Weight gain and obesity in general adult psychiatric inpatients: a longitudinal and cross-sectional study

Aumento di peso e obesità nella popolazione adulta in ospedali psichiatrici: uno studio longitudinale e trasversale

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SUMMARY. Background. Weight gain and obesity are significantly linked to mental illness. There have been different theories trying to explain weight gain to psychiatric inpatients, such as physical inactivity and lifestyle, the effect of psychotropic drugs, increased food intake triggered by depression, and comorbidity between mental illness and obesity. The current research is a longitudinal and cross-sectional study collecting the electronic records of weight of psychiatric inpatients in a period spanning from one to ten years to address these theories. **Methods.** We collected the electronic records relative to weight measurement that are conducted weekly and relative to 240 non-forensic psychiatric inpatients (124 males and 116 females) and for a period from 1 to 10 years. Mean ages for males was 39.65 years (SD=±11.66) and females 40.88 years (SD=±13.73). They accessed a psychiatric inpatient service in the United Kingdom. The coefficient of determination R2 calculated the time variation in bodyweight in the period span, while the Chi-square statistic evaluated the differences in outcomes. **Results.** Our longitudinal study shows that R2=0.17 (95% CI=0.14-0.20) for males and 0.27 (95% CI=02.0-0.34) for females. There was a statistically significant difference between the R2 (χ^2: $p<.05$) for both genders. The average Body Mass Index (BMI) for male psychiatric inpatients was 27.05 (SD=±5.92), corresponding to WHO Overweight Class. The average BMI for female psychiatric inpatients was instead 31.21 (SD=±7.73), corresponding to WHO Obesity Class I. The difference in BMI was statistically significant for both genders (χ^2: p<.001). **Discussion.** In our study, only 27% of the difference in body weight in females and 17% in males was explainable by the time variable with a small to moderate effect size. Our findings appear to support the theory that overweight and morbid obesity might be comorbid with psychiatric illnesses and independent from the therapeutic regimen. Overall, females' BMI is more pathological. **Conclusion.** During lengthy admissions, only modest changes in body weight were observed in our research. Our findings would suggest that metabolic syndrome and therefore elevated BMI, overweight, and obesity might be comorbid with psychiatric illnesses and might be independent of the length of admissions.

KEY WORDS: obesity, weight gain, psychiatry, inpatients, Body Mass Index.

RIASSUNTO. Scopi. L'aumento di peso e l'obesità sono significativamente legati alle malattie mentali. Ci sono state diverse teorie che cercano di spiegare l'aumento di peso nei pazienti psichiatrici come l'inattività fisica e lo stile di vita, l'effetto dei farmaci psicotropi, l'aumento dell'assunzione di cibo innescato dalla depressione e la comorbilità tra malattia mentale e obesità. La ricerca in corso è uno studio longitudinale e trasversale che raccoglie le registrazioni elettroniche del peso dei pazienti psichiatrici in un periodo che si estende da uno a dieci anni. **Metodi.** Abbiamo raccolto le registrazioni elettroniche relative alla misurazione del peso che vengono condotte settimanalmente e relative a 240 pazienti ricoverati psichiatrici non forensici (124 maschi e 116 femmine) e per un periodo da 1 a 10 anni. L'età media dei maschi era di 39,65 anni (DS=±11,66) e delle femmine di 40,88 anni (DS=±13,73). Tali pazienti hanno avuto accesso a un servizio di degenza psichiatrica nel Regno Unito. Il coefficiente di determinazione R2 ha calcolato la variazione nel tempo del peso corporeo nell'intervallo di tempo, mentre la statistica del test Chi-quadrato ha valutato le differenze nei risultati. **Risultati.** Il nostro studio longitudinale mostra che R2=0,17 (95% CI=0,14-0,20) per i maschi e 0,27 (95% CI=02,0-0,34) per le femmine. Non vi era alcuna differenza statisticamente significativa tra R2 (χ^2: p<0,05) in entrambi i sessi. L'indice di massa corporea medio (IMC) per i pazienti psichiatrici maschi era di 27,05 (SD=±5,92), corrispondente alla classe di sovrappeso dell'OMS. L'IMC medio per i pazienti psichiatrici femminili era invece di 31,21 (SD=±7,73), corrispondente alla classe di obesità dell'OMS I. La differenza di IMC è stata statisticamente significativa per entrambi i sessi (χ^2: p<0,001). **Discussione.** Nel nostro studio, solo il 27% della differenza nel peso corporeo nelle femmine e il 17% nei maschi era spiegabile dalla variabile di tempo con una minima dimensione effetto. Pertanto i risultati della ricerca sembrano confermare la teoria che l'obesità sovrappeso e morbosa potrebbe essere comorbile con malattie psichiatriche e indipendenti dal regime terapeutico o dallo stile di vita. In generale, l'IMS nelle pazienti femmine è quello più patologico. **Conclusione.** Durante lunghe ammissioni, nella nostra ricerca sono stati osservati solo modesti cambiamenti sostanziali nel peso corporeo. I nostri risultati suggerirebbero che la sindrome metabolica e quindi l'IMC elevato, il sovrappeso e l'obesità sono in comorbilità con malattie psichiatriche e sono indipendenti dalla durata delle ammissioni.

