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20-year progression of BMI in a county-wide cohort people with schizophrenia and bipolar disorder identified at their first episode of psychosis

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Abstract

Objective—There is increased prevalence of obesity in schizophrenia and bipolar disorder, leading to disproportionate risk of adverse health conditions. Prospective, long-term weight gain data, however, are scarce.

Methods—We analyzed data from the Suffolk County Mental Health Project cohort of consecutive first admissions with psychosis recruited from September 1989 to December 1995 and subsequently followed for 20 years, focusing on people with schizophrenia (n=146) and bipolar disorder (n=87). The time course of weight gain was examined using a 2 (group) × 5 (time) mixed-model repeated measures ANOVA, and body mass index (BMI) scores at the first (6 months) and second (2 years) assessments were compared to examine whether early overweight predicted later obesity.

Results—There was a statistically significant effect of time, $F(1,210)=68.06$, $p<.001$, and diagnosis on BMI, $F(1,210)=29.18$, $p<.001$, but not the interaction of time × diagnosis, $F(1,210)=0.88$, $p=.48$. Most participants had normal BMIs at the first two assessments. Early overweight was a predictor of eventual obesity for both groups. At the 20 year follow-ups, approximately 50% of the bipolar and 62% of the schizophrenia sample were obese, with greater prevalence of obesity in schizophrenia at each assessment (all $p<.02$), except for years 4 ($p=.12$) and 20 ($p=.27$).

Conclusions—Nearly two thirds of the participants with schizophrenia and over half with bipolar disorder were obese 20 years after first hospitalization for psychosis, considerably higher than the rate for adults in New York State (27%). Early intervention may be required to prevent long-term consequences of obesity-related morbidity and mortality.

Introduction

Obesity is a major public health problem in the United States (US), with an estimated 35% of the population considered to be obese (BMI over 30 kg/m²) (1). Obesity has significant negative impacts on morbidity and mortality. Obesity increases the risk of adult onset type 2 diabetes, stroke, hyperlipidemia, coronary artery disease, hypertension and cancer (2). Moreover, obesity is associated with greater impairment in everyday activities, especially physical domains of functioning and disability (3). In addition, its economic impact on healthcare expenditures poses a substantial stress on the US healthcare system (4).

Individuals with both schizophrenia (SCZ) and bipolar disorder (BP) have an increased prevalence of obesity and related comorbidities compared to the general population (5), although slightly less common and severe in people with BP compared to SCZ (6, 7, 8). Individuals with both diagnoses are at increased risk of metabolic comorbidities (9, 10, 11, 12), including dyslipidemia, insulin resistance and hyperglycemia (13, 14), as well as cardiovascular disease, diabetes, hypertension (15) and metabolic syndrome (16). The lifespan is 20 years shorter on average for SCZ and 10 years shorter for BP compared to population norms (17–21). The reasons for high rates of obesity and attendant medical comorbidities are manifold, extending beyond genetic vulnerability and pharmacological treatments offered (22, 23). In the context of socioeconomic challenges, persistent mental illness leads to particularly unhealthy lifestyles that include poor diets and little or no physical exercise (24, 25, 26, 27). Cognitive, symptomatic (i.e., negative and depressive symptoms), medication-induced extrapyramidal symptoms, and functional capacity limitations add challenges to lifestyle-relevant activities of daily living (ADLs) and instrumental ADLs (i.e., diet, exercise, shopping for food, medical care), creating a vicious cycle that exacerbates health and functional declines (28) with health risk amplified by low rates of medical screening, monitoring, and intervention across healthcare delivery systems (29, 30).

Most of our knowledge derives from cross-sectional or short-term studies of clinical samples. In fact, there is little evidence from long-term, prospective studies that sheds light on the extent and timing of weight gain in SCZ and BP. This gap leads to an inability to pinpoint the critical time periods that would lend themselves to prevention efforts. Further, if the diagnostic-specific long-term correlates of weight gain differ for SCZ and BP, it would suggest the need for differential timing and tailoring of prevention and intervention efforts.

In this paper, we present data from a unique 20-year prospective follow-up of a county-wide sample of first-admission patients hospitalized with a psychotic condition in the early 1990s. In-person follow-ups occurred at 6 months, and at 2, 4, 10, and 20 years after their first admission; telephone contacts occurred in between these time points. Self-reported weights were obtained at each follow-up. Further, the 20 year assessment included systematic measurement of height and weight along biomarkers for determining metabolic abnormalities.

