

DOI: <http://dx.doi.org/10.21123/bsj.2022.6317>

## Violation of the Hormonal Spectrum in Polycystic Ovaries in Combination with Insulin Resistance. What is the Trigger: Insulin Resistance or Polycystic Ovary Disease?

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Received 27/5/2021, Accepted 30/12/2021, Published Online First 20/3/2022, Published 1/10/2022



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### Abstract:

Polycystic ovary syndrome (PCOS) is the main cause of female infertility. The role of insulin resistance in the development of polycystic ovary is actively discussed here. The study included patients with PCOS without insulin resistance (n = 48) and with insulin resistance (n = 39). The comparison groups were patients with no history of PCOS: a control group without insulin resistance (n = 46) and a group of patients with insulin resistance (n = 45). The following parameters were determined in patients: FSH, LH, TSH, T<sub>3f</sub>, T<sub>4f</sub>, PRL, E<sub>2</sub>, 17-OHd, Pr, AMH, Test total, Test<sub>f</sub>, DHEAS, DHEAS<sub>s</sub>, SHBG, ACTH, cortisol, IRI, IGF-1, C-peptide, and glucose level. The HOMA-IR index and the LH / FSH ratio and the total / SHBG test were calculated. Correlation analysis was also performed between HOMA IR and indicators of the hormonal profile, IGF-1, and C-peptide. Unidirectional changes in the levels of the following hormones were found in insulin resistance, PCOS and / or insulin resistance relative to control values: estradiol, total testosterone, cortisol, prolactin, AMG, and SHBG. As a result of the correlation analysis, negative relationships were established between the HOMA IR Index and the levels of E<sub>2</sub>, cortisol and AMH in patients (except for the control group). We assume that the formation of the phenotype of polycystic ovary with a combination of insulin resistance can be formed in patients with insulin resistance as a result of a decrease in the level of estradiol, SHBG and an increase in the content of total testosterone and AMH.

**Keywords:** Hormonal profile, Infertility, Polycystic ovary syndrome, Resistance of insulin.

### Introduction:

One of the most common endocrine pathologies in women of reproductive age is polycystic ovary disease, which, according to a number of authors, occurs in 2-20% of women<sup>1,2</sup>.

The etiology of PCOS is still not entirely clear. It is a multifactorial endocrine disorder that combines inheritance of gene clusters and environmental factors<sup>3,4</sup>.

Polycystic ovary syndrome (PCOS) is a heterogeneous disease characterized by hyperandrogenism and chronic ovulatory dysfunction with malfunctioning of the vertical hormonal axis of the hypothalamus-pituitary-ovary, as well as other metabolic changes<sup>5-7</sup>. As a result of hormonal imbalance and increased production of

LH, there is an increase in circulating LH levels, which stimulates the synthesis of androgens by the ovarian cortex<sup>8</sup>. The resulting hyperandrogenemia is one of the main culprits in the clinical picture of PCOS<sup>9,10</sup>. Compensatory hyperinsulinemia, in synergy with LH, enhances the stimulation of androgen production. At the same time, a high level of androgens is one of the possible causes of insulin resistance. Androgens, acting directly on the insulin signaling system, may contribute to the development of peripheral insulin resistance in PCOS patients<sup>10</sup>.

Despite the fact that the role of hyperandrogenism in the initiation of PCOS has been identified as the main cause of polycystic

ovary disease, metabolic disorders may also underlie the increase in androgen levels. Therefore, in recent years, the literature has been actively discussing the problem: can insulin resistance determine the development of polycystic ovary disease?

Obesity and IR, associated with subsequent hyperinsulinemia, in adolescence contribute to the activation of excessive production of androgens by the ovaries, which leads to impaired fertility and the development of PCOS<sup>11,12</sup>. IR is thought to be the metabolic precursor of PCOS<sup>13</sup>. In the work of Z. Shaaban (2019)<sup>14</sup> there is a direct indication that hyperandrogenism can be caused by insulin resistance.

