Psychiatr. Pol. 2023; 57(1): 51–64
PL ISSN 0033-2674 (PRINT), ISSN 2391-5854 (ONLINE)
www.psychiatriapolska.pl
DOI: https://doi.org/10.12740/PP/146196

Risk factors for weight gain in patients with first-episode psychosis

Bogna Batko[1], Marta Szwajca[1], Natalia Śmierciak[1], Wirginia Krzyściak[2], Aleksander Turek[1], Maciej Pilecki[1]

[1] Department of Child and Adolescent Psychiatry, Chair of Psychiatry, Faculty of Medicine, Jagiellonian University Medical College

[2] Department of Medical Diagnostics, Faculty of Pharmacy, Jagiellonian University Medical College

Summary

Aim. Assessment of the association between weight gain in patients with first-episode psychosis (FEP) and biopsychosocial and sociological factors.

Method. 25 subjects with FEP aged 14–35 examined in week 1 (P1) and after three months of hospitalization (P3) were enrolled in the study. Within 3 months all patients were diagnosed with schizophrenia. The study used: a socio-demographic survey, *Positive and Negative Syndrome Scale* (PANSS), *State-Trait Anxiety Inventory* (STAI), *Coping Inventory for Stressful Situations* (CISS), *Questionnaire Eating Behaviors* (QEB), and routine biochemical test findings. For some variables, the differences (variable_D) between the values at P1 and P3 were calculated.

Results. Statistically significant correlations were shown between body weight_P1, _P3, _D, and healthy diet index_P1, _P3, severity of psychotic symptoms measured by the PANSS_P1 and _D, the CISS focused on emotions and task_P1, _P3 and _D, mother's body weight in youth and now, father's body weight in youth and now, and the number of the patient's siblings. In the linear regression analysis, body weight_P1 and the CISS focused on emotions_P1 turned out to be significant predictors of body weight_P3.

Conclusions. Multifactorial influence of weight gain in FEP in schizophrenia was observed. Countermeasures against weight gain should refer not only to the diet, but also to the way the eating habits are related to psychopathology associated with psychosis and to the emotional functioning of the patient.

Key words: FEP, schizophrenia, weight gain

Introduction

Schizophrenia is a chronic illness which impairs social functioning in everyday life, whose etiology remains to be unknown. Aside from typical psychopathological symptoms, 35–70% of patients also develop concurrent somatic diseases [1]. Obesity in subjects with schizophrenia occurs in 45–55% of patients, while they most frequently develop abdominal obesity (up to 60%), associated with reduced insulin sensitivity [2]. Accumulated adipose tissue increases the risk of diabetes mellitus type 2, dyslipidemia, hypertension, insulin resistance, and, as a result, cardiovascular diseases [3, 4].

The use of antipsychotic drugs is one of the main risk factors for weight gain in schizophrenia. Even patients with normal values of body mass index (BMI) show higher values of visceral adipose tissue and lower values of lean body mass, as compared with healthy controls with similar values of subcutaneous adipose tissue [5]. Early metabolic disorders may precede weight gain, even if the latter remains to be related to the use of atypical neuroleptics. The highest weight gain is observed within the first months of treatment with neuroleptics, and the latest meta-analyses show that in the period of 3–12 weeks, body weight of the patients increased on average by ≥ 7% versus placebo [6].

However, Ryan et al., in their studies, pointed to a 3-fold higher content of visceral adipose tissue in subjects with first-episode psychosis (FEP), without introduction of antipsychotic drugs, as compared to healthy subjects with corresponding BMI values [7]. Although the mechanism is still unknown, studies suggest that people with schizophrenia have a tendency to accumulate visceral fat, which may result from higher cortisol and insulin values in subjects with FEP, even before introducing neuroleptics which, together with improper diet, may potentiate central obesity [5, 8]. Antipsychotic-naive patients with FEP already reveal metabolic disorders which are not associated with lipid parameters [9]. They also show impaired glucose tolerance and increased insulin resistance [10, 11].

The occurrence of schizophrenia does not overrule other risk factors for developing obesity in the developmental period, such as genetic, environmental (prenatal and perinatal factors, parenting styles, socio-economic status, bad childhood experience, time spent in front of the screen of devices, physical activity, sleep, diet), or psychosocial factors [12–16].

Although there are studies relating individual factors to weight gain in patients with FEP, there are no such studies, however, which would examine at a wide angle the effect of biological, environmental and socio-demographic factors together.

