Published in final edited form as:

Compr Psychiatry. 2014 February ; 55(2): 233–247. doi:10.1016/j.comppsy.2013.09.009.

Prevalence of cardiovascular risk factors among racial and ethnic minorities with schizophrenia spectrum and bipolar disorders: a critical literature review

Hannah Carliner^{a,*}, Pamela Y. Collins^b, Leopoldo J. Cabassa^{c,d,e}, Ann McNallen^f, Sarah S. Joestl^g, and Roberto Lewis-Fernández^{c,d,h}

^aDepartment of Social and Behavioral Sciences, Harvard School of Public Health, Boston, MA, USA

^bOffice for Research on Disparities and Global Mental Health, National Institute of Mental Health/NIH, Bethesda, MD, USA

^cDepartment of Psychiatry, Columbia University, New York, NY, USA

^dNew York State Center of Excellence for Cultural Competence, New York State Psychiatric Institute, New York, NY, USA

^eColumbia University School of Social Work, New York, NY, USA

^fDepartment of Population and Family Health, Mailman School of Public Health, Columbia University, New York, NY, USA

^gDepartment of Sociomedical Sciences, Mailman School of Public Health, Columbia University, New York, NY, USA

^hHispanic Treatment Program, New York State Psychiatric Institute, New York, NY, USA

Abstract

Objective—People with serious mental illness (SMI) die at least 11 years earlier than the general U.S. population, on average, due largely to cardiovascular disease (CVD). Disparities in CVD morbidity and mortality also occur among some U.S. racial and ethnic minorities. The combined effect of race/ethnicity and SMI on CVD-related risk factors, however, remains unclear. To address this gap, we conducted a critical literature review of studies assessing the prevalence of CVD risk factors (overweight/obesity, diabetes mellitus, metabolic syndrome, hypercholesterolemia, hypertension, cigarette smoking, and physical inactivity) among U.S. racial/ethnic groups with schizophrenia-spectrum and bipolar disorders.

Methods and Results—We searched MEDLINE and PsycINFO for articles published between 1986 and 2013. The search ultimately yielded 40 articles. There was great variation in sampling, methodology, and study populations. Results were mixed, though there was some evidence for increased risk for obesity and diabetes mellitus among African Americans, and to a lesser degree for Hispanics, compared to non-Hispanic Whites. Sex emerged as an important possible effect

modifier of risk, as women had higher CVD risk among all racial/ethnic subgroups where stratified analyses were reported.

Conclusions—Compared to general population estimates, there was some evidence for an additive risk for CVD risk factors among racial/ethnic minorities with SMI. Future studies should include longitudinal assessment, stratification by sex, subgroup analyses to clarify the mechanisms leading to potentially elevated risk, and the evaluation of culturally appropriate interventions to eliminate the extra burden of disease in this population.

1. Introduction

People with mental illness die on average eight years earlier than the general U.S. population [1]. Life expectancy for people with psychotic disorders (e.g. schizophrenia) is even lower, at 63.4 years, a difference of 11 years of average life expectancy [1]. Studies using clinical samples of people with serious mental illness (SMI; e.g., schizophrenia and bipolar disorder) receiving services in the public mental health system [2] or participants with lower socioeconomic position find even greater disparities in mortality — as great as 25 years difference [1,3,4]. Much of the difference in mortality rates is attributed to cardiovascular, cerebrovascular, and pulmonary diseases. Paralleling the leading cause of death in the general U.S. population, cardiovascular disease (CVD) is the leading cause of death among public mental health clients [4]. People with schizophrenia have 2.3 times the standardized mortality rate for CVD [3] and a 34% higher mortality following hospitalization for a myocardial infarction [5,6] than the general population.

According to the Centers for Disease Control and Prevention (CDC), major risk factors for CVD include overweight/obesity/diet, diabetes mellitus, elevated cholesterol, hypertension, current cigarette smoking, and physical inactivity [7,8]. Metabolic syndrome (MS) is also considered a risk factor for CVD both independently and because its symptoms overlap with these risk factors [9]. People with schizophrenia-spectrum disorders (SSD), bipolar disorder (BD), and other SMI have higher rates of many of these CVD risk factors than the general U.S. population — in the range of 1.5 to 2 times the population prevalence of diabetes, dyslipidemia, hypertension, and obesity [6].

