) and 1.40±0.054 mmol/l (P<0.01). The indicator is below the control level, but is dynamically recovering to a normal level.

Harmful low density lipoproteins - LDL cholesterol in the first and second groups of the disease were 3.58±0.24 mmol/l (P<0.01) and 2.92±0.12 mmol/l (P<0.01), and after the disease - 4.01±0.03 mmol/l (P<0.01) and 3.31±0.04 mmol/l (P<0.01) in the first and second groups, respectively. It was found that it increased by 1.6 and 1.4 times compared with the control.

The basic metabolism was 1860±1.04 kcal (P<0.05) in the 1st group and 1722±0.67 (P<0.05) kcal in the 2nd group with a mild course of the disease. It was found that the basal metabolic rate was 10.85% higher than in the control group, and 3.7% higher than in the second group. It amounted to 1875±0.86 kcal (P<0.05) in the 3rd group and 1780±0.51 (P<0.05) kcal in the fourth group, which had a mild course of the disease. It was found that in the third and fourth groups, the basal metabolic rate was 11% higher than in the control group, and in the second group - by 6.2% (table 1).

Table 3. Analysis of metabolic indicators during recovery (M±m)

| Experiment group | Basic metabolism (Kcal) | Metabolic Deviation Index (%) | Total Metabolism (Kj) |
|---|---|---|---|
| Control 1 (n=12) | 1658±0,84 | 7,5±0,01 | 7142±6,27 |
| 1st group (n = 25) | 1860±1,04* | 17,5±0,05*** | 8735±8,01 |
| 2nd group (n = 25) | 1722±0,67* | 12,5±0,04*** | 7205±7,48 |
| Control 2 (n=12) | 1670±0,52 | 7,5±0,01 | 7232±2,01 |
| 3rd group (n = 25) | 1875±0,86* | 18,5±0,12** | 8230±8,01 |
| 4th group (n = 25) | 1780±0,51* | 12,5±0,65* | 7125±7,48 |

*- P<0,05; **-P<0,01; ***-P<0,001

The main mechanism of this process is that the accumulated lactic acid is partially oxidized with the consumption of additional oxygen. Hypercapnia also stimulates breathing and increases heart rate with an increase in basal metabolic rate. It was analyzed that the level of deviation is high in groups with rapid fatigue and general weakness due to the occurrence of these processes in the recovery period - 18.5±0.12% (P<0.01).

Due to the fact that changes in metabolic parameters, especially lipid and carbohydrate metabolism, require investigation at the level of deeper regulatory mechanisms, polymorphisms in the metabolic marker genes ADRB2 (reacts to glucose, lipolysis, hypertension) and FABP2 (lipid transport) were studied.

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When analyzing the Arg16Gly polymorphism in the ADRB2 gene, in the main group (n=92), which was the COVID-19 recovery group, there were 11 Arg/Arg normal genotypes, 11.97%, Arg/Gly heterozygous genotypes-37, 40.21. %, genotypes mutated by the Gly/Gly polymorphism amounted to 44 47.82% ($\chi 2=3.84$; $P<0.05$, $Df=1$). It was found that there were 3.6 times more persons with Gly/Gly polymorphism mutations than in the control group. ( Table 2).

Table 2 Analysis of the frequency of Arg16Gly polymorphism in the ADRB2 gene between patients and controls

| № | Experiment group | Frequency allele | | | | Frequency genotyping | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|
| | | Arg | | Gly | | Arg / Arg normal | | Arg / Gly heterozygous | | Gly / Gly mutation | |
| | | n | % | n | % | n | % | n | % | n | % |
| 1 | Main group: A group of people who have recovered from COVID-19 (n=92) | 59 | 32,1 | 125 | 67,9 | 11 | 12,0 | 37 | 40,2 | 44 | 47,8 |
| a | post COVID-19 syndrome n=53 | 23 | 21.7 | 83 | 78.3 | 5 | 9,4 | 13 | 24,5 | 35 | 66,0 |
| b | Uncomplicated COVID-19 n=39 | 36 | 46.1 | 42 | 53.8 | 6 | 15,3 | 24 | 61,5 | 9 | 23,0 |
| 2 | Control n=96 | 66 | 35,1 | 122 | 64,9 | 9 | 9,6 | 48 | 51,1 | 39 | 39,4 |

## Conclusions

Entering the cell, the virus destroys the cell nucleus and its genetic apparatus. Cells and organs lose their regulatory abilities and there is a total metabolic disorder known as oxidative stress. Oxidative stress is accompanied by a massive formation of free radicals and metabolites that cannot be neutralized and excreted from the body in a timely manner, thereby forming a toxic environment in the body. This is facilitated by stress and suppression of the body's immune system. The immune system plays not only a protective role, but also performs a number of other vital functions. It is generally accepted that the immune system spends its resources on: fighting external negative agents; restoration of damaged tissues; excretion of metabolic products from the body; maintenance of sanogenic homeostasis; analysis of the experience gained in order to prevent its repetition in the future; replenishment of forces and self-healing, and a number of other processes. Numerous literature sources [1, 8, 9] note the fact that oxidative stress and inflammation potentiated by it form the basis of the

pathogenesis of various chronic diseases that aggravate the course of the COVID-19 coronavirus infection (diabetes mellitus, severe obesity, bronchopulmonary diseases, cardiovascular diseases). , cancer, kidney and liver disease). The cause of oxidative stress is an imbalance in the redox homeostasis system, characterized by excessive production of reactive oxygen species and insufficient activity of the antioxidant defense system. 77% of people admitted to the hospital due to chronic diseases caused by the metabolic syndrome have glutathione deficiency [7]. Among all the potential antioxidants in the body, provides stable conditions for the effective functioning of the antioxidant system, and its deficiency can cause disorders of various organs and systems.

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