Material

Patients aged 14 to 35 years admitted to the In-Patient Unit for Adults and In-Patient Unit for Adolescents at the Department of Child and Adolescent Psychiatry of the University Hospital in Krakow between January 2017 and December 2019 were qualified for the study. A total of 25 subjects were examined, including 15 females and

10 males at two time points: in week 1 (P1) and after three months of treatment (P3). Mean age of patients was 18.88 ± 5.59 years (min. 14, max. 35).

All the patients were diagnosed with acute polymorphic psychotic disorders (F23), and in case of all patients the diagnosis was changed during a 3-month follow-up to schizophrenia (F20), in accordance with the International Statistical Classification of Diseases and Related Health Problems, version 10 (ICD-10) [17]. The diagnosis was made by a physician experienced in psychiatric assessment.

None of the patients were diagnosed with comorbidities which could significantly affect the development of overweight and obesity, or cardiovascular diseases (including metabolic syndrome or hypertension). Before admission to hospital, the patients were not treated with neuroleptics.

The study was approved by the Ethics Committee of the Jagiellonian University (KBET 122.6120.23.2016).

Methods

The analysis included patient body weight as the dependent variable, since after its conversion to BMI, correlations between variables were less pronounced, especially because its interpretation must be performed with a classification according to established criteria, which have a wide range.

The methods used are described in Table 1.

Table 1. **Description of methods used**

Method	Scope of information
Socio-demographic survey	Patient data: age, education, permanent residence, marital status, family structure, financial situation. Parent data: education, job, social background, number of children.
Clinical data	Detailed admission history, duration and severity of psychotic symptoms before admission (DUP), pharmacotherapy and stimulants before admission, disease severity and treatment with neuroleptics during hospitalization (at subsequent time point).
Positive and Negative Syndrome Scale (PANSS) [18]	Assessment of degree and severity of psychotic symptoms. PANSS consists of 30 items divided into 3 subscales: PANSS – Positive Symptoms (PANSS P), PANSS – Negative Symptoms (PANSS N) and PANSS – General Psychopathology (PANSS G). It also allows calculation of total score (PANSS – Total – PANSS T).
Spielberger STAI questionnaire in the Polish adaptation by Wrześniewski et al. [19]	Assessment of the anxiety level as a personality trait and as a momentary situational state.
Coping Inventory for Stressful Situations (CISS) in the Polish adaptation by Szczepaniak, Strelau and Wrześniewski [20]	Coping with situations of mental pressure. The results are included in three scales: TO – task-oriented style; EO – emotion-oriented style; AO – avoidant style. The latter can take two forms: D – distraction seeking, and SD – social diversion].

table continued on the next page

Questionnaire Eating Behaviors (QEB) [21].	Diet quality expressed in healthy and unhealthy indices. It is composed of 21 questions on eating habits and 21 questions on the frequency of consumption of particular groups of products. The healthy diet index defines more frequent consumption of whole-meal bread, milk and fermented milk products, cottage cheese, fish and their products, foods from legume seeds, fruit and vegetables, while the unhealthy diet index refers to more frequent consumption of fast food, fried food, cheese, sweets and confectionery, canned food, sweet fizzy drinks, energetic drinks and alcoholic beverages.
Anthropometric data	The analysis also included body weight of patients and their parents (body weight of mother and father at 20–24, and current body weight).
Routine laboratory tests	Laboratory tests included complete blood count, biochemical tests, i.e., lipid profile [low density lipoproteins and high density lipoproteins (LDL and HDL), triglycerides (TG) and total cholesterol (TC)], inflammatory markers [C-reactive protein (CRP). Calculated indices: Neutrophil-to-lymphocyte ratio (NLR), Platelet-to-lymphocyte ratio (PLR)].

In the analysis of the correlation between body weight and treatment, chlorpromazine equivalents were used [22]. Additionally, treatment with olanzapine was analyzed as the drug with the greatest potential to influence body weight [23]. Olanzapine during therapy was used at least once in 14 out of 25 subjects (56%). Olanzapine at P1 only was used in 3 out of 25 subjects (12%), at P3 only was used in 7 out of 25 subjects (28%). Table 2 summarizes the weight gain in relation to the treatment with olanzapine.

Table 2. **Weight gain in relation to the treatment with olanzapine (n = 22)**

Variable	Weight gain ≥5kg (n = 12)		Weight gain <5 kg (n = 10)	
	Frequency	%	Frequency	%
Olanzapine used at least once	8	66.57 %	4	40.00 %
Olanzapine used at P1 only	1	8.33 %	2	20.00 %
Olanzapine used at P3 only	5	41.67 %	1	10.00 %

In order to assess the dynamics of the parameter change, differences (Variable_D) between baseline (Variable_P1) and after 3 months of treatment (Variable_P3) were calculated.