However, there is another opinion. So, according to the results of the meta-analysis<sup>15</sup>, about 60% of women with obesity and insulin resistance do not suffer from hyperandrogenism and PCOS. This meta-analysis has a number of significant limitations - insufficient sample of surveyed women with insulin resistance and / or PCOS. In addition, it did not take into account a number of hormones that are involved in the development of PCOS: follicle-stimulating and luteinizing hormones, prolactin, thyroid hormones, anti-Müllerian hormone, etc.<sup>16-20</sup>.

The aim of this study is to test the hypothesis of whether insulin resistance is a trigger for polycystic ovary disease. In this regard, the hormonal spectrum in patients with polycystic ovary disease and / or insulin resistance was analyzed in comparison with the hormonal status of patients with insulin resistance and no history of polycystic ovary disease.

## Methods:

A prospective case-control study was conducted at the Center for Human Reproduction and IVF (Rostov-on-Don) from 2018 to 2020. In accordance with the Helsinki Declaration of the World Medical Association "Ethical Principles for Scientific Medical Research with Human Participation" (as amended in 2000), as well as the "Rules of Clinical Practice in the Russian Federation", all studies were carried out with the informed consent of patients signed under agreements for the provision of medical care for infertility with the use of ART programs and the use of the results of biomaterial research for scientific purposes.

The study included women who were diagnosed with PCOS (n = 87) based on the diagnostic criteria of the 2003 ASRM (American Society for Reproductive Medicine) / ESHRE (European Society for Human Reproduction and

Embryology) Rotterdam agreement. Patients were diagnosed with PCOS if two of the following three criteria were confirmed: a) anovulation or oligomenorrhea; b) clinical or biological evidence of hyperandrogenism; c) the presence of polycystic ovaries on ultrasound<sup>21</sup>. The final diagnostic criteria for PCOS when using transvaginal ultrasound<sup>22</sup>. This group included patients without insulin resistance (PCOS, n = 48) and with insulin resistance (PCOS + IR, n = 39).

The comparison groups were patients with no history of PCOS: a control group without signs of insulin resistance (control, n = 46) and a group of patients with insulin resistance (IR, n = 45).

Patients with diseases such as Cushing's disease, hypothyroidism, hyperprolactinemia, adrenal hyperplasia, or ovarian tumors were excluded from the study.

The patients that included in the examination, the analysis of the hormonal profile was checked for the following indicators: serum levels of FSH, LH, TSH, T<sub>4</sub>, cortisol, E<sub>2</sub>, Pr, AMH, and IRI by the Beckman Coulter test systems using the Access 2 automatic analyzer. PRL levels, Total test, DHEAS<sub>s</sub>, and SHBG was assessed using the Alkor-Bio test systems on an Alisey QS automatic immunochemical analyzer. This device was also used to quantify ACTH (Biomeric test system), Test<sub>f</sub> and 17-OHd (DRG test system), DHEAS (DBC test system), IGF-1 (Mediagnost kit), and C-peptide (Vector Best test system). The analysis of T<sub>3</sub><sub>f</sub> was performed on a Cobas 6000 electrochemiluminescence immunoanalyzer with a Roche Diagnostics reagent kit. All biochemical parameters were determined in blood serum taken in the morning on an empty stomach after 12-14 hours of fasting. Fasting blood plasma glucose was determined using a LabSystem analyzer (Finland) and reagents from Biocon (Germany).

The HOMA-IR index was calculated using the formula:  $HOMA-IR = \text{fasting insulin } (\mu\text{U} / \text{ml}) \times \text{fasting glucose } (\text{mmol} / \text{l}) / 22.5$ . The values of the LH / FSH ratios and the total / SHBG test were also evaluated.

Statistical data processing was performed using the statistical 10.10 software package. Since the distribution of the calculated data was nonparametric, they were presented as a median and interquartile range (Me; 25-75%). Intergroup differences were assessed using the Mann-Whitney test. To establish the relationship between the level of immunoreactive insulin and the analyzed parameters, Spearman's rank correlation coefficient (r) was used. Differences were considered significant at a significance level of p < 0.05.

**Results:**

When distributing patients into groups, the value of the HOMA IR Index was taken into account, the indicators of which, as well as the level

of glucose, insulin, IGF-1 and C-peptide in the blood serum of the examined patients are presented in Table 1.