The marked morbidity and mortality among people with SMI associated with these CVD risk factors stem from several causes. These include adverse effects of antipsychotic medications [3,10], reduced access to preventive health services and to high-quality medical care due to financial and structural barriers [11,12], disease-related harmful health behaviors [13], and health care professionals' mistaken belief that patients with SMI are incapable of achieving physical health and wellness [14,15]. Furthermore, people with SMI may be more likely to refuse care [3], less likely to properly adhere to prescribed treatment regimens [16], and less capable of articulating symptoms [17] than those without mental illness. All of these factors may create an increased burden of CVD and premature mortality among people with SMI despite the improved adherence to medical treatment for comorbidities that may result from frequent use of mental health services [18,19].

Similar disparities in morbidity and mortality due to CVD and its associated risk factors occur among racial and ethnic minority groups in the U.S. African Americans, Hispanics,

and American Indians have higher rates of CVD and shorter life expectancies compared with non-Hispanic Whites [20–22]. African Americans show higher rates of age-adjusted heart disease and diabetes-related deaths, overweight or obesity (among women), physical inactivity, diabetes, and hypertension than Whites [21,23–26]. Hispanics and American Indians/Alaska Natives also have higher rates of overweight or obesity, physical inactivity, and age-adjusted diabetes than Whites [26–28]. Many of these risk factors also vary by sex within these racial/ethnic groupings [8,20–22,25]. For example, in nationally-representative samples, prevalence of obesity is higher among women compared to men for Whites, African Americans, and Mexican Americans, in some cases by as much as 20%; however, the size of this sex difference varies by race/ethnicity and education [21]. Sex and gender are therefore important biological, social, and cultural covariates to include in risk assessments of CVD [29].

The elevated risk for higher CVD-related morbidity and mortality in racial/ethnic minority populations in the U.S. is likely due to many intersecting causes, such as genetic and epigenetic factors [30–32], physiological stress responses secondary to racism and discrimination [33,34], reduced access to preventive health services [35], adverse migration-related factors [35], worse health education [36], limited culturally competent health care [37], stress-induced harmful health behaviors [38], predisposing neighborhood factors [39], structural and financial limitations on positive health behaviors [40], lower socioeconomic position [33,35], and cultural characteristics affecting diet [41], among others [35].

For racial/ethnic minorities who also have SSD or BD, it is therefore possible that their CVD-related outcomes are even worse than minorities in the general population, or than majority Whites with these psychiatric disorders. Theoretical justification for the proposition of a double burden of risk due to race/ethnicity and psychiatric diagnosis, for CVD is based on the following factors: [1] the obesogenic and diabetogenic effects of antipsychotic medications may be intensified by yet unknown biological factors in some racial/ethnic subgroups [42]; (2) poorer communication between healthcare providers and their racial/ethnic minority patients, relative to White patients, may be exacerbated by social and cognitive deficits associated with SSD and BD, resulting in poorer quality treatment and less access to preventive interventions [43,44]; (3) patients' mistrust of the medical system due to past experiences of racism and well-documented unethical practices may interact with stigmatization of mental illness on the side of providers and with the symptoms of SSD or BD on the side of patients, thus preventing the strong patient-provider relationship needed for successful treatment [43]; (4) the widespread lack of health services and culturally-appropriate care in poor and minority neighborhoods may lead to even worse access to care among patients with SMI [3,11]; and (5) navigating the fragmented physical and mental healthcare systems may be even harder for individuals facing the combination of discrimination due to their race/ethnicity and disability and miscommunication due to their psychiatric condition [14,43].