PAROLE CHIAVE: obesità, aumento di peso, psichiatria, pazienti ricoverati, indice di massa corporeo.

INTRODUCTION

Obesity is associated significantly with mental health illnesses[1]. In psychiatric-hospital patients, the rate of overweight and obesity hits 66%, with the tendency for female patients to raise their weight during their admissions instead of males who normally enter a stable state[2,3]. In the population detained inside mental health protected units, obesity and overweight (with rates of up to 80 percent reported) are more common than in the general population (about 60 percent), and patients tend to be at risk of weight gain while detained[4]. The UK National Obesity Observatory suggests a bidirectional aspect of the problem, where obesity can cause psychiatric illnesses or where mental health illnesses can cause obesity[5]. Theories that link obesity to mental illnesses can be summarized into four major clusters. The first hypothesis suggests that psychiatric patients' physical inactivity and lifestyle explain their elevated BMI (Body Mass Index)[6,7]. The second theory hypothesizes that psychotropic drugs are accountable for weight gain, especially SSRI (Serotonin System Reuptake Inhibitor) antidepressants, and antipsychotics[8,9]. Some authors suggest a mixture of these aspects, where patients decrease physical activity, and via psychotropic medicines and augmented appetite, gain more weight due to some preference for carbohydrates[10]. The third hypothesis suggests that some form of diet is accountable for psychological disorders, which may lead to depression, in particular, elevated intake of fast food and baked goods[11]. Studies in mice show a neuroinflammatory effect of a high-fat diet[12]. The fourth theory postulates that pathological weights are comorbid, particularly in female patients, with psychiatric diseases[13,14]. The prevalence of women with the diagnosis of borderline personality disorder in the general adult inpatient population[15], and the correlation between borderline personality disorder and obesity[16], can partly elucidate why morbid weight is mostly discovered in the female psychiatric population with borderline personality disorder[17]. Besides, a general association has been discovered between psychiatric illnesses, metabolic syndrome, and overweight[18]. Research shows that augmented BMI is related to reduced brain size with axonal/myelin irregularities in the white matter, with a decrease in the grey matter and consequent damage to neurons[19]. Preliminary experiments in mice indicate that high-fat diets change the mice's intestinal microbiome while interfering with their neuro-biology, including elevated anxiety, erratic behavior, and diminished memory[12]. One proposal to overcome these effects is to use non-steroidal anti-inflammatory drugs and omega-3 fatty acids to stimulate the development of beneficial intestinal bacteria in patients at risk of high BMI and psychiatric disorders[20], thus having beneficial effects on mental health[21]. Research that has examined the impact of obesity on the brain proposes that increased body weight is linked to a reduced brain size of the hippocampus, parietal, and frontal lobes[22]. Compared to a healthy population, persons with obesity show reduced volumes of the grey substance (frontal, temporal gyri, thalamus, and hippocampus) and white substance (internal capsule and optic radiation)[23].