**Table 1. Levels of immunoreactive insulin (IRI), glucose, IGF-1 and C-peptide and HOMA IR index in the examined patients**

Groups / indicators	control	IR	PCOS	PCOS +IR
IRI level, $\mu$ U / ml, p level	6.41 (5.91-6.89)	14.34 (13.56-15.27) ****	8.87 (7.73-10.06) ***	11.32 (10.74-12.94) ***
Glucose level, mmol / l, p level	4.62 (4.28-4.73)	5.89 (5.62-6.13) ***	5.34 (5.14-5.55) ***	6.41 (6.19-6.67) ****
IGF-1, p level	136.58-184.44	205.46 (187.04-219.63) **	190.34 (165.22-207.85) *	216.58 (188.63-239.31) ****
C-peptide, p level	115.73-140.78	158.20 (145.39-172.64) **	51.05 (47.82-54.05) ***	163.37 (158.94-168.29) **
HOMA IR index, p level	1.19 (0.96-1.34)	3.75 (3.29-4.18) ****	2.12 (1.84-2.37) **	3.22 (3.17-3.49) ****

Note: the levels of reliability of the difference in indicators relative to the values in the control group \* -  $p < 0.05$ ; \*\* -  $p < 0.01$ ; \*\*\* -  $p < 0.001$ ; \*\*\*\* -  $p < 0.0001$

The analysis of the hormonal profile showed that the levels of most analyzed parameters in the groups of patients with PCOS and insulin resistance, including patients with combined pathology, differed from the control values. However, 20% of these indicators were within the reference values. All groups of patients with PCOS and / or insulin resistance were characterized by a significant decrease in the level of E2, PRL,  $T3_f$ , cortisol, and SHBG against the background of an increase in the levels of Test total and AMG. In contrast to the group with IR, the patients with PCOS, as well as PCOS + IR, had an increase in the levels of LH, ACTH, and  $Test_f$  compared to the control group. In insulin resistance and PCOS, unidirectional changes ( $p < 0.05$ ) in the levels of E2, prolactin,  $T3_f$ , cortisol, Test total, SHBG, DHEAS, AMG, and IGF-1 were established relative to the control (Table 2).

In the group with PCOS and, especially, PCOS + IR, relative to the control values, the FSH level was decreased ( $p < 0.05$ ), while the LH level, on the contrary, was increased ( $p < 0.05$ ). In patients with insulin resistance, these indicators were within the control values (Table 2).

When studying the role of insulin resistance in the development of polycystic ovary syndrome, in addition to the absolute values of the studied parameters, the LH / FSH ratios and the total / SHBG test were analyzed, an increase in which is characteristic of hyperandrogenism and PCOS<sup>15,23</sup>. A significant increase in the LH / FSH ratio was

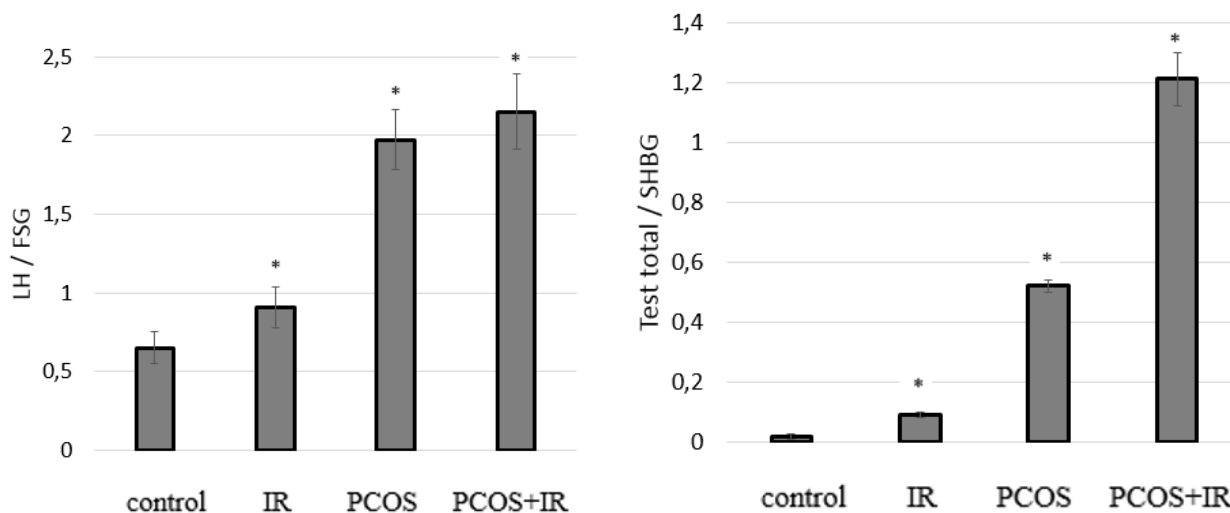
shown in the groups with PCOS and PCOS + IR relative to control ( $p < 0.001$ ), as well as in insulin resistance, although to a lesser extent ( $p < 0.05$ ).

