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Key factors involved in the feeding and eating disorders among schizophrenic patients and non-clinical controls

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Summary

Background and objectives: Factors associated with comorbidity of schizophrenia and feeding and eating disorders (FEDs) need to be precisely investigated due to their endocrine and cardiometabolic complications. Therefore, the present study was performed aiming to examine the prevalence of FEDs and its involved key factors among schizophrenic patients and non-clinical controls

Material and methods: In this cross-sectional correlational study, a total of 268 Iranian participants (including 134 schizophrenic patients and 134 non-clinical controls) were selected through the convenient sampling method from people living in Zahedan, Iran. They were assessed using demographic information form, Eating Attitudes Test-26 Item (EAT-26), and Structured Clinical Interviews for DSM-5: Research Version.

Results: The findings of this study showed that almost one third of schizophrenic patients had FEDs that this prevalence was about 2.5 times that in non-clinical controls (32.1% vs. 12.7%, $p<0.001$). in schizophrenic patients, the results of regression analysis also revealed that higher scores of EAT-26 were associated with higher levels of anxiety, depression, and body mass index (BMI), Type 2 diabetes, tobacco smoking, earlier stages of disease, more severe psychotic symptoms, and taking atypical antipsychotics ($R^2=0.93$, p□0.001). Contrarily, no relationship was observed between the EAT-26 scores and both gender and different phases of schizophrenia.

Discussion: Clinicians need to assess the risk of FEDs during the entire course of schizophrenia, particularly in its earlier stages.

Conclusions: It is necessary to address the role of key factors, such as anxiety, depression, BMI, Type 2 diabetes, tobacco smoking, severity of psychosis, category of antipsychotic medications in the modeling of eating pathology among schizophrenic patients.

feeding and eating disorders, patients, schizophrenia

1. INTRODUCTION

Schizophrenia is one of the most common serious mental illnesses that involve 1% of the general population [1]. However, one of the current pharmacological strategies approved for treating various aspects of schizophrenia is to use atypical antipsychotics. On the other hand, a more sophisticated and perhaps effective approach to its treatment is independently targeting the pathophysiological mechanisms of each clinical dimension using more selective drugs, which can be separately combined or titrated de-

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pending on the need of each patient [2]. Despite that, diagnostic confusion and uncertainty about the treatment of schizophrenia might be caused by its comorbidity with feeding and eating disorders (FEDs) [3]. Previous studies have indicated that abnormal eating behaviors and FEDs among schizophrenic patients probably increase mortality and morbidity risks 2-3 times higher than that of the general population by provoking endocrine and cardiometabolic alterations (such as obesity, dyslipidemia, hypertension, and Type 2 diabetes) [4-6]. Although comorbidity of these two disorders has been addressed since the 19th century [7], it has rarely received clinical attention [8]. This is probably because typical criteria of FEDs in schizophrenic patients have not been met, which has caused clinicians to assume their eating behaviors as a secondary problem [9]. Nevertheless, both initial investigations [10] and current studies [1] have suggested that disordered eating attitudes, particularly binge eating disorder and night-eating syndrome, are prevalent among schizophrenic patients. Although some of these behaviors could be a consequence of pharmacological treatments (especially atypical antipsychotics) [11,12], there is evidence that they can be observed in drug-naïve individuals [13]. These contradictions between hypotheses and clinical findings show that the development of FEDs in schizophrenic patients is a multifactorial phenomenon [1]. In this regard, recent genome-wide association studies have shown a positive genetic correlation between schizophrenia and anorexia nervosa and an inverse genetic correlation between these two mental disorders and body mass index (BMI) [14]. Further, in recent years, various factors such as gender, duration of psychosis [11], mood disorders, stress [15], tobacco smoking [16], and Type 2 diabetes [17] have been found to play significant roles in the high prevalence of abnormal eating behaviors of schizophrenic patients.

Despite these limited and scattered findings, no study has comprehensively and princely investigated the factors associated with FEDs in schizophrenic patients compared to the non-clinical population [1,18]. Since the development of FEDs in schizophrenic patients might be accompanied by the risk of obesity and cardiometabolic disorders and an increase in its mortality and morbidity rates [18], it seems necessary to identify relevant factors and propose preventive approaches [19]. This study has been intended to not only investigate and compare the prevalence of FEDs in schizophrenic patients and non-clinical controls but also examine their associated key factors in each group separately.