Research involving 896 patients showed that obesity was associated with reduced cognitive functioning relative to non-obese patients, while patients with obesity and schizophrenia had poorer scores[24]. One systematic review found that the most affected cognitive abilities in obesity are psychomotor performance, speed, and time underestimation[25]. Another study supports the co-occurrence of causes that increase body weight in psychiatric hospital patients, such as extended sleep hours, alcohol, incorrect diets, antipsychotics; however, a link between weight change and admission period was not identified in the same research[26]. While diet instruction, sports syllabi, health awareness groups, open gym, hikes, and outdoor cycling are the most embraced interventions to counteract weight gain in psychiatric hospital patients, one study found that these initiatives were only relatively successful in 55 percent and unsuccessful in 25 percent of cases[27].

Previous findings by the current study investigators indicate a prevalence of women with borderline personality disorder in adult psychiatric hospital patients, proposing that this demographic group could be the one with the largest number of extreme overweight and obesity findings[15,17]. The theory indicating that regulating impulsive feeding problems could be responsible for obesity in psychiatric hospitals appears to be verified in one study[28]. Concerning age, one study found no association between weight gain and age in patients who showed an association of hypertension and diabetes with psychotropic medication[29]. It is understood that psychotropic medications raise the weight, although certain drugs are also responsible for an increased incidence of type 2 diabetes and hypertension, such as olanzapine[29]. Second-generation antipsychotics (SGAs), particularly olanzapine and clozapine, have been linked with adverse metabolic syndrome and, most importantly, type 2 diabetes[30]. However, one theory indicates that glucose metabolism dysfunction and clozapine and olanzapine hyperinsulinemia are both associated with augmented antipsychotic action; thus, it is speculated that the therapeutic and metabolic actions of clozapine and olanzapine may be compatible while functioning on insulin brain receptors[31]. Obesity itself is correlated, from a wider viewpoint, with a higher incidence of hospitalizations in studies documenting prevalence in various countries and a comparable prevalence in general medical wards and psychiatric wards, with a mean BMI of 25.4 kg/m^2 for males and females[32]. Patients at higher risk are female psychiatric patients with bipolar disorder or unipolar depression[33,34].

However, the same incidence of obesity impacting the same catchment area population may be present in psychiatric inpatients. A National Health Service study in the UK shows that 58% of women and 68% of men were overweight or obese in 2015, while two out of three admissions had a primary diagnosis of obesity and 74% were female[35]. Our new study reveals a prevalence of admissions of females of white ethnic origin with borderline personality disorder in general adult psychiatric wards[17]. The prevalence of extreme obesity in these patients also is high[36]. In 26.9% of the obese population, a US report found a prevalence of borderline personality disorder that ties personality disorder to compulsive eating[16]. Research shows that child abuse in women is linked later in life with obesity[37]. Studies also correlate depression with obesity. For example, obese teenagers have a greater prevalence of neurological and psychological disorders than teenagers with a stable BMI, including bad academic records, decreased self-esteem, depression, and higher suicide risk[38]. In general, major depressive disorder is associated with a greater risk of obesity and is likely to be associated with ele-

vated appetite and sleep[39]. The relation between obesity and chronic depression has been linked to reduced hippocampus volume[40]. Metabolism and weight problems arise separately from psychotropic treatment in subjects with psychosis[41].

In comparison, depression and obesity can already be conducive to brain modifications in the early stages of psychosis[42]. The theory is that high BMI in schizophrenia induces alterations of the cerebral white matter by disrupting neurotransmission in the cortico-limbic networks that play a key role in schizophrenia for neurocognition, impulses, and mental wellbeing[43]. A connection between obesity and bipolar disorder sharing the same causes, along with the obesogenic activity of mood stabilizers, has also been identified in research[44]. Another research showed that increased BMI in adolescents with bipolar disorder is related to the volume of the frontal cortical lobes[45]. The overall study indicates that obesity is linked with schizoaffective disorder, marked by a history of major depressive disorder and suicidal attempts[46].