In patients with PCOS and / or insulin resistance (especially in the group of patients with PCOS + IR), the Test total / SHBG ratio was significantly increased relative to the control values (Fig.1).

As a result of the study, in the control group of patients, correlations were revealed between the HOMA IR index and  $T3_f$  ( $r = 0.62$ ;  $p < 0.001$ ) and progesterone ( $r = 0.34$ ;  $p < 0.01$ ) (Table 3).

The largest number of correlations between the HOMA IR index and the studied parameters was found in the group of patients with insulin resistance. In particular, correlations were revealed between the HOMA IR index and the levels of FSH ( $r = -0.38$ ;  $p < 0.01$ ), prolactin ( $r = -0.46$ ;  $p < 0.01$ ), TSH ( $r = 0.50$ ;  $p < 0.01$ ),  $T3_f$  ( $r = -0.47$ ;  $p < 0.01$ ), cortisol ( $r = -0.62$ ;  $p < 0.01$ ), ACTH ( $r = 0.56$ ;  $p < 0.01$ ), E2 ( $r = -0.84$ ;  $p < 0.01$ ), progesterone ( $r = 0.35$ ;  $p < 0.05$ ),  $Test_f$  ( $r = -0.32$ ;  $p < 0.05$ ), 17-OH ( $r = -0.42$ ;  $p < 0.01$ ), AMG ( $r = -0.66$ ;  $p < 0.01$ ), IGF-1 ( $r = 0.49$ ;  $p < 0.01$ ), and C-peptide ( $r = 0.69$ ;  $p < 0.01$ ) (Table 3).

In the group of patients with PCOS, a close relationship was shown between the HOMA IR index and the levels of PRL ( $r = 0.57$ ;  $p < 0.01$ ),  $T3_f$  ( $r = -0.68$ ;  $p < 0.01$ ), cortisol ( $r = -0.51$ ;  $p < 0.01$ ), E2 ( $r = -0.69$ ;  $p < 0.01$ ), AMG ( $r = -0.79$ ;  $p < 0.01$ ), IGF-1 ( $r = -0.66$ ;  $p < 0.01$ ), and C-peptide ( $r = -0.86$ ;  $p < 0.01$ ) (Table 3).



**Figure 1. LH / FSH ratios and Test total / SHBG test in the examined patients**

\* - significant differences in indicators relative to values in the control group (at  $p < 0.05$ )