Our analysis focuses on the course of weight gain from the initial follow-up to the 20 year point and the correlates of weight at the 20-year assessment. Weight gain was defined by a

standard measure of body mass index (BMI). We compared study participants with SCZ and BP, examining changes over the course of 20 years and associated outcomes. To our knowledge this is the first study comparing the long-term course of weight gain across different psychotic disorders. In addition to comparison of participants with SCZ and BP, we reference their weight to population normative standards at approximately the same age that these measurements were taken (Figure 1.) in order to assess the relative excess in the study sample (31).

Methods

The sample is part of the Suffolk County Mental Health Project, a cohort of consecutive first admissions with a psychotic disorder to 12 psychiatric facilities in Suffolk County, NY, ascertained between September 1989 and December 1995. For a detailed description of sample selection and retention, see Bromet et al. (32, 33).

All participants gave written informed consent at the initial and each subsequent assessment, with Stony Brook University IRB approvals throughout. Baseline approvals were granted by each assessment site. Written parental approval and verbal approval were obtained for patients in the 15–17 year age range at study entry. Inclusion criteria at study entry were age 15–60 years of age, residence in Suffolk County NY, and psychosis; exclusion criteria were psychiatric hospitalization more than 6 months before the index admission, more than moderate mental retardation (now intellectual disability), and non-English speaking.

The Structured Clinical Interview for DSM-III-R was administered at baseline, and at relevant follow up visits (34). On the basis of the SCID interviews, medical records information, and interviews with the subjects' relatives, DSM-IV consensus diagnoses were reached for each participant. For the current analysis, we used the 10-year follow up diagnosis, the most recent diagnosis on file, and we selected participants with schizophrenia/schizoaffective disorder (SCZ) (n=146) and bipolar disorder (BP) (n=87). We excluded patients with psychosis related to depression, substance use, and 'other' psychoses. A flowchart of participation in the larger study is available; attrition was random, that is, the number of assessments was not associated with age, sex, negative symptoms, positive symptoms, employment, independence in living, homelessness, or baseline diagnosis. Nonresponse was primarily accounted for by refusal to participate and loss to follow up. (35).

Weight and Height Measurements

Weight and height were self-reported except for the 20 year assessment when height and weight were measured. Internal consistency for self-reported height vs. measured height was very high and no significantly different in the SCZ and bipolar group. Height based on self-report in the combined sample at year 10 and measured height at year 20 were highly intercorrelated (N=122, The body weight data were collected at 6 months, and 2, 4, 10, and 20 years after the index assessment. mass index (BMI) score for each participant was calculated at each assessment (kg/m^2). unding constraints impacted the frequency of assessments..

Other measures and biomarkers at the 20 year follow-up

Biomarkers collected include blood pressure, cholesterol (HDL, LDL, VLDL), triglycerides, hemoglobin A1c, body fat percentage, and waist circumference. Blood pressure was taken three times and averaged. We further derived a mean pulse pressure from these measurements.

Statistical Analyses

We examined the time course of BMI using a 2 (group) $\times$ 5 (time) mixed-measures repeated measures analysis of variance (MMRM ANOVA). We compared BMI scores at each assessment across the diagnoses using regular t-tests. Following those analyses, we characterized all patients as normal weight (BMI 24.9 or less), overweight (BMI 25–29.9), and obese (BMI $\geq$ 30) at each of the assessments and examined the prevalence of overweight and obesity at each assessment by diagnosis. We examined the pattern of development of obesity in participants who were overweight early in the course (i.e., either 6 months or 2 years), also as a function of diagnosis. Participants were divided into those receiving antipsychotic medication or not at each follow-up, and their subsequent weight gains were compared. We then examined the correlates of BMI at the 20 year mark in order to identify the factors that were and were not associated with increased weight. Finally, we explored weight gain trajectories in view of changing prescription patterns for antipsychotics: patients enrolled in the study from 1989 to 1992 could initially only have received first generation antipsychotics, whereas those enrolled from 1993 and later may have been exposed to second generation antipsychotics.

Results

Descriptive characteristics on the patients are presented in Table 1. The two groups (SCZ $n=146$; BP $n=87$) were comparable in terms of age and sex but the SCZ group had significantly poorer values on several of the blood –based biomarkers. We next determined the changes across time in BMI for the two groups. There was a statistically significant effect of time, $F(1,210)=68.06$, $p<.001$, and diagnosis, $F(1,210)=29.18$, $p<.001$, but no interaction of time $\times$ diagnosis, $F(1,210)=0.88$, $p=.48$. There were no differences in weight gain for gender and race across every possible two point comparison post baseline (6 months, 2 years, 4 year, 10 year 20 year). The largest t value for gender effects on any weight gain variable was $-.75$, $p=.4$ and the largest t value for race effects on any weight gain variable was $.95$, $p=.34$.