OBJECTIVES

There are no general and longitudinal studies to show if weight gain is already present when patients access psychiatric services or if, instead, it could be attributed to the theories mentioned above, mainly the effect of psychotropic medication, altered diet, and reduced physical activity. In the United Kingdom, data relative to psychiatric patients, including body weight, are commonly recorded on an electronic database. Consequently, longitudinal data collection using electronic support can improve research outcomes[47]. Electronic medical records (EMR) offer a chance for useful and extensive scientific research in psychiatry[48]. The research hypothesis is to verify if weight gain is linked to the time factor. The research objective was to collect longitudinal data of the non-forensic psychiatric population for a period spanning between one to ten years, corresponding to the creation of electronic data in participating clinical centers.

POPULATION AND METHODS

This current one is a longitudinal and cross-sectional study relative to a period spanning from one to ten years. The sample consisted of 240 non-forensic psychiatric inpatients (124 males and 116 females) from the general adult population with the mean age for males of 39.65 years (SD =±11.66) and females of 40.88 years (SD=±13.73), accessing several psychiatric services in the United Kingdom. Nurses routinely and electronically store data relative to body weight during weekly measurements, relative to each patient. A researcher collected these data on a spreadsheet and relative to a period covering 1 to 10 years for the sample observed. The cross-sectional study averaged the global bodyweights for all patients. The coefficient of determination R2 calculated the time variation in bodyweight in the period span, while the Chi-square statistics evaluated the differences in outcomes. R2 measures the proportion of variation of the dependent variable (weight gain) justified by the variation of the independent variable or predictor (time)[49]. In our case, R2 also has a predictive meaning as it allows for the extrapolation of the correlation curve and makes some estimates on probable future behaviors of the dependent variable (weight). In the current research, patients' weight readings were collated into a spreadsheet, which automatically converted subsequent readings into R2 values. The BMI was calculated by applying the national standards for the height of the male (175.3 cm) and female (161.6 cm) residents in the United Kingdom[50]. Exclusion criteria were patients presenting with less than three weight observations. The software Meta-Excel by EpiGear calculated the statistical significance of χ^2 for the differences between genders given the outcome values and their 95% Confidence Intervals (95% CI)[51]. Daniel Soper (2021) online calculator computed the effect sizes (ES) f^2 for R2 (www.danielsoper.com). We used the UK National Health Service BMI Calculator for computing the body weights and BMI[52]. BMI is classified according to World Health Organization (WHO) international classification, in 'underweight' BMI less than 18.5, 'healthy weight' BMI between 18.5 and 24.9, 'overweight' BMI between 25 and 29.9, 'obesity class I' BMI between 30 and 34.9, 'obesity class II' BMI between 35 and 39.9, and 'obesity class III' BMI from 40 and over[53,54]. BMI scores were also condensed in a box-plot. The current research was conducted according to the conventions of the Declaration of Helsinki principles of 1975, as revised in 2008. Data were stored in encrypted files accessible only to the clinicians involved in the research. The anonymity of patients was maintained at all stages of the research.

RESULTS

Global results are shown in Table 1. Our longitudinal study shows that R2=0.17 (95% CI=0.14-0.20) for males and 0.27 (95% CI=0.20-0.34) for females. There was a statistically significant difference between the R2 (χ^2: $p<0.05$) between both genders (Table 1). The average BMI for male psychiatric inpatients was 27.05 (SD=±5.92), corresponding to WHO Overweight Class. The average BMI for female psychiatric inpatients was instead 31.21 (SD=±7.73), corresponding to WHO Obesity Class I. The difference in BMI was statistically significant for both genders (χ^2: $p<0.001$). BMI evenly distributed in all classes for females while concentrating on overweight for males (Table 1, Figure 1). Overall, the female psychiatric population's BMI tends to be higher than the BMI in the male psychiatric population and more dispersed within all BMI categories. When body weights were compared, 44% of males were in the normal weight limits compared to 22% of females who, instead, were obese in 30% of cases and 20% of cases in Obesity Class II. Weight differences between males and females were statistically significant (χ^2=22.44; $p<0.05$). Statistically significant differences in WHO bodyweight categories were for Normal Weight, with prevalence of males (44% vs. 22%; $p<0.01$), and Obesity Class II, with the prevalence of females (20% vs. 5%; p=0.015). BMI in females was more dispersed compared to males (Figure 1). ES f^2 was 0.20 for males and 0.36 for females.