Statistical analyses were performed with the use of IBM SPSS 26 (descriptive statistics and Kendall tau-b correlation coefficients) and jamovi 1.8.4.0 (linear regression analysis). In all the analyses, the significance level was set at 0.05.

Results

Table 3 shows descriptive statistics of the study group.

Table 3. **Descriptive statistics of the study group (n = 25)**

Variables	Mean	Standard deviation	Minimum	Maximum
Age (years)	18.88	5.59	14	35
Number of children in the family	1.88	0.88	1.00	4.00
Weight_P1 (kg)	60.79	10.52	45.70	86.10
Weight_P3 (kg)	65.77	12.48	44.90	103.50
Weight_D (kg)	4.98	3.90	-2.00	17.40
PANSS G_P1 (points)	44.72	13.76	25.00	70.00
PANSS G_P3 (points)	24.60	8.06	15.00	49.00
PANNS G_D (points)	20.12	11.23	5.00	45.00
STAI trait_P1 (points)	57.60	6.29	48.00	70.00
STAI trait_P3 (points)	49.48	3.14	45.00	55.00
STAI trait_D (points)	8.12	4.70	1.00	20.00
STAI state_P1 (points)	55.04	5.51	47.00	65.00
STAI state_P3 (points)	47.32	2.91	43.00	52.00
STAI state_D (points)	7.72	4.20	2.00	17.00
CISS EO_P1 (points)	52.96	7.06	37.00	65.00
CISS EO_P3 (points)	49.64	5.07	42.00	58.00
CISS EO_D (points)	3.32	4.04	-11.00	8.00
CISS D_P1 (points)	25.56	5.38	17.00	35.00
CISS D_P3 (points)	23.00	4.02	16.00	31.00
CISS D_D (points)	2.56	2.33	-1.00	9.00
Conversion dose of chlorpromazine_P1 (mg)	180.00	73.60	50.00	300.00
Conversion dose of chlorpromazine_P3 (mg)	470.67	256.10	200.00	1200.00
Healthy diet index (nHDI-8)_P1 (points)	33.01	16.76	7.50	69.12
Healthy diet index (nHDI-8)_P3 (points)	31.52	1.19	31.00	36.00
Mother's body weight at the age of 20–24 (kg)	58.8	8.62	48.00	74.00
Mother's current body weight (kg)	62.56	7.50	50.00	80.00
Father's body weight at the age of 20–24 (kg)	68.40	6.78	55.00	83.00
Father's current body weight (kg)	72.48	6.94	60.00	90.00

Analysis of correlation

Non-parametric Kendall correlations between Weight_P1, Weight_P3,Weight_D, and the study variables were analyzed. The results where statistically significant correlations appeared are shown in Table 4.

Table 4. **Kendall correlations without bootstrap between Weight_P1, Weight_P3 and Weight_D with the study variables**

Variables	Weight_1(kg)	Weight_3 (kg)	Weight_D (kg)
Mother's body weight at the age of 20–24 (kg)	0.430**	0.445**	0.174
Mother's current body weight (kg)	0.336*	0.426**	0.202
Father's body weight at the age of 20–24 (kg)	0.461**	0.441**	0.003
Father's current body weight (kg)	0.309*	0.241	0.031
Number of children	0.421**	0.414**	-0.008
Healthy diet index (nHDI-8, points) _P1	-0.420**	-0.354*	-0.027
Healthy diet index (nHDI-8, points) _P3	0.466**	0.285	-0.045
Olanzapine used at least once	0.355*	0.286	0.079
Olanzapine used at P3 only	0.341*	0.337*	0.196
PANSS – G_P1 (points)	-0.382**	-0.390**	-0.115
PANSS G_D (points)	-0.412**	-0.386**	-0.068
STAI trait_D (points)	-0.090	0.003	0.323*
STAI state_P1 (points)	0.021	0.220	0.454**
STAI state_P3 (points)	-0.127	0.096	0.482**
CISS EO_P1 (points)	0.045	0.255	0.635**
CISS EO_P3 (points)	0.078	0.256	0.599**
CISS EO_D (points)	0.060	0.193	0.313*
CISS D_P1 (points)	-0.021	0.186	0.502**
CISS D_P3 (points)	-0.098	0.091	0.339*
CISS D_D (points)	0.054	0.219	0.571**

The statistical significance level was set at $p < 0.05$ and marked *: $*p < 0.05$; $**p < 0.01$

Also, correlations of the healthy diet index with the mother's and father's body weight at 20–24 and their current body weight were performed (Table 5). Negative correlations were observed between mother's body weight at 20–24, mother's current body weight, father's body weight at 20–24, and the patient healthy diet index.