2. MATERIAL AND METHODS

2.1. Study design and participants

In this cross-sectional correlational study, based on the Green's method [20] and assuming 15% attrition of participants, 134 schizophrenic patients (from May 2018 to November 2019) were recruited by convenience sampling from the outpatients who referred to Baharan psychiatric hospital in Zahedan, Iran. Moreover, non-clinical controls were recruited through one-to-one matching from the residents of the same geographical region (i.e., one case dedicated to one control; n=134). The inclusion criteria were as follows: 1) Schizophrenia diagnosis by a psychiatrist using Structured Clinical Interviews for DSM-5: Research Version (SCID-5-RV); 2) ranged between 18 to 70. Also, the exclusion criteria comprised the following elements: 1) acute psychotic episode; 2) intellectual disability; 3) history of neurological disorders; 4) hearing loss; 5) treatment with antidepressants or mood stabilizers over the past three months; 6) incorrectly-filled questionnaire.

2.2. Data collection

After approving the present study by the Zahedan University of Medical Sciences Research Ethics Committee (REC) Reg no. IR.ZAUMS.REC.1398.210 and providing the required details about the concerned objectives, consent forms were given to all of the participants. Also, the personal information of subjects was kept confidential, and they were allowed to leave the study for any reason to obey the Declaration of Helsinki thoroughly. After obtaining the consent from both groups, subjects with the scores of ≥20 in Eating Attitudes Test-26 Item (EAT-26) were examined by a psychiatrist using SCID-5-RV to identify whether they had FEDs. Eventu-

ally, 47 schizophrenic patients and 17 non-clinical controls diagnosed with FEDs were involved in the study, with no significant differences in age, gender, marital status, employment, and level of education (see Table 1). Then, all of the participants in both groups were evaluated in terms of levels of anxiety, depression, and BMI (weight (kg) divided by squared height (m^2)), Type 2 diabetes (according to fasting blood sugar ≥136mg/dL on two separate tests), and tobacco smoking (yes/no). Furthermore, factors such as duration of psychosis (i.e., from 6 months to 2 years, 2 to 5 years, 5 to 10 years, and greater than or equal to 10 years), phases of schizophrenia (active phase or remission), severity of psychosis, and category of antipsychotic medications (typical or atypical) were determined for schizophrenic patients.

Table 1. Socio-demographic characteristics among two study groups

Variables	Categories	Schizophrenic patients (*n* = 43)	Non-clinical controls (*n* = 17)	
		n (%)	*n* (%)	Test[a]
Age	20-29	24 (55.8)	9 (52.9)	$\chi^2 = 0.126$
	30-39	12 (27.9)	4 (23.5)	
	40-49	3 (7.0)	2 (11.8)	
	50-60	4 (9.3)	2 (11.8)	
Gender	Male	18 (41.9)	7 (41.2)	$\chi^2 = 0.002$
	Female	25 (58.1)	10 (58.8)	
Marital status	Single	28 (65.1)	11 (64.7)	$\chi^2 = 0.001$
	Married	15 (34.9)	6 (35.3)	
Employment	Employed	25 (58.1)	11 (64.7)	$\chi^2 = 0.219$
	Unemployed	18 (41.9)	6 (35.3)	
Level of education	Illiterate	8 (18.6)	7 (41.2)	$\chi^2 = 3.280$
	Elementary grade	4 (9.3)	2 (11.8)	
	Middle grade	8 (18.6)	4 (23.5)	
	High school	10 (23.3)	0 (0.0)	
	College	13 (30.2)	4 (23.5)	

Note. [a]Statistical analyzing applied chi-square test and Kruskal-Wallis test.

Note. *p < 0.05; **p < 0.01; ***p < 0.001.

2.3. Measures

2.3.1. EAT-26

This test consists of 26 items scored based on a 6-point Likert scale. For question No. 1-25, answers "always", "usually", and "often" have the scores of 3, 2, and 1, respectively, and other responses such as "never", "rarely", and "sometimes score 0. Only question No. 26 is scored inversely. The scores of this test range between 0 and 78, and a score of ≥20 stands for the risk of FEDs. The reliability and validity of this questionnaire have been reported to be suitable (Cronbach's alpha of 0.75) in Iran [21]. In this study, the Cronbach's alpha coefficient for total scores was obtained of 0.80.

2.3.2. Beck Anxiety Inventory (BAI)

This 21-item questionnaire is scored on a 4-point scale (0-3), with the minimum and maximum scores of 0 and 63, respectively. It has been found suitable in Iran in terms of reliability and validity (Cronbach's alpha of 0.92) [22]. In the present study, the Cronbach's alpha coefficient for total scores was 0.88.