DISCUSSION

In hospitalized psychiatric patients of our study, overweight in males and obesity in females were found. Also, incremental weight gain is more important during admissions in female patients than in male patients, while individual differences were greater in male patients than in female patients. In our study, however, only 27 percent of the difference in body weight in females and 17 percent in

Table 1. Outcomes of the research.

Type of study	Outcome	Subcategories	Males (N=124)	95%CI	Females (N= 16)	95%CI	Significance p of χ^2
Longitudinal:	R2		0.17	0.14-0.20	0.27	0.20-0.34	<.05
Cross-sectional:	BMI		27.1 (±5.92)	26.05-28.14	31.9 (±7.73)	30.49-33.30	<.001
	Body weight	Normal weight	55 (44%)	35%-52%	25 (22%)	14%-29%	<.01
		Over-weight	36 (29%)	21%-37%	34 (30%)	21%-38%	n.s.
		Obesity class I	21 (17%)	10%-23%	16 (14%)	7%-20%	n.s
		Obesity class II	6 (5%)	1%-8%	22 (20%)	12%-27%	.015
		Obesity class III	6 (5%)	1%-8%	19 (15%)	8%-21%	n.s.
							Total for Body weight: p<.05

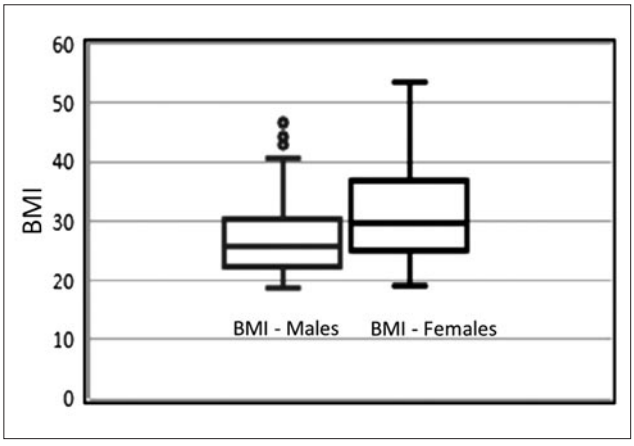

Figure 1. Box-plots for the global BMI of the female psychiatric inpatients shows global higher values than the BMI in the male psychiatric inpatients. In the female population, the average BMI is higher than males' values while the BMI scores are more dispersed than in the male population.

males was explainable by the time variable. The ES of the time variable was small for males and moderate for females. Similarly, meaningful variation was observed in the mean weights for both male and female patients. Therefore, during long admissions, only a small to moderate change in body weight could be observed in our research. Psychiatric hospitals in the UK have been introducing interventions to optimize body weight. For example, with psychotropic drugs' wise procurement, many hospitals have specific strategies to influence body weight. One hypothesis from our research is that metabolic syndrome and elevated BMI, overweight, and obesity might be comorbid with psychiatric diseases and could also be difficult to treat. The theory of the relationship between obesity and mental health, implying a shared effect, would support these observations. In either cases, it is beneficial to reduce the effect of a metabolic syndrome caused by abnormal BMI in the general adult psychiatric community by promoting a dramatic shift in food habits. Besides, while physical fitness policies exist and are facilitated, in psychiatric wards, rare gyms plus small physical spaces make obesity epidemic in psychiatric hospital inpatients. Eventually, to cope with anxieties or heightened drowsiness, often, patients use comfort feeding and energy food, and this activity will make obesity comorbid but not a cause of mental illnesses. The utilization of energy drinks is common in psychiatric inpatients who try to balance the over-sedation from medication. Besides, the findings that relate the current analysis to previous research by the same authors are that the category of psychiatric hospital patients primarily affected by obesity is the borderline female patients. They can use impulsive eating to relieve mental turmoil or as a means of self-harm, often presenting with other types of eating disorders such as anorexia and bulimia. Preliminary research indicates that given the association of high BMI and depression, an improved metabolic state through the normalization of obesity shall also affect the underlying depression[55]. Obesity is correlated with a rise in the risk of mood and anxiety disorders by about 25 percent and a reduction in the probability of substance use disorders by about 25 percent[56]. There is an important and bidirectional link between obesity and depression; for anxiety disorders, the data is modest and insufficient for most medical problems; in these relations, gender seems to be a significant mediator[57]. These data appear to confirm our initial hypothesis that the most affected by morbid obesity are female psychiatric patients with emotionally unstable personality disorder and dysthymia. Nonetheless, as there is an insignificant correlation between weight gain and hospitalization time, the likely hypothesis is that this fringe population suffering from a mood disorder and borderline personality disorder has obesity as a comorbid condition.