Table 5. **Kendall correlation of the healthy diet index with the study variables (n = 25)**

Dependent variables / Variables	Healthy diet index (nHDI-8, in points)_P1	Healthy diet inde (nHDI-8, in points)_P3
Mother's body weight at the age of 20–24 (kg)	-0.345*	0.318
Mother's current body weight (kg)	-0.350*	0.296
Father's body weight at the age of 20–24 (kg)	-0.279	0.308
Father's current body weight (kg)	-0.176	0.367*

The statistical significance level was set at $p < 0.05$ and marked *: *$p < 0.05$

Linear regressions

The Weight_P3 dependent variable was analyzed. All linear regression models turned out to be statistically significant ($p < 0.001$). The results explain 97% (98% after removal of leverage observations) of the dependent variable variations. Final regression models for the Weight_P3 dependent variable are shown in Table 6 and 7. The leverage observation was removed based on the value of Cook's distance >1.

Weight_P1 turned out to be a significant predictor for the Weight_P3 variable ($p < 0.001$). Weight_P1 ($p < 0.001$) and CISS EO_P1 ($p = 0.001$) turned out to be significant predictors after removal of the leverage observation. An increase of Weight_P1 by 1 kg results in an increase of Weight_P3 by 0.88 kg, whereas an increase in the CISS EO_P1 score by 1 point results in an increase of Weight_P3 by 0.31 kg.

Table 6. **Results of linear regression (n = 25) for Weight_P3 (kg)**

$R^2 = 0.971$; $F(7;17) = 81.2$; $p < 0.001$	b	SE	95% Confidence interval		t	p	beta
			Lower	Upper			
Intercept	-23.56431	7.2949	-38.9552	-8.1734	-3.2302	0.005	
Weight_P1 (kg)	0.98263	0.0780	0.8181	1.1472	12.5989	<0.001	0.83268
Mother's body weight at the age of 24 (kg)	0.08279	0.0834	-0.0932	0.2587	0.9927	0.335	0.05721
Healthy diet index (nHDI-8, points) _P1	-0.02739	0.0428	-0.1178	0.0630	-0.6394	0.531	-0.03680
CISS TO_P1 (points)	0.00376	0.0757	-0.1560	0.1635	0.0497	0.961	0.00226
CISS EO_P1 (points)	0.33754	0.1169	0.0909	0.5842	2.8875	0.010	0.19107
CISS D_P1 (points)	0.19111	0.1637	-0.1542	0.5364	1.1677	0.259	0.08238
CISS SD_P1 (points)	0.22053	0.2290	-0.2627	0.7037	0.9629	0.349	0.04918

Table 7. **Results of linear regression (n = 24) for Weight_P3 (kg) after removal of leverage observations**

R^2 = 0.979, F(7;16) = 108; p <0.001	b	SE	95% Confidence interval		t	p	beta
			Lower	Upper			
Intercept	-9.55794	5.7977	-1.84851	2.7326	-1.649	0.119	
Weight_P1 (kg)	0.88210	0.0571	0.76106	1.0031	15.449	<0.001	0.83435
Mother's body weight at the age of 24 (kg)	0.11222	0.0568	-0.00812	0.2326	1.977	0.066	0.09543
Healthy diet index (nHDI-8, points) _P1	-0.00453	0.0294	-0.06685	0.0578	-0.154	0.879	-.00751
CISS TO_P1 (points)	-0.10215	0.0562	-0.22119	0.0169	-1.819	0.088	-0.07800
CISS EO_P1 (points)	0.30693	0.0793	0.13873	0.4751	3.868	0.001	0.20915
CISS D_P1 (points)	0.09106	0.1128	-0.14808	0.3302	0.807	0.431	0.04942
CISS SD_P1 (points)	0.01741	0.1611	-0.32404	0.3589	0.108	0.915	0.00496

Discussion of results

Our studies show a positive correlation between mother's and father's body weight, both at the age of 20–24 years and currently, and the body weight of patients with schizophrenia. This is consistent with studies conducted in various countries showing that the risk of obesity in children is higher when at least one parent is obese, while the highest correlation is observed when both parents are obese [24].

We observed a positive correlation between the number of siblings and the body weight of study subjects. This correlation may first of all point to a socio-cultural relationship. In Poland, having more children is related to a lower socioeconomic status (higher poverty and hardship) [25]. According to meta-analyses, low socioeconomic status of parents is a predictor of developing schizophrenia in the offspring [26]. Socioeconomic status of parents, especially higher income and education level has a significant influence on the nutritional safety of the family, which determines eating behaviors and body weight of their children. If there are more people in the family, it involves a reduction of income allocated to nutrition or choosing poor quality products [27]. No other relationships, however, were observed between the family status and body weight. The relationship found may also be of a biological nature related to evolutionary mechanisms or family dynamics.