2.3.3. Beck Depression Inventory-II (BDI-II)

This 21-item questionnaire is scored on a 4-point scale (0-3), with the minimum and maximum scores of 0 and 63, respectively. It has been found suitable in Iran in terms of reliability and validity (Cronbach's alpha of 0.87) [23]. In the present study, the Cronbach's alpha coefficient for total scores was 0.85.

2.3.4. SCID-5-RV

This tool is a semi-structured clinical interview for the main diagnoses of DSM-5, which is carried out by a clinician or trained health expert who is familiar with diagnostic criteria and categorization of disorder in DSM-5. SCID-5-RV has been reported as reliable and valid in different studies [24].

2.3.5. Positive and Negative Syndrome Scale (PANSS)

This questionnaire includes 30 items scored based on a 5-point Likert scale (absent, minimal, moderate, severe, and extreme). Accordingly, the minimum and maximum scores would be 30 and 150, respectively. The validity and reliability of this questionnaire have been reported suitable in Iran (Cronbach's alpha of 0.77) [25]. In this study, the Cronbach's alpha for the total scores of PANSS was obtained of 0.75.

2.4. Statistical analysis

The data were analyzed using the SPSS v.25 software, and a significance level of less than 0.05 was considered. Descriptive statistical methods, including mean and standard deviation, were implemented to investigate the data. Also, the Chi-square test and Kruskal-Wallis test were conducted to compare socio-demographic characteristics between the two study groups. The correlation among the variables was evaluated using the point-biserial correlation coefficient, Pearson correlation coefficient, and Spearman's rank correlation coefficient. Further, hierarchical linear regression was used for inves-

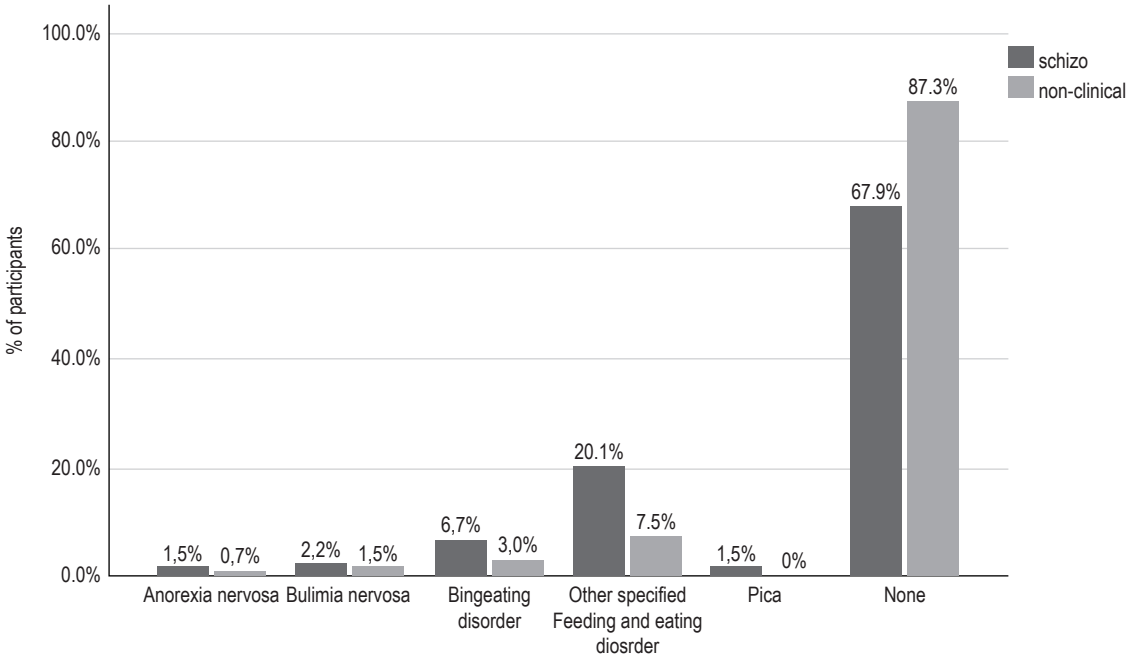

Figure 1. The prevalence of different types of feeding and eating disorders (FEDs) in two study groups

tigating the relationship between independent and dependent variables.

3. RESULTS

The preliminary investigations reported the prevalence of FEDs in schizophrenic patients and non-clinical controls to be 32.1 and 12.7%, respectively ($\chi^2(1)=14.51$, $p<0.001$). The most prevalent clinical diagnosis in both study groups was attributed to other specified feeding and eating disorders (OSFEDs) (10% in schizophrenic patients and 3.7% in non-clinical controls), as shown in Figure 1. Also, as a new finding in this study, coexistence of pica and polydipsia was observed in two schizophrenic patients.