There was a significant positive linear slope ($b=.588$, $p<.001$) and a significant negative quadratic slope ($b=-.012$, $p=.001$), suggesting a plateauing of weight gain between 10 and 20 years. Diagnostic status significantly predicted BMI at the start of illness for schizophrenia ($b=-1.845$, $p=.012$), but did not significantly predict the linear slope ($b=-.061$, $p=.622$), or quadratic slope ($b=.003$, $p=.675$) for SCZ and BP. T-tests revealed that the SCZ group had significantly higher BMIs than BP apart from year 4, although the differences were relatively small at each assessment (Table 2).

Figure 2 presents the proportion of cases with obesity at each of the assessments. The majority of individuals in both groups had normal weight at the first two assessments, with the proportion of BP with normal weight staying at two thirds (65.8%) for the first 10 years. Obesity was prevalent in approximately 50% of the SCZ sample at the 10 and 62.1% at the 20-year follow-up assessments, with the prevalence of obesity at 34.2% for BP at 10 years and 54% at 20. Mann-Whitney U tests found that obesity was more prevalent in SCZ at month 06 ($U=3629.5$; $p=0.019$), year 2 ($U=3075$; $p=0.035$), and year 10 ($U=4301$; $p=0.024$) but the groups did not differ at year 4 ($U=3061.5$; $p=0.12$) and the year 20 assessment ($U=3175$; $p=0.27$).

Figure 3 presents the prevalence of obesity over the follow-up period from years 2 to 20 for the subgroup of patients that were overweight at the first assessment (SCZ $n=43$; BP $n=22$). As can be seen in the figure, the prevalence of obesity at year 10 was 55.8 % in SCZ and 54.4% in BP, and almost equivalent at year 20 across the SCZ (60.5%) and BP participants (63.3%) who had been overweight at the first assessment. Mann-Whitney U tests found that amongst people overweight at the first assessment, SCZ patients were more likely to be obese at year 2 ($U=2358$; $p=0.035$) but not at any of the subsequent visits (all $p>0.17$).

Eighty percent or more of the SCZ group was receiving antipsychotic medications at each assessment, rendering an examination of the weight gain in non-medicated patients underpowered. In BP, 35% were receiving antipsychotic treatment at year 2, 44% were receiving this treatment at year 4, and 48% were receiving antipsychotics at year 10. Interestingly, when weight gain from years 2, 4, and 10 to year 20 were compared across patients receiving antipsychotic treatment at each assessment or not with paired t-tests, antipsychotics had no effect on weight in any of the comparisons (all $t<1.10$, all $p>.30$). With antipsychotic use as a covariate, diagnostic status still predicted BMI at the start of illness ($b=-1.772$, $p=.044$), but still did not predict the linear slope ($b=.199$, $p=.151$) or the quadratic slope ($b=-.007$, $p=.311$). Thus, both groups had a similar increase in weight gain over time which nearly plateaued at the 20 year assessment.

Exploring potential cohort effects of antipsychotics on weight gain due to lack of availability of second-generation antipsychotics at the beginning of the recruitment period did not reveal different weight gain trajectories: the gain was linear when time periods were considered and there were no time periods where, on a years of follow-up corrected basis, there were greater gains. In fact, during years 10–20 when the likelihood of being exposed to weight-gain causing drugs was greatest, there is evidence of a plateauing of weight gain.

Table 3 presents the correlations between BMI and the other indices of metabolic health at the 20-year follow-up assessment. We combined the two groups because there was no reason to believe that healthy correlates of obesity would vary by psychiatric diagnosis. Although cholesterol, triglycerides, hemoglobin A1c, and blood pressure were not correlated with BMI in the group as a whole, body fat and waist circumference were substantially correlated, with waist circumference sharing 74% of the variance with BMI and body fat sharing 40% of the variance, suggesting that a central obesity pattern was prevalent.