The subject literature shows clear evidence of poor quality diet in patients with depression, anxiety or schizophrenia [28–30]. Insufficient consumption of fruit and vegetables, and wholemeal products results in a reduced supply of not only vitamins, micro – and macroelements to the body, but also of fibre, which improves blood glucose and body weight control, and reduces the risk of cardiovascular diseases, diabetes or metabolic syndrome [32, 33]. Moreover, increasing number of studies reveal a cor-

relation between an increased amount of vegetables in the diet and reduced depressive symptoms, anxiety or psychosis [33, 35]. Consumption of refined sugar in food and drinks favors the incidence of obesity alone [28] and also symptoms of psychosis, although the mechanism of action is still unclear [29]. We have shown, however, that lower body weight of patients at P1 and P3 is related to a higher healthy diet index at P1. The correlation of the healthy diet index at P1 with a lower body weight at P3 indicates transfer of learned eating habits to the hospital, where in theory diet is regulated by the staff. However, patients have a possibility of extra eating between meals, which is the subject of subsequent analyses.

We have not observed a correlation between chlorpromazine equivalents and weight gain of the patients. Nevertheless, in 8 out of 12 subjects with a body weight gain of more than 5 kg between two time points, olanzapine was used at least once. Studies show the highest risk of body weight gain, as well as BMI, when this drug is used versus placebo [23, 31].

In our study, we have observed a correlation between weight gains, and the PANNS, STAI and CISS scales.

The correlation between the presented psychopathology and weight gain may involve numerous aspects. We have observed a negative correlation with regard to psychotic symptoms PANSS – G_P1 and patient body weight at P1 and P3. The interpretation of this correlation is not clear. Supposedly, increased severity of psychotic symptoms may lead to a reduced intake of kilocalories during the day. Avolition and apathy which accompany schizophrenia may lead to a reduced motivation to prepare meals at home [28]. Studies reveal that patients with psychosis more often avoid breakfasts, hard products or meals without detectable structure, and at the same time have a tendency to eat late in the evening [29]. This may indicate a complex relationship between psychoticism and body weight, where some aspects of psychosis, such as intensive pharmacotherapy, psychomotor delay, symptoms of depression or anxiety, may cause an increase in body weight, while others may cause a decrease in body weight.

The obtained results indicate that higher weight gain is associated with higher anxiety, perceived both as emotional state and as personality trait. Studies suggest that 30–62% of patients with schizophrenia have concurrent anxiety disorders [32]. These subjects also reveal a higher incidence of panic disorder, social anxiety disorder (SAD) and obsessive-compulsive disorder, as compared with healthy subjects [33]. Meta-analyses show that anxiety occurs in 29% of subjects with FEP, and depression has a similar incidence [34]. SAD is the most frequently reported anxiety disorder in the group of subjects with psychosis, also in subjects with FEP [35]. Social anxiety is characterized by avoiding social relationships, shyness and fear of relations with others, which results in poorer function in the society and lower quality of life [36]. Nine-year-old follow-up of 1,634 subjects with depressive and/or anxiety disorders showed that the diet quality of subjects with disorders (especially concurrent ones) is the lowest, as compared to subjects in remission and to healthy subjects. Increased symptoms and chronicity of disorders are related to a less healthy diet [37]. The mechanism relating anxiety disorders with a lower quality diet involves emotional eating, eating addiction and binge eating [38]. New studies of Teasdale et al. [39] indicate more than twice

higher incidence of food addiction among patients with schizophrenia versus meta-analyses of this phenomenon in subjects with depressive disorders, anxiety or binge eating. Our study suggests, however, that anxiety may be a specific risk factor here, independent of schizophrenia symptoms. Inability to distinguish hunger from other impulses may lead to increased consumption of food, especially highly-processed, high-calorie and high-fat food, excluding more healthy alternatives, which in turn leads to an increase in body weight [37, 40].

Our studies are consistent with studies of other authors which show that subjects with psychosis choose dysfunctional styles of handling stress, focused on emotions or avoidance [41, 42]. Stress, if not buffered by adequate methods of coping, reduces self-control and threatens goal-congruent eating [43]. Ineffective coping with stress, related to getting involved in replacement activities may lead to emotional eating, which is an attempt to temporary reduce negative feelings and to experience comfort. Studies show that these coping styles are mediators between negative emotions and binge eating [43]. Differences in the selection frequency of the strategies used may result from severity of psychopathological symptoms [44]. This indicates a correlation between degree of stress severity and stress coping methods.