Moreover, correlation results in schizophrenic patients revealed that the EAT-20 scores had a significant positive correlation with anxiety (r=0.52, p<0.001), depression (r=0.44, p=0.003), BMI (r=0.76, p<0.001), Type 2 diabetes (r=0.82, p<0.001), tobacco smoking (r=0.76, p<0.001), severity of psychosis (r=0.66, p<0.001), and category of antipsychotic medications (r=0.62, p<0.001), and a significant negative correlation with the duration of psychosis (r=-0.36, p=0.017). Furthermore, these results in non-clinical controls indicated a significant positive correlation between the EAT-20 scores and gender (r=0.84, p<0.001), anxiety (r=0.88, p<0.001), depression (r=0.48, p=0.049), BMI (r=0.72, p=0.001), Type 2 diabetes (r=0.66, p=0.004), and tobacco smoking (r=0.49, p=0.042), as listed in Table 2.

Table 2. Correlations between the scores of Eating Attitudes Test-26 Item (EAT-26) and explanatory factors among two study groups

Variables	Schizophrenic patients (*n* = 43)	Non-clinical controls (*n* = 17)
Age	0.24	-0.00
Gender	0.09	0.84***
Marital status	0.16	0.07
Employment	0.25	0.07
Level of education	-0.08	-0.01
Anxiety	0.52***	0.88***
Depression	0.44**	0.48*
Body mass index (BMI)	0.76***	0.72**
Type 2 diabetes	0.82***	0.66**
Tobacco smoking	0.76***	0.49*
Duration of psychosis	-0.36*	-
The phases of schizophrenia	-0.08	-
Severity of psychosis	0.66***	-
Category of antipsychotic medications	0.62***	-

Note. Statistical analyzing applied point-biserial correlation coefficient, Pearson correlation coefficient, and Spearman's rank correlation coefficient.
Note. *p < 0.05; **p < 0.01; ***p < 0.001.

The above variables were incorporated in the linear regression analysis to identify the key factors associated with FEDs in both study groups. The results of regression analysis in schizophrenic patients (Table 3) showed that higher levels of anxiety (β=0.17, p=0.006) and depression (β=0.14, p=0.009), high BMI (β=0.14, p=0.049), Type 2 diabetes (β=0.20, p=0.023), tobacco smoking (β=0.19, p=0.015), earlier stages of disease (β=-0.12, p=0.007), more severe psychotic symptoms (β=0.27, p<0.001), and taking atypical antipsychotics (β=0.14, p=0.011)

were related to higher scores of EAT-26 ($F(8,34)=65.80$, $R^2=0.93$, $p<0.001$). For non-clinical controls (Table 4), female gender ($\beta=0.65$, $p=0.001$), higher levels of anxiety ($\beta=0.63$, $p=0.001$) and depression ($\beta=0.28$, $p=0.012$), high BMI ($\beta=0.43$, $p=0.008$), Type 2 diabetes ($\beta=0.26$, $p=0.023$), and tobacco smoking ($\beta=0.22$, $p=0.023$) were found to be significantly associated with higher scores of EAT-26 ($F(6,10)=36.53$, $R^2=0.95$, $p<0.001$).

Table 3. Summary of regression analysis to determine the key factors involved in the feeding and eating disorders (FEDs) among schizophrenic patients (n = 43)

				95% C.I.	
Explanatory factors	B (β)	SE	t	Lower bound	Upper bound
Anxiety	0.15 (0.17)**	0.05	2.95	0.04	0.25
Depression	0.12 (0.14)**	0.04	2.75	0.03	0.21
Body mass index (BMI)	2.64 (0.14)*	1.29	2.04	0.01	5.28
Type 2 diabetes (Yes)	3.86 (0.20)*	1.61	2.38	0.57	7.51
Tobacco smoking (Yes)	3.53 (0.19)*	1.37	2.56	0.74	6.33
Duration of psychosis	-0.84 (-0.12)**	0.29	-2.85	-1.45	-0.24
Severity of psychosis	0.12 (0.27)***	0.02	4.89	0.07	0.17
Category of antipsychotic medications (Atypical)	2.64 (0.14)*	0.98	2.69	0.65	4.64
R	0.96				
R^2	0.93				
Adj. R^2	0.92				
F (df1, df2)	65.80 (8, 34)***				
Note. $^*p < 0.05$; $^{**}p < 0.01$; $^{***}p < 0.001$.					