**Table 2 . Indicators of hormonal status and the level of SHBG in the serum of the examined patients**


Groups / indicators	control	IR	PCOS	PCOS +IR
FSH	6.98 (5.54-7.11)	6.42 (5.94-7.23)	5.89 (5.12-6.76)	5.42 (4.73-6.19) *
LH	5.45 (4.94-6.11)	5.71 (5.10-6.49)	11.87 (10.92-12.23) ***	12.58 (11.35-13.17) ***
E2	171.73 (159.23-179.65)	70.06 (64.81-74.32) ***	78.31 (71.54-85.68) ***	72.81 (65.22-78.91) ***
Pr	4.30 (3.86-4.92)	0.85 (0.74-0.91) ***	4.51 (4.15-5.06)	3.05 (2.71-3.22) ***
17-OH	2.41 (1.97-2.69)	1.68 (1.41-2.07) *	2.64 (2.29-2.95)	2.86 (2.61-3.11) *
PRL	342.64 (313.06-368.49)	224.87 (213.15-251.77) ****	291.37 (262.83-318.04) **	246.39 (214.08-254.33) ***
TSH	1.61 (1.53-1.77)	1.57 (1.26-1.71)	1.73 (1.49-1.83)	1.31 (1.02-1.44) ***
T3 <sub>f</sub>	4.97 (4.38-5.46)	2.44 (2.21-2.58) ***	3.74 (3.34-3.96) *	3.42 (3.12-3.56) ***
T4 <sub>f</sub>	13.12 (12.53-14.69)	10.01 (8.94-11.07) ***	12.41 (11.06-13.45)	11.35 (10.20-11.96) ***
Cortisol	275.31 (246.82-274.96)	184.58 (147.88-205.75) ***	248.82 (213.75-277.16) **	227.20 (179.28-243.39) ***
ACTH	22.27 (19.35-24.06)	22.24 (18.95-24.68)	26.25 (23.99-30.81) ***	27.94 (24.17-29.59) ***
Test total	1.69 (1.46-1.92)	4.23 (3.89-4.51) ****	4.39 (4.03-4.58) ****	3.74 (3.32-3.92) ****
Test <sub>f</sub>	1.17 (0.98-1.37)	0.95 (0.89-1.21)	1.48 (1.31-1.64)*	1.37 (1.29-1.56)*
SHBG	72.18 (67.02-78.31)	47.44 (43.64-50.65) ***	8.42 (8.11-9.15) ****	17.53 (15.89-18.23) ****
DHEAS	9.56 (8.83-10.04)	5.18 (4.79-5.36) ***	9.11 (8.37-10.25)	11.15 (10.21-12.19) *
DHEAS <sub>s</sub>	103.75 (75.68-119.51)	49.09 (46.69-52.88) ***	89.81 (84.39-104.72) *	98.76 (94.05-114.74)
AMG	2.39 (2.08-2.74)	4.08 (3.52-4.96) ****	8.27 (7.15-9.49) ****	11.58 (9.66-13.75) ****

Note: see table 1.

In patients with PCOS + IR, close correlations were found between the HOMA IR index and the levels of E2 ( $r = -0.89$ ;  $p < 0.01$ ), prolactin ( $r = -0.48$ ;  $p < 0.05$ ), cortisol ( $r = -0.72$ ;  $p < 0.01$ ), Test total ( $r = 0.48$ ;  $p < 0.05$ ), AMG ( $r = -0.84$ ;  $p < 0.01$ ), IGF-1 ( $r = 0.66$ ;  $p < 0.01$ ), and C-peptide ( $r = 0.86$ ;  $p < 0.01$ ) (Table 3).

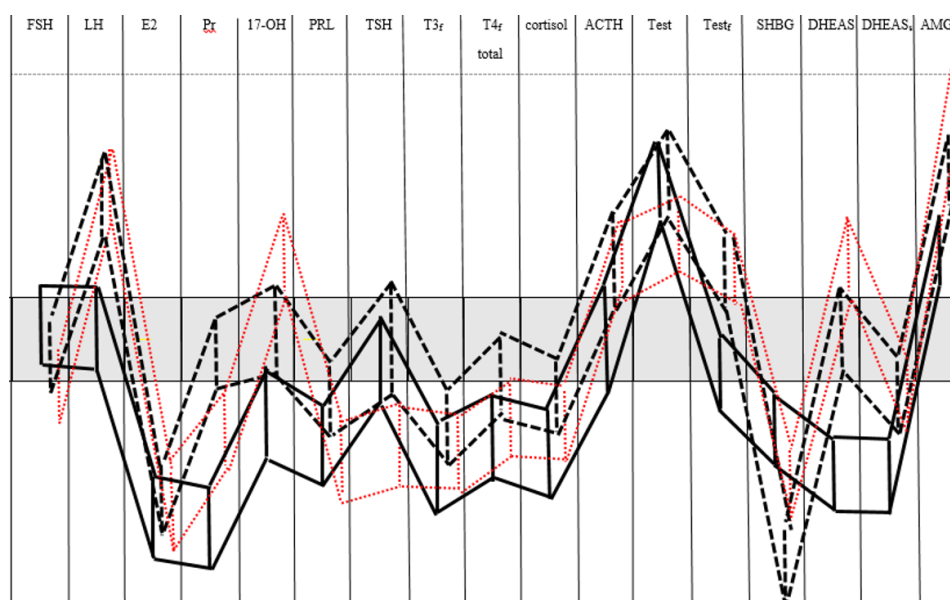
**Table 3. Correlation links ("+" - positive, "-" - negative) between the HOMA IR index and indicators of the hormonal profile, IGF-1 and C-peptide**