The current study shows several limitations. One weakness is that the population's entry and exit weight could have shown higher variations than the longitudinal study. Besides, when first becoming inpatients, the sample might have start-

ed to assume psychotropic medication that affects body weight and then recorded at the first admission. Therefore, further research is needed to verify if pathogenic factors act individually or consensually in psychiatric inpatients, suggesting a multifactorial cause for pathological weight-gain.

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REFERENCES

1. Lin HY, Huang CK, Tai CM, et al. Psychiatric disorders of patients seeking obesity treatment. BMC Psychiatry 2013; 13: 1-8.
2. Long C, Rowell A, Gayton A, Hodgson E, Dolley O. Tackling obesity and its complications in secure settings. Mental Health Review Journal 2014; 19: 37-46.
3. Public Health England PH. Obesity in mental health secure units [Internet]. GOV.UK; 2017 [cited 2021 Jan 10]. Available from: https://bit.ly/2UvAmt1
4. Day M, Maxine J. Working together to address obesity in adult mental health secure units. A systematic review of the evidence and a summary of the implications for practice. London: Public Health England, 2017. Available from: https://bit.ly/3zPKkFS
5. Gatineau M, Dent M. Obesity and mental health. Oxford: National Obesity Observatory and National Health Service (NHS) 2011. Available from: https://bit.ly/2Ulsc6e
6. Lopresti AL, Drummond PD. Obesity and psychiatric disorders: commonalities in dysregulated biological pathways and their implications for treatment. Prog Neuropsychopharmacol Biol Psychiatry 2013; 45: 92-9.
7. Hilton NZ, Ham E, Lang C, Harris TG. Weight gain and its correlates among forensic inpatients. CJP 2015; 60: 232-8.
8. Gebhardt S, Haberhausen M, Heinzel-Gutenbrunner M, et al. Antipsychotic-induced body weight gain: predictors and a systematic categorization of the long-term weight course. J Psychiatr Res 2009; 43: 620-6.
9. Uguz F, Sahingoz M, Gungor B, Aksoy F, Askin R. Weight gain and associated factors in patients using newer antidepressant drugs. Gen Hosp Psychiatry 2015; 37: 46-8.
10. Kucuk L, Kaya H, Comez T, Kacar S, Kutlu Y, Zulfikar H. Eating behaviors and related factors in psychiatric patients. Arch Psychiatr Nurs 2018; 32: 194-9.
11. Sánchez-Villegas A, Toledo E, de Irala J, Ruiz-Canela M, Pla-Vidal J, Martínez-González MA. Fast-food and commercial baked goods consumption and the risk of depression. Public Health Nutr 2012; 15: 424-32.
12. Bruce-Keller AJ, Salbaum MJ, Luo M, et al. Reply to: high-fat diet-induced dysbiosis as a cause of neuroinflammation. Biol Psychiatry 2016; 80: e5-e6.
13. Sholtz S, Morgan JF. Obesity and psychiatry. Psychiatry 2009; 8: 198-202.
14. Dickerson FB, Brown CH, Kreyenbuhl JA, et al. Obesity among individuals with serious mental illness. Acta Psychiatr Scand 2006; 113: 306-13.
15. Lazzari C, Shoka A, Papanna B, Kulkarni K. Predominant diagnoses, gender, and admission duration in an adult psychiatric inpatient hospital in United Kingdom. OJPAS 2017; 9: 37-40.
16. Sansone RA, Sansone LA. The relationship between borderline personality and obesity. Innov Clin Neurosci 2013; 10: 36-40.
17. Lazzari C, Shoka A, Papanna B, Rabottini M. Long-term weight gain and prevalence of obesity in general adult psychiatric inpatients. America Journal of Psychiatry and Neuroscience 2018; 6: 86-94.