Table 4. Summary of regression analysis to determine the key factors involved in the feeding and eating disorders (FEDs) among non-clinical controls (n = 17)

				95% C.I.	
Explanatory factors	B (β)	SE	t	Lower bound	Upper bound
Gender (Female)	12.86 (0.65)**	2.88	4.46	6.44	19.29
Anxiety	0.61 (0.63)**	0.12	5.02	0.34	0.89
Depression	0.18 (0.28)*	0.05	3.04	0.04	0.31
Body mass index (BMI)	8.41 (0.43)**	2.53	3.31	2.76	14.07
Type 2 diabetes (Yes)	5.91 (0.26)*	2.20	2.68	1.00	10.82
Tobacco smoking (Yes)	5.51 (0.22)*	2.06	2.69	0.96	10.14
R	0.97				
R^2	0.95				
Adj. R^2	0.93				
F (df1, df2)	36.53 (6, 10)***				
Note. $^*p < 0.05$; $^{**}p < 0.01$; $^{***}p < 0.001$.					

4. DISCUSSION

The present cross-sectional correlational study was carried out aiming to determine the prevalence of FEDs and its relevant key factors in schizophrenic and non-clinical controls. The initial results reported the prevalence of FEDs in schizophrenic patients and non-clinical controls to be 32.1% and 12.7%, respectively. Among them, OSFEDs was found the most prevalent clinical diagnosis in both study groups (10% in schizophrenic patients and 3.7% in non-clinical controls). In a previous review, Kouidrat et al. [1] showed a high rate of comorbidity between schizophrenia and FEDs and a relatively high prevalence of OSFEDs among schizophrenic patients (8-25%) and the general population (1.5% in total and 8.9-27% in obese people). They attributed the heterogeneity of prevalence to the difference in methodology, sample sizes, and scales of the studies [1].

A new finding of this study was the coexistence of pica and polydipsia observed in two schizophrenic patients. Pica is generally defined as repeated ingestion of one or more nonnutritive and nonfood substances (e.g., hair, string, chalk, paint, clay, ash, ice, etc.) over a period of at least 1 month, which is prevalent in children (rarely adults) with developmental disorders (e.g., autism spectrum disorder or intellectual disability) [26]. In schizophrenia, pica is also defined as an impulsive consumption associated with delusions [1] that has been previously reported in various forms such as saturnism (lead poisoning) [27], coprophagia (ingestion of feces) [28], and potomania (drinking excessive amounts of beverages, around 8-10 liters per day) [29]. Schizophrenic patients with pica might display polydipsia for two main reasons [30]: (i) schizophrenic patients with pica and polydipsia have a generalized tendency to ingest who probably drink different kinds of liquids excessively from inappropriate places (e.g., a puddle of water) since they cannot differentiate between healthy and harmful fluids; (ii) hiding any feelings of pain or anxiety, e.g., a patient with gastrodynia might drink a large volume of water to alleviate pain and distracts himself/herself from it as well. These results as to the comorbidity of pica and polydipsia in schizophrenic patients might offer a critical clinical implication. Since polydipsia can pose the risk of hyponatremia, convulsions, and coma by causing water intoxication and, in turn, severe metabolic imbalances [29], clinicians need to investigate this comorbidity in schizophrenic patients with pica, conscientiously.

As further, the results of regression analysis in schizophrenic patients and non-clinical controls revealed that the higher scores of EAT-26 were associated with higher levels of anxiety and depression. Based on the "escape from awareness" model [31], overeating in both schizophrenic patients and non-clinical controls seems to be a method of coping with negative affect applied for the maintenance of self-control [32, 33]. Previously, neurologic studies have also proposed that some types of overeating experiences might be related to the reduction of amygdala activity [34].

The regression analysis also demonstrated that, in both study groups, Type 2 diabetes and tobacco smoking might lead to an increase in the vulnerability to FEDs, which was consistent with the results obtained by García-Mayor et al. [17], Kouidrat et al. [18], and Essawy et al. [35]. These researches showed that high mean of BMI, Type 2 diabetes, and tobacco smoking could increase the risk of FEDs due to various reasons, including dietary regimens, overweight or obesity, and exacerbating interpersonal conflict and violence. Additionally, in schizophrenic patients, higher scores of EAT-26 were also associated with earlier stages of disease and more severe psychotic symptoms (probably due to the use of overeating as an adaptive defense against stress) [36,37], and taking atypical antipsychotics (probably due to gaining weight followed by altered appetite sensations and increased susceptibility to hunger) [11,12]. Sallemi et al. [38], Sentissi et al. [11], Blouin et al. [12], and Fawzi et al. [13] have presented similar results in this respect. However, only the study conducted by Kouidrat et al. [18] indicated that abnormal eating behaviors were not related to the duration of psychosis and category of antipsychotic medications. These contradictory results seem to suggest that the effects of antipsychotic medication switching as a strategy to reduce metabolic problems in schizophrenic patients won't always be accompanied by clear evidence for better outcome [39].