Discussion

Nearly two-thirds of participants with SCZ and over half of those with BP in this thoroughly characterized cohort were obese 20 years after their first hospitalization for psychosis. Early overweight predicted future weight gain: a large majority of people who were overweight at year 2 turned out to be obese at the 20-year mark. We also observed similar weight gain trajectories in SCZ and BP with slightly different start points. The SCZ group gained twice as much weight within the first ten years of diagnosis and had half as much weight gain in the subsequent 10 years, while those with BP experienced less weight gain during the initial 10 year period as compared to the subsequent 10 years. Moreover, it is quite possible that participants in the sample followed for 20 years will continue to gain weight even after the 20-year observation period, and we infer, continue to incur increasingly detrimental metabolic changes. Another 5 year observation period is currently underway, bringing the total follow up to 25 years. This additional observation period may help to answer some of these important questions.

Interestingly, the use of antipsychotic medication was not associated with longitudinal differences in the course of weight gain, nor was the type/availability of antipsychotic (first- vs second generation). In fact, during years 10–20 when the likelihood of being exposed to the group of weight-gain causing second-generation drugs was greatest, there is evidence of a plateauing of weight gain. Moreover, neither gender nor race were associated with different rates of weight gain post baseline. Our assessments of medication treatment are necessarily limited by the widely spaced follow-up intervals, and may have been further mitigated by the use of mood stabilizers in BP, and to a lesser degree in SCZ. However, short term data suggests substantial morbidity associated with certain treatments and most patients are switched across medications very frequently, making any inferences about the specific influences of different medications challenging, and over the long term, perhaps modestly useful at best.

General population obesity rates in the US for adults aged 40–59 years in 2016 was about 35% (36) and in New York State, the 8th leanest state in the US, 27% in 2014, the most recent data point available (37). Both the SCZ and BP groups had a higher prevalence of obesity. Relative to people with normal weight, all-cause mortality of obese persons is higher (38). We surmise the increased prevalence of obesity in SCZ and BP puts them at greater health and mortality risk than the population at large, consistent with previous observations (8; 9; 19–22). The trajectory of weight gain is also important. Obese persons with upward weight gain trajectories, even after controlling for demographic and socioeconomic characteristics, smoking status, limitations in activities of daily living, and a wide range of chronic illnesses, have even higher mortality risk than those with static obesity (39). This suggests increasing obesity as observed in our sample poses an increasingly substantive mortality threat even 20 years after treatment and later if their weight continues to increase, a fact that clinicians treating chronic patients need to keep in mind.

Waist circumference (WC) at year 20 was significantly higher in SCZ than BP. Women with SCZ had higher WCs than men or women with BP. In both BP and SCZ, BMI was more strongly correlated with WC than with % body fat, suggesting a central obesity pattern in

addition to suggesting that WC represented a reasonably accurate proxy measure for body fat distribution. Long term follow-up studies in mentally healthy people show that abdominal obesity is strongly associated with an increased risk of type 2 diabetes, cardiovascular disease and death, even after controlling for BMI (40, 41, 42). In line with their comparatively increased WC, individuals with SCZ were significantly more metabolically compromised at the 20 year follow up period than those with BP, although the prevalence of obesity in BP was still higher than the US population prevalence. The cholesterol profile (total, HDL, LDL, VLDL) was significantly worse in SCZ than in BP, suggesting an earlier onset of complications such as heart disease and stroke reflected in their now widely reported increased CVD mortality. Cholesterol elevations especially are quite simple to treat: statins for example are widely available but are still underutilized (43). Interestingly enough, because weight gain appears to occur later in BP than SCZ, it is possible that the measured metabolic parameters will also show increasingly detrimental changes, albeit after an even longer observation period. This would allow for a longer window to execute preventative strategies. Moreover, because weight gain continues for 20 years, interventions can be effective in mitigating further weight gain even many years into treatment. Finally, another important takeaway of our data is that waist circumference should be measured frequently, especially in women with SCZ.

There is very little truly long-term weight gain data available for both SCZ and BP. Most 'long term' trials are limited to a one-year duration if not shorter (44–48). Our data represent the most comprehensive weight gain trajectory available to date, in a well-controlled epidemiological setting and carefully conducted over 20 years. Points of obesity prevention efforts in both SCZ and BP should include early overweight, in that patients who were overweight at 6 months had uniformly high rates of obesity at 20 years, along with increased waist circumference, accounting for much of the metabolic risk subsequently incurred (49). Moreover, given that weight gain unfolds over 20 years in both groups, with BP experiencing later development of obesity relative to SCZ probably due to lower initial BMI, there may be more time to intervene in BP. While early weight gain could be targeted with preventative strategies, well-validated preventative strategies remain scarce. Our results call for more investigative effort on how prevention of early weight gain might favorably alter long term weight trajectories. Long-term interventions aimed at obesity and related cardiovascular risk factors, which have been investigated more comprehensively, should study implementation strategies that effectively translate these modestly efficacious interventions into real-world settings (50), for the mitigation of further weight gain and weight loss along the 20 year weight gain trajectory.