Given the limited research in this area, it is unclear whether there actually is a double burden of risk for CVD associated with race/ethnicity and psychiatric illness. Furthermore, it remains unclear whether prevalence of CVD and its risk factors is higher among racial/ethnic minorities with SSD and BD compared to non-Hispanic Whites with the same

disorders. To begin to fill this gap, and in line with consensus statement recommendations of experts [10], we conducted a literature review to (1) quantify the number of research papers published between 1986 and 2013 that examine the association between CVD risk factors, racial and ethnic minority status, and diagnosis of schizophrenia-spectrum and bipolar disorders; and (2) document the prevalence of CVD risk factors among racial and ethnic minorities with these psychiatric disorders, in order to assess whether a double burden of morbidity and mortality occurs in this population.

2. Methods

We searched the MEDLINE and PsycINFO databases using combinations of the following search terms: cardiovascular disease, hypercholesterolemia, dyslipidemia, low HDL cholesterol, metabolic syndrome, hyperlipidemia, physical activity or inactivity, physical exercise, smoking, obesity or overweight, hypertension, and diabetes mellitus. To these we added the psychiatric terms of interest: schizophrenia, bipolar disorder, schizoaffective disorder, and severe or serious mental illness; and finally the following race and ethnicity terms: minority groups, Black or African American, Hispanic or Latino, American Indian or Native American, Asian American or Pacific Islander. This search yielded 239 articles, which we manually reviewed and found additional articles from the reference lists. We discovered that many articles do not include race/ethnicity in their title, abstract, or keywords, yet may still stratify certain analyses by race/ethnicity. This then required a more thorough manual review of articles assessing physical health factors in people with SMI, and we removed the racial/ethnic search terms from additional searches.

We included in our review articles published between 1986 and 2013 that met the following criteria: (1) study sample included adult racial/ethnic minorities with SSD (e.g., schizoaffective disorder and schizophrenia) or BD; (2) study delimited and reported CDC-defined CVD risk factors (overweight/obesity/diet, diabetes mellitus, high cholesterol, high blood pressure, current cigarette smoking, and physical inactivity) or metabolic syndrome, which comprises several CVD risk factors (e.g. hypertension, hypercholesterolemia, high fasting blood sugar) [7,8]; (3) study reported results for racial/ethnic minority groups; and (4) article was published in English. We selected 1986 as the starting date — the year CDC began to report obesity rates on a national level [45]. Essentially, to be included in this review, a study needed to present prevalence or risk estimates for CVD or its risk factors among people with SMI in the United States, stratified or moderated by race/ethnicity.

We excluded case studies, review articles, letters, dissertations, government reports, and reports that did not undergo peer review. We further excluded all studies that were not carried out in the U.S., as our racial and ethnic categories of interest are specific to the historic, sociodemographic, and cultural setting of this country. Finally, we excluded studies that only controlled for racial/ethnic origin without presenting outcomes by race/ethnicity. Authors 1, 2, 4, and 5 selected the studies reporting prevalence, odds ratios, or other outcome measures, by racial or ethnic group, for review and presentation in Table 1.

Unfortunately, a systematic literature review following established guidelines was not possible, given the nature of the data we used from published articles. To the extent that it

was possible, the authors followed standard guidelines for reviewing and reporting results of observational [46,47] and randomized controlled studies [48]. However, a thorough assessment of possible biases in the reviewed studies was not appropriate, as the data of interest for the current study were often merely baseline demographic data from the reviewed articles, and not subject to bias from analytic strategies, statistical methodology, or outcome assessment. When appropriate, we cite the potential for sampling bias in the studies presented in Table 1.

3. Results

The combination of psychiatric diagnosis and physical health search terms yielded 12,456 documents, after we removed duplicates, restricted to English language journal articles from the 1986–2013 timeframe, and limited the search to article titles or abstracts. Combining searches of psychiatric diagnoses, cardiovascular risk factors, and race/ethnicity yielded 164 studies in MEDLINE and 75 studies in PsycINFO. However, only 69 articles reported analytical results separately by racial/ethnic group. Of these, plus the studies found in manual review, 40 articles met the remaining inclusion criteria (see Table 1). Some studies examined more than one variable of interest. Three studies presented data by race/ethnicity on overall CVD-related risk or mortality [49–51], six on body mass index (BMI) [51–56], six on obesity [51,57–61], five on weight gain [58,62–65], eleven on diabetes mellitus [12,49,51,64,66–72], fourteen on metabolic syndrome and its symptoms [51–53,73–83], two on physical activity [55,84], and four on smoking [51,85–87].