18. Rajan TM, Menon V. Psychiatric disorders and obesity: a review of association studies. JPGM 2017; 63: 182-90.
19. Marques C, Meireles M, Faria A, Calhau C. High-fat diet-induced dysbiosis as a cause of neuroinflammation [Letter to the editor]. Biol Psychiatry 2016; 80: e3-e4.
20. Peet M, Stokes C. Omega-3 fatty acids in the treatment of psychiatric disorders. Drugs 2005; 65: 1051-9.
21. Bruce-Keller AJ, Keller JN, Morrison CD. Obesity and vulnerability of CNS. Biochim Biophys Acta 2009; 1792: 395-400.
22. Hidese S, Ota M, Matsuo J, et al. Association of obesity with cognitive function and brain structure in patients with major depressive disorder. J Affect Disorders 2018; 225: 188-94.
23. Monda V, La Marra M, Perrella R, et al. Obesity and brain illness: from cognitive and psychological evidences to obesity paradox. Diabetes Metab Syndr Obes 2017; 10: 473-9.
24. Guo X, Zhang Z, Wei Q, Lv H, Wu R, Zhao J. The relationship between obesity and neurocognitive function in Chinese patients with schizophrenia. BMC Psychiatry 2013; 13: 1-6.
25. Prickett C, Brennan L, Stolwyk R. Examining the relationship between obesity and cognitive function: a systematic literature review. Obes Res Clin Pract 2015; 9: 93-113.
26. Huthwaite M, Elmslie J, Every-Palmer S, Grant E, Sarah Romans SE. Obesity in a forensic and rehabilitation psychiatric service: a missed opportunity? Journal of Forensic Practice 2017; 19: 269-7.
27. Haw C, Jean Stubbs J. What are we doing about weight management in forensic psychiatry? A survey of forensic psychiatrists. The British Journal of Forensic Practice 2011; 13: 183-90.
28. Deux N, Schlarb AA, Martin F, Holtmann M, Hebebrand J, Legenbauer T. Overweight in adolescent, psychiatric inpatients: a problem of general or food-specific impulsivity. Appetite 2017; 112: 157-66.
29. Kisely S, Cox M, Campbel LA, Cooke C, Gardner D. An epidemiologic study of psychotropic medication and obesity-related chronic illnesses in older psychiatric patients. Can J Psychiatry 2009; 54: 269-74.
30. Hirsch L, Yang J, Bresee L, Jette N, Patten S, Pringsheim T. Second-generation antipsychotics and metabolic side effects: a systematic review of population-based studies. Drug Saf 2017; 40: 771-81.
31. Raben AT, Marshe VS, Chintoh A, Gorbovskaya I, Muller DJ, Hahn MK. The complex relationship between antipsychotic-induced weight gain and therapeutic benefits: a systematic review and implications for treatment. Front Neurosci 2018; 11: 741.
32. Folling IS, Kulseng B, HelviK A-S. Overweight, obesity and related conditions: a cross-sectional study of adult inpatients at a Novergian hospital. BMC Research Notes 2014; 7: 1-6.
33. Hussain T, Margoob MA, Shoib S, Shafat M, Chandel KR. Prevalence of metabolic syndrome among psychiatric inpatients: a hospital-based study from Kashmir. J Clin Diagn Res 2017; 11: VC05-VC08.
34. Santini I, Stratta P, D'Onofrio S, et al. The metabolic syndrome in an Italian psychiatric sample: a retrospective chart review of inpatients treated with antipsychotics. Riv Psichiatr 2016; 51: 37-42.
35. National Health Service Digital (NHS Digital). Statistics on obesity, physical activity and diet. 2017. Available from: https://bit.ly/2Uq5F8p
36. Powers AD, Oltmanns TF. Borderline personality pathology and chronic health problems in later adulthood: the mediating role of obesity. Personal Disord 2013; 4: 152-9.
37. Danese A, Tan M. Childhood maltreatment and obesity: systematic review and meta-analysis. Mol Psychiatr 2014; 19: 544-54.