Limitations

This is a single-site study. We did not determine if the rates of weight gain were different than what would be expected across the early adult life course in healthy comparators. The ability to analyze effects of antipsychotic medications was somewhat limited in that cumulative exposure was not assessed. There may be survivorship effects in our data. We also combined schizophrenia and schizoaffective disorder into one category (SCZ). We cannot exclude that schizoaffective disorder patients had a different weight gain propensity, as some prior literature has suggested. However, we believe that the weight gain trajectory

may not have been too dissimilar from BP and SCZ, both of whom gained weight in parallel, but with a somewhat different time course.. There also was no data presented on whether there were treatments for hyperlipidemia or diabetes that might have altered the biomarkers.

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References

1. Flegal KM, Kruszon-Moran D, Carroll MD, Fryar CD, Ogden CL. Trends in Obesity Among Adults in the United States, 2005 to 2014. *JAMA*. 2016; 315(21):2284–91. [PubMed: 27272580]
2. Pi-Sunyer X. The medical risks of obesity. *Postgrad Med*. 2009; 121(6):21–33. [PubMed: 19940414]
3. Anton SD, Woods AJ, Ashizawa T, Barb D, Buford TW, Carter CS, Clark DJ, Cohen RA, Corbett DB, Cruz-Almeida Y, Dotson V, Ebner N, Efron PA, Fillingim RB, Foster TC, Gundermann DM, Joseph AM, Karabetian C, Leeuwenburgh C, Manini TM, Marsiske M, Mankowski RT, Mutchie HL, Perri MG, Ranka S, Rashidi P, Sandesara B, Scarpace PJ, Sibille KT, Solberg LM, Someya S, Uphold C, Wohlgemuth S, Wu SS, Pahor M. Successful aging: Advancing the science of physical independence in older adults. *Ageing Res Rev*. 2015; 24(Pt B):304–27. [PubMed: 26462882]
4. Apovian CM. The clinical and economic consequences of obesity. *Am J Manag Care*. 2013; 19(11 Suppl):s219–28.
5. Janssen EM, McGinty EE, Azrin ST, Juliano-Bult D, Daumit GL. Review of the evidence: prevalence of medical conditions in the United States population with serious mental illness. *Gen Hosp Psychiatry*. 2015; 37(3):199–222. [PubMed: 25881768]
4. Yatsuya H, Li Y, Hilawe EH, Ota A, Wang C, Chiang C, Zhang Y, Uemura M, Osako A, Ozaki Y, Aoyama A. Global trend in overweight and obesity and its association with cardiovascular disease incidence. *Circ J*. 2014; 78(12):2807–18. [PubMed: 25391910]
5. Lobstein T, Jackson-Leach R, Moodie ML, Hall KD, Gortmaker SL, Swinburn BA, James WP, Wang Y, McPherson K. Child and adolescent obesity: part of a bigger picture. *Lancet*. 2015; 385(9986):2510–20. [PubMed: 25703114]
6. Conus P, Macneil C, McGorry PD. Public health significance of bipolar disorder: implications for early intervention and prevention. *Bipolar Disord*. 2014; 16(5):548–56. [PubMed: 24127825]
7. Chodick G, Shalev V. Epidemiology and comorbidity of severe mental illnesses in the community: findings from a computerized mental health registry in a large Israeli health organization. *Soc Psychiatr Psychiatr Epidemiol*. 2012; 47(11):1775–82.
8. Crump C, Sundquist K, Winkleby MA, Sundquist J. Comorbidities and mortality in bipolar disorder: A Swedish national cohort study. *JAMA Psychiatry*. 2013; 70(9):931–9. [PubMed: 23863861]
9. Hennekens CH, Hennekens AR, Hollar D, Casey DE. Schizophrenia and increased risks of cardiovascular disease. *Am Heart J*. 2005; 150(6):1115–21. [PubMed: 16338246]
10. McEvoy JP, Meyer JM, Goff DC, Nasrallah HA, Davis SM, Sullivan L, Meltzer HY, Hsiao J, Scott Stroup T, Lieberman JA. Prevalence of the metabolic syndrome in patients with schizophrenia: baseline results from the Clinical Antipsychotic Trials of Intervention Effectiveness (CATIE) schizophrenia trial and comparison with national estimates from NHANES III. *Schizophr Res*. 2005; 80(1):19–32. [PubMed: 16137860]
11. Jin H, Folsom D, Sasaki A, Mudaliar S, Henry R, Torres M, Golshan S, Glorioso DK, Jeste D. Increased Framingham 10-year risk of coronary heart disease in middle-aged and older patients with psychotic symptoms. *Schizophr Res*. 2011; 125(2–3):295–9. [PubMed: 21093219]
12. Miller C, Bauer MS. Excess mortality in bipolar disorders. *Curr Psychiatry Rep*. 2014; 16(11):499. [PubMed: 25194314]