The obtained results indicate multi-factor conditions of weight gain in FEP in schizophrenia, including those unrelated to the disease process itself. In the context of the performed regression analyses, the baseline body weight of patients and style of coping with stress turn out to be of crucial importance. The baseline body weight is closely associated with variables of the diet and parents' body weight in their youth. Both these variables are also correlated with one another.

As reported by a systematic review of recent years, despite impairment of cognitive functions, poorer social interactions, motivation difficulties, and occurrence of psychotic symptoms, the use of dietary interventions seems to be accepted and manageable by subjects with schizophrenia. Moreover, it may give good results in the form of voluntary reduction of food and calorie intake during the day, as well as increased consumption of vegetables by subjects with schizophrenia, including subjects with FEP [29, 45]. Unfortunately, the results of analyses show that the most effective dietary interventions at hospital do not include voluntary use of diet, but forcing it by prohibition of eating food brought to hospital by the family [46]. Perhaps dietary intervention should involve not only patients, but also their family. Moreover, it would be good to ensure that hospital stores sell above all healthy food.

An important conclusion of the present studies is that the intervention should refer not only to the diet, but also to the way the eating habits are related to psychopathology associated with psychosis and to the general emotional function of the patient. It is not only about the effect of psychosis, but also the ways eating is integrated into coping with tensions and stresses of everyday life long before psychotic symptoms occur. The medical interview and dietary interventions should take place after admission to hospital, and should include not only the patient, but also his/her family. Issues related to care of the somatic condition of patients with schizophrenia require an interdisciplinary approach, since, as shown by the present study, significant observations in this respect may be related to medical, psychological and diet studies.

The present study has a number of limitations which affect the possibility of making conclusions. The group size prevents more complex analyses to determine the importance and correlations between particular factors. The study limitation is also related to the use of the consumption frequency questionnaire, since it is biased by overestimation and underestimation of the real consumption of products. Such bias may depend on the severity of disorders, since schizophrenia may affect cognitive functions. In the present analysis, we did not study a direct effect of the healthy diet index at the second study point because all patients were hospitalized and received a hospital diet. This diet at P3 was not associated with their eating habits, like the healthy diet index calculated on the basis of the QEB completed in the first week of treatment.

Conclusions

Out studies suggest that an increase in body weight of young patients with FEP is directly and indirectly influenced by their parents' body weight, both at the age of 20–24, and currently. This may indicate two things: copying nutritional patterns by the children and importance of biological factors. This explains unhealthy nutritional choices and behaviors, which are manifested by using an abnormal diet with a low healthy diet index which influence the body weight of schizophrenia patients. Body weight gain is also directly affected by anxiety symptoms occurring in schizophrenia, as well as non-adaptive and ineffective styles of coping with stress, which may lead to inability to differentiate between hunger and other stimuli, escaping from the problem and getting involved in replacement activities.

References

1. Nielsen RE, Banner J, Jensen SE. *Cardiovascular disease in patients with severe mental illness*. Nat. Rev. Cardiol. 2021; 18(2): 136–145.
2. Dudek D. *Zespół metaboliczny u pacjentów ze schizofrenią*. Forum Zaburzeń Metabolicznych 2010; 1(3): 123–130.
3. Kędzior A, Jakubek-Kipa K, Rzuszek M, Mazur A. *Trendy w występowaniu nadwagi i otyłości u dzieci na świecie, w Europie i Polsce*. Endokrynol. Pediatr. 2017; 16.1.58: 41–48.
4. Friedemann C, Heneghan C, Mahtani K, Thompson M, Perera R, Ward AM. *Cardiovascular disease risk in healthy children and its association with body mass index: Systematic review and meta-analysis*. BMJ 2012; 345: e4759.
5. Konarzewska B, Stefańska E, Wendołowicz A, Cwalina U, Golonko A, Małus A et al. *Visceral obesity in normal-weight patients suffering from chronic schizophrenia*. BMC Psychiatry 2014; 14(1): 35.
6. Barton BB, Segger F, Fischer K, Obermeier M, Musil R. *Update on weight-gain caused by antipsychotics: A systematic review and meta-analysis*. Expert Opin. Drug Saf. 2020; 19(3): 295–314.
7. Ryan MC, Flanagan S, Kinsella U, Keeling F, Thakore JH. *The effect of atypical antipsychotics on visceral fat distribution in first episode, drug-naïve patients with schizophrenia*. Life Sci. 2004; 74(16): 1999-2008.

