

INTERNAL DISEASES

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**Factor analysis of leptin, adiponectin effect
and resistin during bronchial asthma***T. M. Lalaeva, V. N. Mineev, V. I. Trofimov*Pavlov First St. Petersburg State Medical University,
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To date, the participation of particular adipokines (leptin, adiponectin, resistin) in the pathogenesis of bronchial asthma has been well studied, however, their interaction, in particular their cooperative effect on the course of the disease, has not been practically studied. The aim of the study is to identify the effect of the cooperative interaction of key adipokines on the course of bronchial asthma, depending on the pathogenetic variant of the disease. Factor analysis was used to analyze the data from the study of the levels of leptin, adiponectin, resistin in 56 patients with bronchial asthma. Factor analysis made it possible to identify 3 factors. Factor 1 reflects the direct effect of leptin signaling on the severity of bronchial asthma, to a lesser extent on the exacerbation of the disease. In this case, the adiponectin component, with a high negative factor load, is associated with the severity of the course and the phase of bronchial asthma. Factor 2 reflects the direct effect of another adipokine resistin on the severity of bronchial asthma, but we draw attention to the fact that the resistin component with a large positive factor loading is associated with a non-allergic variant of bronchial asthma and exacerbation of the disease. Factor analysis revealed a fundamentally different pathogenetic role in relation to the severity of bronchial asthma of such adipokines as leptin and resistin in different variants of the disease.

Keywords: leptin, adiponectin, resistin, bronchial asthma, severity of the course, factor analysis.

To date, the participation of adipokines in the pathogenesis of bronchial asthma has been well studied, and this concerns the participation of individual specific adipokines (leptin [1], adiponectin [2], resistin [3]). Nevertheless, in the body, both a healthy person

and a patient with bronchial asthma, there is undoubtedly a complex interaction (a kind of complex microset of signaling molecules) of key adipokines, which ultimately determines the vector of normal or pathological regulatory changes.

We have previously made an attempt to develop an analytical approach to identifying the nature of the interaction of a number of adipokines using integral indicators in an allergic variant of bronchial asthma [4]. The idea of [4] a certain specialization of the effects of adipokines in allergic bronchial asthma was put forward: apelin signaling is mainly involved in the regulation of bronchial resistance, and adiponectin signaling is involved in the regulation of the activity of inflammation cells.

As the **goal** of this article, the task was formulated to identify the influence in the cooperative interaction of key adipokines on the course of bronchial asthma, depending on the pathogenetic variant of the disease.

For this purpose, the well-known method of mathematical analysis — factor analysis — was used. As is well known, factor analysis helps to find hidden, but objectively existing regularity of the investigated process.

Materials and methods

56 patients with bronchial asthma were examined. Diagnosis was established according to criteria and standards of international consensus on AD diagnosis and treatment (GINA, 2012).

All examined patients of BA were in the hospital therapy clinic named after M. V. Cernorutsky of the Pavlov First St. Petersburg State Medical University.

The database is provided by associate professor, Doctor of Medical Sciences T. M. La-laeva, the results of studies of adipokine levels are presented in the dissertation work for the degree of Doctor of Medical Sciences [5].

Leptin, adiponectin, resistin, as well as the soluble plasma leptin receptor were determined by the enzyme immunosorbent method (ELISA) using a standard sandwich-based protocol. Reagent kits “Leptin ELISA” (DRG Diagnostics, Germany), “Adiponectin ELISA” (DRG Diagnostics, Germany), “Human Resistin ELISA” (DRG Diagnostics, Germany) were used. The analysis of adipokines was carried out on ELISA immunoanalyzer Uniplan-M version 1.04 with a wavelength of 450 nm.

Statistical analysis of research results was carried out using the SPSS program for Windows (Statistical Package for the Social Science) — (Russified version 21.0).

Results and discussion

The factor analysis includes (Table) both values of leptin levels, leptin receptor expression, adiponectin, resistin and body mass index, reflecting to a certain extent the content of adipose tissue, and key characteristics of the course of bronchial asthma (pathogenetic variant of the disease, severity of the course, phase of the disease).