Indicators	control	IR	PCOS	PCOS +IR
FSH		-		
LH				
E2		-	-	-
Pr	+	+		
17-OH		-		
PRL			+	-
TSH		+		
T3 <sub>f</sub>	+	-	-	
T4 <sub>f</sub>				
Cortisol		-	-	-
ACTH		+		
Test total				+
Test <sub>f</sub>				
SHBG				
DHEAS				
DHEAS <sub>s</sub>				
AMG		-	-	-





Note:  - no connection

Thus, the revealed correlations between the HOMA IR index and the studied parameters in PCOS and / or insulin resistance were not typical for the control group of patients. At the same time, differences were also established between groups of patients with PCOS and / or insulin resistance. In polycystic ovary, in contrast to the group with insulin resistance, no correlations were found between the HOMA IR index and the levels of FSH, TSH, ACTH, progesterone, Test<sub>f</sub>, and 17-OH. At the same time, in the group of patients with PCOS and insulin resistance, a relationship was established between the HOMA IR index and the level of Test total, in contrast to the group with PCOS and patients with IR (Table 3).

The general scheme of hormonal imbalance in PCOS and / or insulin resistance is shown in Fig.2



**Figure 2. Scheme of hormonal imbalance in PCOS and / or insulin resistance**

-  - patients with insulin resistance
-  - PCOS patients
-  - patients with PCOS and insulin resistance
-  - level in patients without PCOS and IR

Based on the results obtained, it can be assumed that insulin resistance is a risk factor for polycystic ovary. Insulin resistance is characterized by unidirectional changes in the level of a number of hormones that play a key role in the development of polycystic ovaries (E2, Test total, SHBG, and AMG). In addition, in PCOS and / or insulin resistance, negative correlations with the levels of E2, corticosterone and AMH were revealed, which probably underlies a single mechanism in the development of pathological processes in these nosologies.

### Discussion:

The most common cause of infertility in women is polycystic ovary disease<sup>24</sup>. PCOS is the subject of ongoing research, since the etiology of this syndrome has not yet been finally determined: there is an idea of a violation of the interaction of genetic, behavioral and environmental factors in PCOS<sup>9,25</sup>.

This study tested the hypothesis of the role of insulin resistance as a trigger for polycystic ovary disease. For this purpose, an analysis of the hormonal status of patients with PCOS with or without insulin resistance, as well as patients with insulin resistance not aggravated by polycystic ovaries, was carried out. The results were obtained according to which in women with PCOS there is an increase in the levels of LH, Test total, Test sv, ACTH and AMH against the background of a decrease in E2, PRL, cortisol, SHBG and DHEAS. Against the background of insulin resistance in PCOS, an increase in the levels of LH, ACTH and AMH was also observed, as well as 17-OH, Test total, Test<sub>t</sub>, DHEA, but in addition, a decrease in the levels of FSH, E2, progesterone, TSH, T3sv, T4sv, cortisol and SHBG ... That is, with a combination of PCOS and insulin resistance, the spectrum of pathological changes in hormonal status increases. In patients with insulin resistance and without PCOS, only the levels of FSH, LH and Test<sub>t</sub> were within the normal range, all other studied indicators of hormonal status differ from the control values. At the same time, in contrast to the PCOS and PCOS + IR groups, patients with insulin-resistivity not burdened by PCOS have a decrease in the level of progesterone compared to the control values.