The present study had two other results. First, there was no significant association between dif-

ferent phases of schizophrenia (i.e., active pahes or remission) and scores of EAT-26, which was inconsistent with the results reported by Khalil et al. [40], who showed that abnormal eating behaviors are reduced by psychotic episodes and relapse when psychosis remit. This finding suggests that abnormal eating behaviors might be a comorbid condition or part of schizophrenia-spectrum disorders [41]. Second, unlike non-clinical controls, no significant relation was observed between scores of EAT-26 and gender for schizophrenic patients, which was consistent with the study of Kouidrat et al. [18] and inconsistent with the results obtained by Sallemi et al. [38] and Sentissi et al. [11]. Aside from the two latter studies, the results of the present study alongside previous research suggested that the prevalence of abnormal eating behaviors were equal in men and women with schizophrenia (unlike the general population wherein women are more affected by abnormal eating behaviors compared to men [1]).

As a limitation, cross-sectional studies cannot provide an accurate estimation of the frequency of a disorder (particularly in schizophrenic patients) using self-reporting questionnaires. On the other hand, precise understanding of relationships' nature (especially causality) is not possible in cross-sectional studies. Therefore, longitudinal studies can be designed to resolve this limitation noticeably. Moreover, the small sample size in this study might hamper an accurate estimation of abnormal eating behaviors among schizophrenic patients. Also, the results cannot be generalized to other regions since the sample size was restricted to a single geographic region (considering individual, social, and cultural differences). Thus, wider research needs to be carried out worldwide. As the last limitation, there was no standardized assessment of abnormal eating behaviors among schizophrenic patients, hindering the possibility of evaluating the frequency of these behaviors.

5. CONCLUSIONS

This study aimed to investigate the prevalence of FEDs and its associated factors in schizophrenic and non-clinical controls. The obtained results revealed that one-third of schizophrenic patients had FEDs that this prevalence was almost 2.5 times that in non-clinical controls. Further, abnormal eating behaviors in schizophrenic patients might be affected by various factors, including anxiety, depression, BMI, Type 2 diabetes, tobacco smoking, severity of psychosis, and category of antipsychotic medications. Therefore, irrespective of gender, it is required to assess the risk of FEDs during the entire course of schizophrenia, particularly at earlier stages of schizophrenia. Also, clinicians must pay further attention to the key role of factors such as anxiety, depression, BMI, Type 2 diabetes, tobacco smoking, severity of psychosis, and category of antipsychotic agents in the modeling of eating pathology among schizophrenic patients. Nevertheless, future studies are required to prove the exact and real role of the above factors in the development of abnormal eating behaviors among schizophrenic patients

REFERENCES

1. Kouidrat Y, Amad A, Lalau JD, Loas G. Eating disorders in schizophrenia: implications for research and management. Schizophr Res Treatment. 2014;2014:791573. https://doi.org/10.1155/2014/791573.
2. Kapur S, Remington G. Atypical antipsychotics: new directions and new challenges in the treatment of schizophrenia. Annu Rev Med. 2001;52:503-17. https://doi.org/10.1146/annurev.med.52.1.503.
3. Seeman MV. Eating disorders and psychosis: Seven hypotheses. World J Psychiatry. 2014;4(4):112-9. https://doi.org/10.5498/wjp.v4.i4.112.
4. Saha S, Chant D, McGrath J. A systematic review of mortality in schizophrenia: is the differential mortality gap worsening over time?. Arch Gen Psychiatry. 2007;64(10):1123-31. https://doi.org/10.1001/archpsyc.64.10.1123.
5. Klump KL, Bulik CM, Kaye WH, Treasure J, Tyson E. Academy for eating disorders position paper: eating disorders are serious mental illnesses. Int J Eat Disord. 2009;42(2):97-103. https://doi.org/10.1002/eat.20589.
6. Treasure J, Claudino AM, Zucker N. "Eating disorders,". Lancet. 2010;375(9714):583-93. https://doi.org/10.1016/S0140-6736(09)61748-7.
7. Hoff P. Eugen Bleuler's concept of schizophrenia and its relevance to present-day psychiatry. Neuropsychobiology. 2012;66(1):6-13. https://doi.org/10.1159/000337174.
8. Solmi F, Mascarell MC, Zammit S, Kirkbride JB, Lewis G. Polygenic risk for schizophrenia, disordered eating behaviors and body mass index in adolescents. Br J Psychiatry. 2019;215(1):428-33. https://doi.org/10.1192/bjp.2019.39.