The majority of articles ($N = 24$) reported on studies that employed chart review or other observational methods to yield cross-sectional data on the prevalence of CVD risk factors among in- and out-patient clinical samples [12,51–56,59–61,66,70,73–79,81,82,84–86]. Five articles reported findings from random samples of national hospital or insurance data sets (Medicare and Medicaid) [57,67–69,72]. Two articles conducted retrospective chart reviews and followed patients to death using Social Security Death Index data [50,87]. The remaining articles ($N = 9$) reported findings from clinical trials involving psychiatric medications [49,58,60,63–65,71,80,83]. None of the studies examined hypertension or hypercholesterolemia as singular risk factors, although these factors were included in several studies of metabolic syndrome. The sample sizes of the reviewed studies ranged from 33 to 140,951 participants. We summarize study findings by CVD risk factor in Table 1.

3.1. CVD risk and mortality

A recent study by Keenan and colleagues assessed overall CVD risk, based on Framingham Risk Score [51]. They found no statistically significant difference in risk between Whites and African Americans with SMI, even when stratified by sex. However, when compared to a general U.S. population sample, both Whites and African Americans with SMI had higher overall CVD risk than their counterparts. Two other studies assessed CVD mortality in the context of antipsychotic treatment. In a longitudinal study of outpatients with SSD taking clozapine, the authors reported increased risk of CVD mortality among African Americans and Hispanics compared to Whites, although the finding for African Americans failed to reach statistical significance [49]. The second study, of patients taking clozapine and risperidone, found that White race was a significant predictor of cardiac-related death; and

that the standardized mortality ratio (SMR) for CVD was higher among Whites with schizophrenia than non-Whites [50]

3.2. Obesity, BMI, and weight gain

Fifteen studies assessed at least one weight-related indicator (weight, weight gain, BMI, and obesity) among racial/ethnic minority populations with SMI. Five studies reported the prevalence of elevated BMI or overweight/obesity by racial or ethnic group, five reported mean weight by group, two reported odds or risk of obesity by race/ethnicity, and five reported mean weight gain or rate of weight gain after psychopharmacological treatment.

3.2.1. Cross-sectional studies—Ten studies reported cross-sectional data for obesity and weight. Three of these studies did not find statistically significant differences in obesity prevalence by race/ethnicity [51,57,59], although one of these found statistically higher mean weight among African Americans [51]. One study found a statistically significant higher prevalence of overall obesity and abdominal obesity among African Americans compared to Whites, but not among Hispanics [53]. Another found a higher prevalence of overweight and obesity among African Americans with BD, compared to Whites, with almost one-third of African Americans having Grade 2 obesity (BMI ≥ 35) [61]. Two studies did not conduct statistical tests, but found the highest mean BMI and relative fat intake among African American women, compared to Whites or people of “Other” race/ethnicity [54,56]. In one study, where mean BMI varied significantly by sex, Hispanic men had higher rates of obesity than other men [52]. In another, there was no difference between White and African American men, but White women had significantly lower BMIs than African American women [55]. One study found that African Americans with mood disorders had statistically higher odds of obesity compared to Whites [60]. In this sample, statistical significance was not achieved when all African Americans with SMI, or just those with schizophrenia, were compared with Whites; though the trend of higher odds was in the same direction.