8. Misiak B, Bartoli F, Stramecki F, Samochowiec J, Lis M, Kasznia J et al. *Appetite regulating hormones in first-episode psychosis: A systematic review and meta-analysis*. Neurosci. Biobehav. Rev. 2019; 102: 362–370.
9. Dasgupta A, Singh OP, Rout JK, Saha T, Mandal S. *Insulin resistance and metabolic profile in antipsychotic naïve schizophrenia patients*. Prog. NeuroPsychopharmacol. Biol. Psychiatry 2010; 34(7): 1202–1207.
10. Perry BI, McIntosh G, Weich S, Singh S, Rees K. *The association between first-episode psychosis and abnormal glycaemic control: Systematic review and meta-analysis*. Lancet Psychiatry 2016; 3(11): 1049–1058.
11. Yang W, Zheng L, Zheng B, Zeng S, Li J, Liang B et al. *A meta-analysis of abnormal glucose metabolism in first-episode drug-naive schizophrenia*. Psychiatr. Danub. 2020; 32(1): 46–54.
12. Ruiz LD, Zuelch ML, Dimitratos SM, Scherr RE. *Adolescent obesity: Diet quality, psychosocial health, and cardiometabolic risk factors*. Nutrients 2019; 12(1):43
13. Gardner R, Feely A, Layte R, Williams J, McGavock J. *Adverse childhood experiences are associated with an increased risk of obesity in early adolescence: A population-based prospective cohort study*. Pediatr. Res. 2019; 86(4): 522–528.
14. Duraccio KM, Krietsch KN, Chardon ML, Van Dyk TR, Beebe DW. *Poor sleep and adolescent obesity risk: A narrative review of potential mechanisms*. Adolesc. Health Med. Ther. 2019; 10: 117–130.
15. Kakinami L, Barnett TA, Séguin L, Paradis G. *Parenting style and obesity risk in children*. Prev. Med. (Baltim). 2015; 75: 18–22.
16. Fang K, Mu M, Liu K, He Y. *Screen time and childhood overweight/obesity: A systematic review and meta-analysis*. Child. Care. Health Dev. 2019; 45(5): 744–753.
17. World Health Organization. *International statistical classification of diseases and related health problems*. 2004.
18. Kay SR, Fiszbein A, Opler LA. *The positive and negative syndrome scale (PANSS) for schizophrenia*. Schizophr. Bull. 1987; 13(2): 261–276.
19. Wrześniewski K, Sosnowski T, Jaworowska A, Fecenec D. *Inwentarz stanu i cechy lęku (polska adaptacja STAI)*. Warsaw: Psychological Test Laboratory of the Polish Psychological Association; 2011.
20. Strelau J, Jaworowska A, Wrześniewski K, Szczepaniak P. *Kwestionariusz Radzenia Sobie w Sytuacjach Stresowych CISS*. Warsaw: Psychological Test Laboratory of the Polish Psychological Association; 2007.
21. Wądołowska L, Krusińska B. *Procedura opracowania danych żywieniowych z kwestionariusza QEB*. 2015. http://www.uwm.edu.pl/edu/lidiawadolowska/ (retrieved: 5.01.2023).
22. Wickham H, Averick M, Bryan J, Chang W, D'Agostino McGowan L, François R et al. *Welcome to the Tidyverse*. J. Open Source Softw. 2019; 4(43): 1686.
23. Pillinger T, McCutcheon RA, Vano L, Mizuno Y, Arumuham A, Hindley G et al. *Comparative effects of 18 antipsychotics on metabolic function in patients with schizophrenia, predictors of metabolic dysregulation, and association with psychopathology: A systematic review and network meta-analysis*. Lancet Psychiatry 2020; 7(1): 64–77.
24. Wang Y, Min J, Khuri J, Li M. *A systematic examination of the association between parental and child obesity across countries*. Adv. Nutr. 2017; 8(3): 436–448.
25. Central Statistical Office of Poland. *Zasięg ubóstwa ekonomicznego w Polsce w 2019 roku*. Department of Living Conditions and Research. 2020.