An important result of the study is the established unidirectional changes in the levels of the following hormones in insulin resistance, PCOS and / or insulin resistance relative to control values: estradiol, total testosterone, cortisol, prolactin, anti-Müllerian hormone, and SHBG. In addition, as a result of the correlation analysis, negative associations were established between the HOMA

IR Index and the levels of E2, cortisol and AMH in patients (except for the control group).

It is known that imbalance in steroid hormones can lead to PCOS. In the ovary, androgens are produced by theca cells and mesenchymal cells, while estrogen is produced by granulosa cells. Androgen is converted to E2 in granulosa cells, while cytochrome P450 aromatase (P450arom), which is a product of cytochrome P450 family 19 (CYP19), limits this process. In turn, when the activity of P450arom is inhibited or the expression of CYP19 is disrupted, the conversion of androgen to estrogen is inhibited as a result<sup>26</sup>. It has been shown that CYP19 can act as a genetic factor in the development of the hyperandrogenic phenotype of PCOS<sup>27</sup>. In addition, it has been shown that the expression of the estrogen and aromatase receptor is reduced in granulosa cells of patients with PCOS<sup>28</sup>. Also, in PCOS, the production of inflammatory factors is increased<sup>29</sup>, which, in turn, is reflected in an increase in the concentration of androgens in PCOS<sup>30</sup>.

The revealed decrease in the levels of prolactin, cortisol in insulin resistance and / or polycystic disease is not supported by the literature data. We can assume that this fact is associated with the individual characteristics of the metabolism of the examined patients. Nevertheless, during the correlation analysis it was found that the HOMA IR index negatively correlated with the indicators of prolactin (except for the PCOS group) and cortisol in the examined patients; in the control group of women, these correlations were not established.

Also, negative correlations were revealed between the HOMA IR index and the AMH level in all patients, except for the control group. Therefore, it can be assumed that an increase in AMH content in insulin resistance may also be a trigger mechanism for triggering polycystic ovary disease. This is confirmed by the results of other studies aimed at identifying the role of AMH in PCOS<sup>31-32</sup>. Also, recent studies have focused on the relationship between AMH and insulin resistance, which may be of clinical importance, since it has been revealed that insulin and AMH affect steroidogenesis and folliculogenesis.<sup>33,34</sup> However, there are also different opinions on this issue. In a study by Park et al. women without PCOS showed a negative correlation between AMH and HOMA-IR.<sup>35</sup>, while Bleil et al. This connection has not been established<sup>36</sup>. There are several more works with conflicting results on this issue<sup>37,38</sup>.

The negative correlation between SHBG and the HOMA IR index in this study is also confirmed by the literature data. Interest in SHBG, which is a globulin regulating the bioavailability of

sex hormones, has grown in recent years due to its inverse with polycystic ovary and insulin resistance<sup>39, 40</sup>.

The question of a decrease in the levels of cortisol, prolactin as predictors of polycystic disease in patients with insulin resistance requires additional research.

### Conclusion:

Thus, according to the results of this study, it can be assumed that the formation of the phenotype of polycystic ovary with a combination of insulin resistance occurs in patients with insulin resistance, in whom there is a decrease in the level of estradiol, SHBG and an increase in the content of total testosterone and anti-Müllerian hormone. These indicators are recommended for inclusion in the protocol for the diagnosis of polycystic ovary disease in patients with insulin resistance.

### Authors' declaration:

- Conflicts of Interest: None.
- We hereby confirm that all the Figures and Tables in the manuscript are mine ours. Besides, the Figures and images, which are not mine ours, have been given the permission for re-publication attached with the manuscript.
- Authors sign on ethical consideration's approval.
- Ethical Clearance: The project was approved by the local ethical committee in Southern Federal University.

This study was funded by the Ministry of Science and Higher Education of the Russian Federation № 0852-2020-0028.