3.2.2. Treatment and longitudinal studies—Five articles reporting on clinical trials also examined weight-related variables in the context of pharmacological interventions using a longitudinal design. Two studies did not find significant differences in weight gain by race/ethnicity [58,65] and two studies found significantly greater or faster weight gain among African Americans and other racial/ethnic minorities compared to Whites [62,63]; in the fifth study the non-White sample was too small for statistical analysis [64]. The three studies with the highest proportion of racial/ethnic minorities (12%–26%) all found significantly greater weight gain among African Americans [62,63], or trends in that direction [65]. In the latter, though differences were not significant, the authors still reported an average weight gain of 17 lb for African Americans and 9 lb for Whites over a 6-month period [65]. However, groups were not assessed separately by sex and standard deviations were quite large, thus limiting the power of significance tests.

3.2.3. Summary—The only study in this group that used a random sampling technique did not find significant differences in obesity by race/ethnicity using a sample of Medicaid recipients with SMI [57]. However, the two studies with the largest overall sample sizes

showed increased risk of weight gain or obesity among African Americans or non-Whites [60,62]. Other studies with large clinical samples point to potential variations in obesity due to race/ethnicity, sex, diagnosis, and treatment; with higher risk for African Americans, women, and people with SSD. Smaller pharmacological studies also point to higher risk of weight gain among African Americans in the clinic setting. However, the remaining studies found non-significant differences and inconsistent associations.

3.3. Diabetes mellitus

Eleven studies focusing on the association between SMI and diabetes mellitus (DM) included race or ethnicity in their data analysis. Seven studies examined the prevalence of DM by race/ethnicity and three reported the incidence of DM or diabetic ketoacidosis following initiation of pharmacotherapy. One study reported too small a sample of racial/ethnic minorities for statistical analysis.

3.3.1. Cross-sectional data—Five studies reported on the cross-sectional prevalence of DM among people with SMI by race/ethnicity [12,51,66,67,70]. The largest of these was based on a total sample of 21,000 individuals with SSD from three cohorts in two states: inpatients and outpatients from diverse psychiatric facilities, Medicaid recipients, and Medicare recipients [67]. Relative to non-Hispanic Whites, a statistically significantly higher prevalence of DM was found in all three cohorts among the African American or “Other” racial/ethnic groups.

Another prevalence study of inpatients with BD found no statistical differences in DM by race/ethnicity [66], whereas a study that compared outpatients with SSD to those with major mood disorders found that racial/ethnic minorities with SSD and Whites with mood disorders had statistically significant higher prevalence of DM compared to their counterparts from other racial/ethnic groups [12]. A different study reported that more African Americans in their sample were diabetic than not, but appropriate statistical tests were not conducted [70]. Another study of overweight/obese outpatients found no difference in DM prevalence between Whites and African Americans in their sample, but did find statistically higher prevalence of DM for SMI patients compared to their respective racial groups from the general U.S. population [51]. Finally, undiagnosed hyperglycemia was not found to differ significantly by race/ethnicity in a clinic sample of 121 patients [71].

3.3.2. Treatment studies—Three studies assessed incidence of DM by race/ethnicity after initiation of antipsychotic medication. Two of the three found statistically significant increased odds of DM among African Americans and Hispanics compared to Whites [49,68]. The third found increased odds of incident diabetic ketoacidosis among African Americans compared to Whites, but not among Hispanics or “Other” racial/ethnic groups [69]. The former two studies assessed only individuals with SSD, while the latter study included Medicaid recipients with a variety of SMI diagnoses. None of these studies examined sex differences.

The final study of DM was a group-level assessment of trends in DM prevalence over time, following the introduction of atypical antipsychotics from 1990 to 2001 [72]. Using a nationally representative sample of hospital discharges with close to 26,000 White and

African American individuals with SSD, the authors found an overall increasing prevalence of DM in SSD patients over time, with particularly sharp increases for African American females, which were statistically significantly larger than for other sex-race groups. The authors interpret their results as implying that the use of atypical antipsychotics may be especially harmful for African American women in terms of risk for future CVD [72].

3.3.3. Summary—African American, Hispanic, and “other” non-White race/ethnicity appear to be at significant risk for developing DM or its complications among people with SMI. Four studies [67–69,72] with large samples of randomly selected participants show statistically significant higher risk or prevalence of DM among African Americans, and less frequently Hispanics, with greater risk apparently associated with a diagnosis of SSD rather than BD.