9. Yum SY, Caracci G, Hwang MY. Schizophrenia and eating disorders. Psychiatr Clin North Am. 2009 Dec;32(4):809-19. https://doi.org/10.1016/j.psc.2009.09.004.
10. Striegel-Moore RH, Garvin V, Dohm FA, Rosenheck RA. Eating disorders in a national sample of hospitalized female and male veterans: Detection rates and psychiatric comorbidity. Int J Eat Disord. 1999;25(4):405-14. https://doi.org/10.1002/(sici)1098-108x(199905)25:4<405::aid-eat5>3.0.co;2-f.
11. Sentissi O, Viala A, Bourdel MC, Kaminski F, Bellisle F, Olié JP, Poirier MF. Impact of antipsychotic treatments on the motivation to eat: preliminary results in 153 schizophrenic patients. Int Clin Psychopharmacol. 2009;24(5):257-64. https://doi.org/10.1097/YIC.0b013e32832b6bf6.
12. Blouin M, Tremblay A, Jalbert ME, Venables H, Bouchard RH, Roy MA, Alméras N. Adiposity and eating behaviors in patients under second generation antipsychotics. Obesity. 2008;16(8):1780-7. https://doi.org/10.1038/oby.2008.277.
13. Fawzi MH, Fawzi MM. Disordered eating attitudes in Egyptian antipsychotic naive patients with schizophrenia. Compr Psychiatry. 2012;53(3):259-68. https://doi.org/10.1016/j.comppsych.2011.04.064.
14. Bulik-Sullivan B, Finucane HK, Anttila V, Gusev A, Day FR, Loh PR, Duncan L, Perry JR, Patterson N, Robinson EB, Daly MJ. An atlas of genetic correlations across human diseases and traits. Nat Genet. 2015;47(11):1236-41. https://doi.org/10.1038/ng.3406.
15. Lundgren JD, Rempfer MV, Brown CE, Goetz J, Hamera E. The prevalence of night eating syndrome and binge eating disorder among overweight and obese individuals with serious mental illness. Psychiatry Res. 2010;175(3):233-6. https://doi.org/10.1016/j.psychres.2008.10.027.
16. Anzengruber D, Klump KL, Thornton L, Brandt H, Crawford S, Fichter MM, Halmi KA, Johnson C, Kaplan AS, LaVia M, Mitchell J. Smoking in eating disorders. Eat Behav. 2006;7(4):291-9. https://doi.org/10.1016/j.eatbeh.2006.06.005.
17. García-Mayor RV, García-Soidán FJ. Eating disorders in Type 2 diabetic people: Brief review. Diabetes & Metabolic Syndrome: Diabetes Metab Syndr. 2017;11(3):221-4. https://doi.org/10.1016/j.dsx.2016.08.004.
18. Kouidrat Y, Amad A, Stubbs B, Louhou R, Renard N, Diouf M, et al. Disordered eating behaviors as a potential obesogenic factor in schizophrenia. Psychiatry Res. 2018;269:450-4. https://doi.org/10.1016/j.psychres.2018.08.083.
19. Kouidrat Y, Amad A, Renard N, Corneille F, Lalau JD, Loas G. Management of eating disorders in schizophrenia. Soins Psychiatr. 2016;(304):39-43. https://doi.org/10.1016/j.spsy.2015.04.011.
20. Burmeister E, Aitken LM. Sample size: How many is enough?. Aust Crit Care. 2012;25(4):271-4. https://doi.org/10.1016/j.aucc.2012.07.002.
21. Gargari BP, Khadem-Haghighian M, Taklifi E, Hamed-Behzad M, Shahraki M. Eating attitudes, self-esteem and social physique anxiety among Iranian females who participate in fitness programs. J Sports Med Phys Fitness. 2010;50(1):79-84.
22. Kaviani H, Mousavi AS. Psychometric properties of the Persian version of Beck Anxiety Inventory (BAI). Tehran University Medical Journal TUMS Publications. 2008;66(2):136-40. (Persian)
23. Ghassemzadeh H, Mojtabai R, Karamghadiri N, Ebrahimkhani N. Psychometric properties of a Persian-language version of the Beck Depression Inventory-Second edition: BDI-II-PERSIAN. Depress Anxiety. 2005;21(4):185-92. https://doi.org/10.1002/da.20070.
24. First MB, Williams JB, Karg RS, Spitzer RL. User's Guide for the SCID-5-CV: Structured Clinical Interview for DSM-5 Disorders, Clinician Version. Arlington: American Psychiatric Association; 2016.
25. Heshmati RM. Exploration of the Factor Structure of Positive and Negative Syndrome Scale in Schizophrenia Spectrum Disorders. Journal of Clinical Psychology. 2010;2:6. (Persian)
26. Delaney CB, Eddy KT, Hartmann AS, Becker AE, Murray HB, Thomas JJ. Pica and rumination behavior among individuals seeking treatment for eating disorders or obesity. Int J Eat Disord. 2015;48(2):238-48. https://doi.org/10.1002/eat.22279.
27. Domingo‐Claros A, Alonso E, Banda ED. Schizophrenia and refractory anaemia with ring sideroblasts. Br J Haematol. 2004;125(5):543. https://doi.org/10.1111/j.1365-2141.2004.04839.x.
28. Beck DA, Frohberg NR. Coprophagia in an elderly man: a case report and review of the literature. Int J Psychiatry Med. 2005;35(4):417-27. https://doi.org/10.2190/6EWR-M247-4W17-HNK1.
29. Mercier-Guidez E, Loas G. Polydipsia and water intoxication in 353 psychiatric inpatients: an epidemiological and psychopathological study. Eur Psychiatry. 2000;15(5):306-11. https://doi.org/10.1016/s0924-9338(00)00399-0.
30. Adetoki A, Evans R, Cassidy G. Polydipsia with water intoxication in treatment‐resistant schizophrenia. Progress in Neurology and Psychiatry. 2013;17(3):20-3. https://doi.org/10.1002/pnp.281.
31. Engelberg MJ, Steiger H, Gauvin L, Wonderlich SA. Binge antecedents in bulimic syndromes: An examination of dissociation and negative affect. Int J Eat Disord. 2007;40(6):531-6. https://doi.org/10.1002/eat.20399.
32. Bruch H. Eating disorders and schizophrenia. In: Usdin G, ed. Psychoneurosis and Schizophrenia. Philadelphia: Lippincott; 1966:113-124.
33. Rosenbaum DL, White KS. The relation of anxiety, depression, and stress to binge eating behav-