### Authors' contributions statement:

Research concept and design: T.P. S., interpretation of research results – K.G.V. Generalization of results and formulation of conclusions – L.S.V. Writing an article – L. S.V., K.G.V. Collection and istematization of data from clinical trials – B. E.S., Z O.S. Analysis and synthesis of literature data - A.A. A.

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- Abbreviation:**  
 17-OHd - 17-hydroxyprogesterone  
 ACTH - adrenocorticotropic hormone  
 AMH - anti-Müllerian hormone  
 DHEAS - dehydroepiandrosterone  
 DHEASs - dehydroepiandrosterone sulfate  
 E2 - estradiol  
 FSH - follicle-stimulating hormone  
 IGF-1 - insulin-like growth factor-1  
 IRI - immunoreactive insulin  
 LH - luteinizing hormone  
 Pr - progesterone  
 PRL - prolactin levels  
 SHBG - sex hormone binding globulin  
 T<sub>4f</sub> - free thyroxine  
 Test<sub>f</sub> - free testosterone  
 Total test - total testosterone  
 TSH - thyroid-stimulating hormone

## انتهاك الطيف الهرموني في المبايض متعددة الكيسات مع مقاومة الأنسولين. ما هو المحفز: مقاومة الأنسولين أم مرض البويضات متعدد الكيسات؟

زولوتيكاس و س<sup>1</sup>

بوجريموفا ي س<sup>1</sup>  
 كارانتش ج ف<sup>2</sup>

شكورات ت ب<sup>2</sup>  
 الكسندروف أ<sup>2</sup>

لومتيفاس ف<sup>1</sup>

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### الخلاصة:

متلازمة المبيض متعدد الكيسات هي السبب الرئيس للعقم لدى النساء. حيث يناقش البحث دور مقاومة الأنسولين في تطور تكيس المبايض. تم تحليل الطيف الهرموني للمرضى الذين يعانون من متلازمة تكيس المبايض و / أو مقاومة الأنسولين. اشتملت الدراسة على مرضى يعانون من متلازمة تكيس المبايض دون مقاومة الأنسولين (عددهم = 48) ومقاومة الأنسولين (عددهم = 39). كانت مجموعات المقارنة من المرضى الذين ليس لديهم تاريخ من متلازمة تكيس المبايض: مجموعة تحكم بدون مقاومة الأنسولين (عددهم = 46) ومجموعة من المرضى الذين يعانون من مقاومة الأنسولين (عددهم = 45). تم تحديد المعلمات التالية في المرضى FSH: ، LH ، TSH ، T<sub>4f</sub> ، T<sub>3f</sub> ، PRL ، E2 ، 17-OHd ، Pr ، AMH ، مجموع الاختبار ، Test<sub>f</sub> ، DHEAS ، DHEAS ، SHBG ، ACTH ، الكورتيزول ، IRI ، IGF-1 ، C-الببتيد ، ومستوى الجلوكوز. تم حساب مؤشر HOMA-IR ونسبة LH / FSH واختبار إجمالي SHBG. تم إجراء تحليل الارتباط أيضًا بين HOMA IR ومؤشرات المظهر الهرموني و IGF-1. تم العثور على تغيرات أحادية الاتجاه في مستويات الهرمونات التالية في مقاومة الأنسولين و / أو متلازمة تكيس المبايض و / أو مقاومة الأنسولين بالنسبة لقيم التحكم: استراديول ، هرمون التستوستيرون الكلي ، الكورتيزول ، البرولاكتين ، AMG ، و SHBG نتيجة لتحليل الارتباط ، تم إنشاء علاقات سلبية بين مؤشر HOMA IR ومستويات E2 والكورتيزول و AMH في المرضى (باستثناء المجموعة الضابطة) تفترض الدراسة أن تكوين النمط الظاهري للمبيض المتعدد الكيسات مع مزيج من مقاومة الأنسولين يمكن أن يتشكل في المرضى الذين يعانون من مقاومة الأنسولين نتيجة لانخفاض مستوى الاستراديول ، SHBG وزيادة محتوى التستوستيرون الكلي و AMH.

الكلمات المفتاحية: المظهر الهرموني، العقم، متلازمة تكيس المبايض، مقاومة الأنسولين