13. Young AH, Grunze H. Physical health of patients with bipolar disorder. *Acta Psychiatr Scand Suppl.* 2013; 442:3–10.
14. Newcomer JW. Metabolic risk during antipsychotic treatment. *Clin Ther.* 2004; 26(12):1936–46. [PubMed: 15823759]
15. Casey DE, Haupt DW, Newcomer JW, Henderson DC, Sernyak MJ, Davidson M, Lindenmayer JP, Manoukian SV, Banerji MA, Lebovitz HE, Hennekens CH. Antipsychotic-induced weight gain and metabolic abnormalities: implications for increased mortality in patients with schizophrenia. *J Clin Psychiatry.* 2004; 65(Suppl 7):4–18.
16. Newcomer JW. Metabolic syndrome and mental illness. *Am J Managed Care.* 2007; 13(7 Suppl):S170–7.
17. Hennekens CH, Hennekens AR, Hollar D, Casey D. Schizophrenia and increased risks of cardiovascular disease. *Am Heart J.* 2005; 150(6):1115–21. [PubMed: 16338246]
18. Beary M, Hodgson R, Wildgust HJ. A critical review of major mortality risk factors for all-cause mortality in first-episode schizophrenia: clinical and research implications. *J Psychopharmacol.* 2012; 26:52–61. [PubMed: 22465947]
19. Laursen TM. Life expectancy among persons with schizophrenia or bipolar affective disorder. *Schizophr Res.* 2011; 131(1):101–4. [PubMed: 21741216]
20. Fagiolini A, Chengappa KN, Soreca I, Chang J. Bipolar disorder and the metabolic syndrome: causal factors, psychiatric outcomes and economic burden. *CNS Drugs.* 2008; 22(8):655–69. Review. [PubMed: 18601304]
21. Kessing LV, Vradi E, Andersen PK. Life expectancy in bipolar disorder. *Bipolar Disorders.* 2015; 17(5):543–8. [PubMed: 25846854]
22. Newcomer J, Hennekens CH. Severe mental illness and risk of cardiovascular disease. *JAMA.* 1997; 298:1794–6.
23. Brown S, Birtwistle J, Roe L, Thompson C. The unhealthy lifestyle of people with schizophrenia. *Psychol Med.* 1999; 29(3):697–701. [PubMed: 10405091]
24. Strassnig M, Brar JS, Ganguli R. Nutritional assessment of patients with schizophrenia: a preliminary study. *Schizophr Bull.* 2003; 29(2):393–7. [PubMed: 14552512]
25. Strassnig M, Brar JS, Ganguli R. Low cardiorespiratory fitness and physical functional capacity in obese patients with schizophrenia. *Schizophr Res.* 2011; 126(1–3):103–9. [PubMed: 21146958]
26. Mitchell AJ, Delaffon V, Vancampfort D, Correll CU, De Hert M. Guideline concordant monitoring of metabolic risk in people treated with antipsychotic medication: systematic review and meta-analysis of screening practices. *Psychol Med.* 2012; 42(1):125–47. [PubMed: 21846426]
27. Fagiolini A, Goracci A. The effects of undertreated chronic medical illnesses in patients with severe mental disorders. *J Clin Psychiatry.* 2009; 70(Suppl 3):22–29. [PubMed: 19570498]
28. Mauksch LB, Tucker SM, Katon WJ, Russo J, Cameron J, Walker E, Spitzer R. Mental illness, functional impairment, and patient preferences for collaborative care in an uninsured, primary care population. *J Fam Pract.* 2001; 50(1):41–7. [PubMed: 11195480]
29. Druss BG, Newcomer JW. Challenges and solutions to integrating mental and physical health care. *J Clin Psychiatry.* 2007; 68(4):e09. [PubMed: 17474799]
30. Morrato EH, Druss B, Hartung DM, Valuck RJ, Allen R, Campagna E, Newcomer JW. Metabolic testing rates in 3 state Medicaid programs after FDA warnings and ADA/APA recommendations for second-generation antipsychotic drugs. *Arch Gen Psychiatry.* 2010; 67(1):17–24. [PubMed: 20048219]
31. Nutrition, Physical Activity and Obesity Data, Trends and Maps web site. U.S. Department of Health and Human Services, Centers for Disease Control and Prevention (CDC), National Center for Chronic Disease Prevention and Health Promotion, Division of Nutrition, Physical Activity and Obesity; Atlanta, GA: 2015. Available at <http://www.cdc.gov/nccdphp/DNPAO/index.html>
32. Bromet EJ, Schwartz JE, Fennig S, Geller L, Jandorf L, Kovasznay B, Lavelle J, Miller A, Pato C, Ram R, et al. The epidemiology of psychosis: The Suffolk County Mental Health Project. *Schizophr Bull.* 1992; 18(2):243–55. [PubMed: 1621071]
33. Bromet EJ, Kotov R, Fochtmann LJ, Carlson GA, Tanenberg-Karant M, Ruggero C, Chang SW. Diagnostic shifts during the decade following first admission for psychosis. *Am J Psychiatry.* 2011; 168:1186–94. [PubMed: 21676994]