ior. J Health Psychol. 2015;20(6):887-98. https://doi.org/10.1177/1359105315580212.

34. Gautier JF, Del Parigi A, Chen K, Salbe AD, Bandy D, Pratley RE, Ravussin E, Reiman EM, Tataranni PA. Effect of satiation on brain activity in obese and lean women. Obes Res. 2001;9(11):676-84. https://doi.org/10.1038/oby.2001.92.
35. Essawy HI, Elghonemy SH, Mahmoud DA, Arafa AM. The relationship between disturbed Eating behavior and Substance abuse in an Egyptian sample. QJM. 2020;113(S1):hcaa054-013. https://doi.org/10.1093/qjmed/hcaa054.013.
36. Dixon LB, Dickerson F, Bellack AS, Bennett M, Dickinson D, Goldberg RW, Lehman A, Tenhula WN, Calmes C, Pasillas RM, Peer J. The 2009 schizophrenia PORT psychosocial treatment recommendations and summary statements. Schizophr Bull. 2010;36(1):48-70. https://doi.org/10.1093/schbul/sbp115.
37. Cook JA, Copeland ME, Jonikas JA, Hamilton MM, Razzano LA, Grey DD, Floyd CB, Hudson WB, Macfarlane RT, Carter TM, Boyd S. Results of a randomized controlled trial of mental illness self-management using Wellness Recovery Action Planning. Schizophr Bull. 2012;38(4):881-91. https://doi.org/10.1093/schbul/sbr012.
38. Sallemi R, Hentati S, Feki I, Masmoudi J, Moala M. Eating disorders in schizophrenia. Eur Psychiatry. 2017;41:S284.
39. Mukundan A, Faulkner G, Cohn T, Remington G. Antipsychotic switching for people with schizophrenia who have neuroleptic‐induced weight or metabolic problems. Cochrane Database Syst Rev. 2010(12). https://doi.org/10.1002/14651858.CD006629.pub2.
40. Khalil RB, Hachem D, Richa S. Eating disorders and schizophrenia in male patients: a review. Eat Weight Disord. 2011;16(3):e150-6. https://doi.org/10.1007/bf03325126.
41. Yum SY, Hwang MY, Halmi KA. Eating disorders in schizophrenia. Psychiatr Times. 2006;23(7):10.