38. Nemiari D, Shim R, Mattox G, Holden K. The relationship between obesity and depression among adolescents. Psychiatr Ann 2012; 42: 3058.
39. Polanka BM, Vrany EA, Patel J, Stewart JC. Depressive disorder subtypes as predictors of incident obesity in US adults: moderation by race/ethnicity. Am J Epidemiol 2017; 185: 734-42.
40. Nigatu YT, Bultmann U, Reijneveld SA. The progressive association between obesity and major depression in the general population: does single or recurrent episode matter? BMC Public Health 2015; 15: 1-8.
41. Oreši M. Obesity and psychotic disorders: uncovering common mechanisms through metabolomics. Dis Model Mech 2012; 5: 614-20.
42. Kolenic M, Frankec K, Hlinka J, et al. Obesity, dyslipidemia and brain age in first-episode psychosis. J Psychiat Res 2018; 99: 151-8.
43. Spangaro M, Mazza E, Poletti S, Cavallaro R, Benedetti F. Obesity influences white matter integrity in schizophrenia. Psychoneuroendocrinology 2018; 97: 135-42.
44. Zhao Z, Okusaga OO, Quevedo J, Soares JC, Teixeira AL. The potential association between obesity and bipolar disorder: a meta-analysis. J Affect Disorders 2016; 202: 120-3.
45. Islam AH, Metcalfe AWS, MacIntosh BJ, Korczak DJ, Goldstein, BI. Greater body mass index is associated with reduced frontal cortical volumes among adolescents with bipolar disorder. J Psychiatry Neurosci 2018; 43: 120-30.
46. Chouinard V-A, Pingali SM, Chouinard G, et al. Factors associated with overweight and obesity in schizophrenia, schizoaffective and bipolar disorders. Psychiatry Res 2016; 237: 304-10.
47. Udtha M, Nomie K, Yu E, Sanner J. Novel and emerging strategies for longitudinal data collection. J Nurs Scholarsh 2015; 47: 152-60.
48. Perlis RH, Iosifescu DV, Castro VM, et al. Using electronic medical records to enable large-scale studies in psychiatry: treatment resistant depression as a model. Psychol Med 2012; 42: 41-50.
49. Zhang D. A coefficient of determination for generalized linear models. Am Stat 2017; 71: 310-6.
50. British Broadcasting Corporation (BBC). Statistics reveal Britain's Mr and Mrs Average. 3 October 2010. Available from: https://bbc.in/3gVpegn
51. Barendregt J. Meta-Excel [Internet]. MetaXL. [cited 2021 Jan 13]. Available from: https://bit.ly/3zPQ2Yk
52. National Health Service. BMI Calculator. 2018 [Online] Available from: https://bit.ly/3gMKshO
53. World Health Organization. Physical status: the use and interpretation of anthropometry – report of a WHO Expert Committee. WHO Technical Report Series No. 854. Geneva: World Health Organization, 1995.
54. Raben AT, Marshe VS, Chintoh A, Gorbovskaya I, Müller DJ, Hahn MK. The complex relationship between antipsychotic-induced weight gain and therapeutic benefits: a systematic review and implications for treatment. Front Neurosci-Switz 2018; 11: 741.
55. Opel N, Redlich R, Grotegerd D, et al. Obesity and major depression: body-mass index (BMI) is associated with a severe course of disease and specific neurostructural alterations. Psychoneuroendocrinology 2015; 51: 219-26.
56. Simon GE, Korff MV, Saunders K, et al. Association between obesity and psychiatric disorders in the US adult population. Arch Gen Psychiatry 2006; 63: 824-30.
57. Rajan TM, Menon V. Psychiatric disorders and obesity: a review of association studies. J Postgrad Med 2017; 63: 182-90.