34. Spitzer RL, Williams JB, Gibbon M, First MB. The Structured Clinical Interview for DSM-III-R (SCID). I: History, rationale, and description. *Arch Gen Psychiatry*. 1992; 49(8):624–9. [PubMed: 1637252]
35. Velthorst E, Fett A-K, Reichenberg A, Perlman G, van Os J, Bromet EJ, Kotov R. The 20-yaer Longitudinal Trajectory of Social Functioning in Individuals with Psychotic Disorders. *Am J Psychiatry*. 2016; doi: 10.1176/appi.ajp.2016.15111419
36. Ogden CL, Carroll MD, Kit BK, Flegal KM. Prevalence of childhood and adult obesity in the United States, 2011–2012. *JAMA*. 2014; 311(8):806–14. [PubMed: 24570244]
37. [Dec 12, 2016] Accessed at https://www.health.ny.gov/statistics/brfss/reports/docs/1605_brfss_overweight_and_obesity.pdf
38. Flegal KM, Kit BK, Orpana H, Graubard BI. Association of all-cause mortality with overweight and obesity using standard body mass index categories: a systematic review and meta-analysis. *JAMA*. 2013; 309(1):71–82. [PubMed: 23280227]
39. Zheng H, Tumin D, Qian Z. Obesity and mortality risk: new findings from body mass index trajectories. *Am J Epidemiol*. 2013; 178(11):1591–9. [PubMed: 24013201]
40. Ohlson LO, Larsson B, Svardsudd K, et al. The influence of body fat distribution on the incidence of diabetes mellitus. 13.5 years of follow-up of the participants in the study of men born in 1913. *Diabetes*. 1985; 34:1055–8. [PubMed: 4043554]
41. Larsson B, Svardsudd K, Welin L, Wilhelmsen L, Bjorntorp P, Tibblin G. Abdominal adipose tissue distribution, obesity, and risk of cardiovascular disease and death: 13 year follow up of participants in the study of men born in 1913. *Br Med J (Clin Res Ed)*. 1984; 288:1401–4.
42. Zhang C, Rexrode KM, van Dam RM, Li TY, Hu FB. Abdominal obesity and the risk of all-cause, cardiovascular, and cancer mortality: sixteen years of follow-up in US women. *Circulation*. 2008; 117:1658–67. [PubMed: 18362231]
43. De Hert M, Yu W, Detraux J, Sweers K, van Winkel R, Correll CU. Body weight and metabolic adverse effects of asenapine, iloperidone, lurasidone and paliperidone in the treatment of schizophrenia and bipolar disorder: a systematic review and exploratory meta-analysis. *CNS Drugs*. 2012; 26(9):733–59. [PubMed: 22900950]
44. Manu P, Dima L, Shulman M, Vancampfort D, De Hert M, Correll CU. Weight gain and obesity in schizophrenia: epidemiology, pathobiology, and management. *Acta Psychiatr Scand*. 2015; 132(2): 97–108. [PubMed: 26016380]
45. Fagioli A. Medical monitoring in patients with bipolar disorder: a review of data. *J Clin Psychiatry*. 2008; 69(6):e16. [PubMed: 18683991]
46. De Hert M, Schreurs V, Vancampfort D, VAN Winkel R. Metabolic syndrome in people with schizophrenia: a review. *World Psychiatry*. 2009; 8(1):15–22. [PubMed: 19293950]
47. Torrent C, Amann B, Sánchez-Moreno J, Colom F, Reinares M, Comes M, Rosa AR, Scott J, Vieta E. Weight gain in bipolar disorder: pharmacological treatment as a contributing factor. *Acta Psychiatr Scand*. 2008; 118(1):4–18. [PubMed: 18498432]
48. Homel P, Casey D, Allison DB. Changes in body mass index for individuals with and without schizophrenia, 1987–1996. *Schizophr Res*. 2002; 55(3):277–84. [PubMed: 12048151]
49. Strassnig M, Clarke J, Mann S, Remington G, Ganguli R. Body composition, pre-diabetes and cardiovascular disease risk in early schizophrenia. *Early Interv Psychiatry*. 2015; doi: 10.1111/eip.12225
50. McGinty EE, Baller J, Azrin ST, Juliano-Bult D, Daumit GL. Interventions to Address Medical Conditions and Health-Risk Behaviors Among Persons With Serious Mental Illness: A Comprehensive Review. *Schizophr Bull*. 2016; 42(1):96–124. [PubMed: 26221050]