It should be emphasized that through the factor analysis, the expediency of using the factor model was checked: Bartlett's sphericity criterion ($p < 0.0002$); the adequacy test of the Kaiser-Meyer-Olkin sample was 0.604, which indicates the acceptable adequacy of the factor analysis we applied.

As can be seen from Table, 3 factors were identified by factor analysis.

Table. Results of factor analysis in bronchial asthma

Factor 1, dispersion 27.3%		Factor 2, dispersion 17.5%		Factor 3, dispersion 13.8%	
Leptin receptor (ng/ml)	-0.787	BA variant 1 — allergic, 2 — nonallergic	0.739	BA phase 1 — remission, 2 — exacerbation	0.869
Leptin (ng/ml)	0.756	Resistin (ng/ml)	0.532	Severity of BA course 1 — mild, 2 — moderate, 3 — heavy	-0.566
BMI	0.728	Severity of BA course 1 — mild, 2 — moderate, 3 — heavy	0.466	Leptin (ng/ml)	-0.132
Adiponectin (µg/ml)	-0.517	Adiponectin (µg/ml)	0.423	Leptin receptor (ng/ml)	-0.112
Severity of BA course 1 — mild, 2 — moderate, 3 — heavy	0.295	BMI	0.334	Resistin (ng/ml)	0.102
BA variant 1 — allergic, 2 — nonallergic	-0.207	BA phase 1 — remission, 2 — exacerbation	0.187	BMI	0.093
BA phase 1 — remission, 2 — exacerbation	0.194	Leptin (ng/ml)	-0.128	Adiponectin (µg/ml)	-0.073
Resistin (ng/ml)	0.106	Leptin receptor (ng/ml)	0.015	BA variant 1 — allergic, 2 — nonallergic	-0.049

Factor 1 reflects the direct effect of leptin signaling on the severity of the course of bronchial asthma, to a lesser extent on the exacerbation of the disease. The adiponectin component with high negative factor load is associated with flow severity and phase of bronchial asthma.

We emphasize that the components of leptin signaling with a high factor load (Factor 1) are associated with an allergic variant of the disease.

In contrast, Factor 2 reflects the direct effect of another adipokine resistin on the severity of bronchial asthma course, but note that a resistin component with a large positive factor load is associated with a nonallergic variant of bronchial asthma and exacerbation of the disease.

It is very interesting that the adiponectin component as well as the resistin component in Factor 2 directly correlates with the severity of bronchial asthma, and with the variant of bronchial asthma of nonallergic genesis.

Factor 3, which characterizes bronchial asthma as such (severity of course, phasicity), does not provide additional significant information to solve our goal, emphasizing only the fact that the severity of course and phasicity of the disease are influenced by other causes not related to adipokines and adipose tissue.

Thus, factor analysis revealed an apparently fundamentally different pathogenetic role in relation to the severity of the course of bronchial asthma of adipokines such as leptin and resistin in various disease variants.

Undoubtedly, in the human body, these adipokines as hormones of adipose tissue, like all other hormones, interact with each other at the level of membrane receptors, the form of interaction can be different (synergism, antagonism, permissive action).

Unfortunately, there are no direct studies regarding the problem of the interaction of adipokines, although, based on the already known mechanisms of individual adipokines, it is possible to assume a certain nature of such interaction. So, in our study, the antagonistic effect of leptin and adiponectin in relation to such an important characteristic of bronchial asthma as the severity of the course is very clearly revealed.

It is also important that if, in an allergic variant of bronchial asthma, leptin and resistin have some synergy with respect to the formation of the severity of the course of the disease, then in a nonallergic version, these adipokines are somewhat antagonistic in effect on the severity of the course.

Further research work remains to be done on the accumulation of data explaining these similarities and differences in these influences.

Our factorial analysis of these studies of adipokines in bronchial asthma — an analysis that, as already noted, helps to find hidden but objectively existing regularity of the studied process in complex microsets, allows us to once again confirm our idea of specializing the effects of adipokines, despite their general direction of their effects in different variants of bronchial asthma.

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