26. Agerbo E, Sullivan PF, Vilhjálmsson BJ, Pedersen CB, Mors O, Børglum AD et al. *Polygenic risk score, parental socioeconomic status, family history of psychiatric disorders, and the risk for schizophrenia: A Danish population-based study and meta-analysis*. JAMA Psychiatry 2015; 72(7): 635–641.
27. Huang H, Bt Wan Mohamed Radzi CWJ, Jenatabadi HS. *Family environment and childhood obesity: A new framework with structural equation modeling*. Int. J. Environ. Res. Public Health 2017; 14(2): 181.
28. Jakobsen AS, Speyer H, Nørgaard HCB, Karlsen M, Hjorthøj C, Krogh J et al. *Dietary patterns and physical activity in people with schizophrenia and increased waist circumference*. Schizophr. Res. 2018; 199: 109–115.
29. Aucoin M, Lachance L, Cooley K, Kidd S. *Diet and psychosis: A scoping review*. Neuropsychobiology 2020; 79(1): 20–42.
30. Teasdale SB, Ward PB, Samaras K, Firth J, Stubbs B, Tripodi E et al. *Dietary intake of people with severe mental illness: Systematic review and meta-analysis*. Br. J. Psychiatry 2019; 214(5): 251–259.
31. Sneller MH, Boer de N, Everaars S, Schuurmans M, Guloksuz S, Cahn W et al. *Clinical, biochemical and genetic variables associated with metabolic syndrome in patients with schizophrenia spectrum disorders using second-generation antipsychotics: A systematic review*. Front. Psychiatry 2021; 12: 625935.
32. Howells FM, Kingdon DG, Baldwin DS. *Current and potential pharmacological and psychosocial interventions for anxiety symptoms and disorders in patients with schizophrenia: Structured review*. Hum. Psychopharmacol. 2017; 32(5): 1–11.
33. Kiran C, Chaudhury S. *Prevalence of comorbid anxiety disorders in schizophrenia*. Ind. Psychiatry J. 2016; 25(1): 35–40.
34. Wilson RS, Yung AR, Morrison AP. *Comorbidity rates of depression and anxiety in first episode psychosis: A systematic review and meta-analysis*. Schizophr. Res. 2020; 216: 322–329.
35. McEnery C, Lim MH, Knowles A, Rice S, Gleeson J, Howell S et al. *Social anxiety in young people with first-episode psychosis: Pilot study of the EMBRACE moderated online social intervention*. Early Interv. Psychiatry 2021; 15(1): 76–86.
36. Aikawa S, Kobayashi H, Nemoto T, Matsuo S, Wada Y, Mamiya N et al. *Social anxiety and risk factors in patients with schizophrenia: Relationship with duration of untreated psychosis*. Psychiatry Res. 2018; 263: 94–100.
37. Gibson-Smith D, Bot M, Brouwer IA, Visser M, Penninx BWJH. *Diet quality in persons with and without depressive and anxiety disorders*. J. Psychiatr. Res. 2018; 106: 1–7.
38. Burrows T, Kay-Lambkin F, Pursey K, Skinner J, Dayas C. *Food addiction and associations with mental health symptoms: A systematic review with meta-analysis*. J. Hum. Nutr. Diet. 2018; 31(4): 544–572.
39. Teasdale SB, Burrows TL, Hayes T, Hsia CY, Watkins A, Curtis J et al. *Dietary intake, food addiction and nutrition knowledge in young people with mental illness*. Nutr. Diet. 2020; 77(3): 315–322.
40. Konttinen H. *Emotional eating and obesity in adults: The role of depression, sleep and genes*. Proc. Nutr. Soc. 2020; 79(3): 283–289.
41. MacAulay R, Cohen AS. *Affecting coping: Does neurocognition predict approach and avoidant coping strategies within schizophrenia spectrum disorders?* Psychiatry Res. 2013; 209(2): 136–141.
42. Allott KA, Rapado-Castro M, Proffitt TM, Bendall S, Garner B, Butselaar F et al. *The impact of neuropsychological functioning and coping style on perceived stress in individuals with first-episode psychosis and healthy controls*. Psychiatry Res. 2015; 226(1): 128–135.

43. Pannicke B, Kaiser T, Reichenberger J, Blechert J. *Networks of stress, affect and eating behaviour: Anticipated stress coping predicts goal-congruent eating in young adults*. Int. J. Behav. Nutr. Phys. Act. 2021; 18(1): 1–14.
44. Piotrowski P, Rymaszewska J, Stańczykiewicz B, Małecka M, Kotowicz K, Samochowiec J et al. *Stress coping strategies and their clinical correlates in patients with psychosis at various stages of illness: A case-control study*. Early Interv. Psychiatry 2020; 14(5): 559–567.
45. Curtis J, Watkins A, Rosenbaum S, Teasdale S, Kalucy M, Samaras K et al. *Evaluating an individualized lifestyle and life skills intervention to prevent antipsychotic-induced weight gain in first-episode psychosis*. Early Interv. Psychiatry 2016; 10(3): 267–276.
46. Issac J, Guerreiro S, Pinheiro C, Pardal A, Correia N, Simões S et al. *Diet and physical activity intervention effectiveness in acute mental patients, during hospitalization: A matched case-control study*. Eur. Psychiatry 2017; 41(S1): S137–S138.

Address: Bogna Batko
Chair of Psychiatry
Department of Child and Adolescent Psychiatry, ,
31-501 Kraków, Kopernika Street 21a
e-mail: bogna.batko@gmail.com