Figure 1.
Percent of Adults 18 and older who are obese* in New York State⁺⁺
*Obese is defined as BMI>30.
⁺⁺Data derived from the Behavioral Risk Factor Surveillance Survey, 2015

Figure 2.

Figure 3.

Table 1

Demographics and Metabolic Measures at 20 years

	Schizophrenia n=146	Bipolar Disorder n=87		
Male/Female	88/58	43/44	$\chi^2(1)=2.61$	$p=0.11$
Age@20 year follow up	47.9±8	47.6±9.6	$t=0.27$, $df=231$	$p=0.8$
<i>Health ratings</i>				
Triglycerides raw mg/dl	170 ±115.1	152.5±116.3	$t=0.95$, $df=172$	$P=0.35$
Total cholesterol mg/dl	188 ±47.8	203.4±40	$t=2.2$, $df=172$	$p=0.03$
HDL cholesterol mg/dl	47±14.7	52.9±18.3	$t=-2.3$, $df=172$	$p=0.022$
LDL cholesterol mg/dl	107±38.3	119±32.8	$t=-2.1$, $df=172$	$p=0.042$
VLDL cholesterol mg/dl	30.2±14.9	52.9±18.3	$t=2.4$, $df=162$	$p=0.02$
HGBA1c	6±1.52	5.6±1.4	$t=1.7$, $df=175$	$p=0.1$
WC/inches				
MEN	45.6 ±6.6	42.8 ±6	$t=2$, $df=98$	$p=0.049$
WOMEN	44.8±8.1	40.9±6.1	$t=2.3$, $df=77$	$p<0.022$
Pulse Pressure*	104±12.1	104±11.4	$t=0.28$, $df=171$	$p=0.78$

Note that dfs are variable because of missing data on some cases;

Not all cases received the physical exam at year 20.

Table 2

Mean BMI Values for Schizophrenia and Bipolar patients over 20 years

	Schizophrenia			Bipolar			t	p
	M	SD	N	M	SD	N		
6 Month	26.7	5.6	117	24.8	3.7	71	2.43	.02
2 year	27.8	6.5	105	25.4	3.6	68	2.98	.003
4 year	28.4	6.8	101	26.7	5.6	69	1.70	.09
10 year	31.1	7.2	135	28.9	5.5	76	2.30	.02
20 Year	33.8	8.5	117	31.5	6.4	63	2.12	.04

Table 3

Correlation of Other Health Status Indicators and Body Mass Index in the Combined Sample

Variable	Mean	SD	Correlation with BMI
Triglycerides	163.6	115.4	.08
HDL Cholesterol	49.1	16.3	−0.44 *
LDL Cholesterol	111.1	36.8	.02
VLDL Cholesterol	28.3	14.1	.15
Hemoglobin A1c	5.9	1.5	.18
% Body Fat			
<i>Men</i>	17.3	8.2	0.71 *
<i>Women</i>	17.3	7.1	0.52 *
Waist Circumference (in.)			
<i>Men</i>	44.7	6.5	0.89 *
<i>Women</i>	43.2	7.6	0.86 *
Pulse Pressure	103.8	11.8	0.16

*
p<.001

Note Mean BMI=32.8, SD=7.9