3.4. Metabolic syndrome

Thirteen articles reported on metabolic syndrome (MS) by race/ethnicity in individuals with SSD or BD. All but three of these studies reported prevalence of MS without any other statistical calculations. Eight of the 13 studies used National Cholesterol Education Program Adult Treatment Panel III criteria for diagnosing MS [88]. The remaining five studies [51,52,77,78,83] used the updated American Heart Association/American Diabetes Association guidelines which lower the level of fasting glucose required for the diagnosis from >110 mg/dl to >100 mg/dl [89]. The findings of these three studies (as discussed below) with respect to race/ethnicity do not appear to be affected by this change in diagnostic criteria.

3.4.1. Cross-sectional data—Eleven studies were cross-sectional. Four of these found no significant differences in prevalence of MS by race/ethnicity [73,75,77,82]. Six others found significant differences, but results were mixed: (a) four of these six studies found higher prevalence of MS among Whites, compared to African Americans or “non-Whites” [51,74,78,79]; (b) one study found higher prevalence among Hispanics, compared to non-Hispanics [76]; and (c) another study found no significant differences between Hispanics and non-Hispanics, although Cuban-Americans had a significantly higher MS prevalence than other groups [81]. The eleventh study reported prevalence of MS without conducting statistical tests and found the highest prevalence among White men and Hispanic women [52].

3.4.2. Treatment studies—Two treatment studies assessed racial/ethnic differences in the prevalence of MS among clinic samples of patients with SSD receiving olanzapine, quetiapine, or risperidone [83]; or olanzapine or aripiprazole [80]. The former study found a significantly lower prevalence of treatment-emergent MS among African Americans compared to patients of other race/ethnicities.

In the latter study, there were no significant differences in the prevalence of MS between Whites and African Americans/Hispanics at baseline. At the end point, Whites were less likely than African Americans/Hispanics to have MS, although the difference was not statistically significant. Significant differences were evident by race/ethnicity in the MS

parameters affected by aripiprazole and olanzapine. Overall, aripiprazole, compared to olanzapine, had fewer adverse metabolic effects on both groups; however, African Americans/Hispanics on aripiprazole vs. olanzapine showed significantly better outcomes only for weight, waist circumference, total cholesterol, and non-HDL cholesterol, whereas Whites demonstrated significantly lower odds of worsening MS on the majority of parameters with aripiprazole [80]. The findings imply that race/ethnicity may moderate the risk of MS following treatment with specific atypical antipsychotics.

3.4.3. Summary—Among the cross-sectional studies that assessed prevalence of MS by race/ethnicity, Whites and Hispanics seem to be at higher risk for developing MS, compared to African Americans; and there is some evidence that this risk may vary by sex. In addition, the effect of antipsychotic medications on risk for MS seems to vary by race/ethnicity as well as by medication type. Antipsychotic medications with greater negative metabolic effects adversely affect all race/ethnic groups, but racial/ethnic differences may be masked. Medications with minimal metabolic effects may display more visible racial/ethnic differences, with greater beneficial effects for Whites. African Americans, Hispanics, and Whites may have different MS risk profiles, dependent on both sex and medication type, but data to date are limited and inconclusive.

3.5. Physical activity

3.5.1. Cross-sectional data—In one of the two studies that examined physical activity, outpatients with SSD, BD, and major depressive disorder were compared with matched participants from the NHANES II study [84]. The psychiatric patients were found to be less physically active than their matched counterparts without psychiatric disorder, but no significant racial/ethnic differences were found when Whites were compared to “non-Whites”.

The second study assessed cardiovascular fitness among overweight and obese community-dwelling patients with schizophrenia, schizoaffective disorder, and psychotic disorder NOS [55]. The authors found no differences between White and African American male participants in BMI, maximal oxygen uptake, or rating of perceived exertion following graded-exercise testing. However, White males exercised statistically significantly longer. No differences were found between White and African American female participants in maximum minutes spent exercising or in rating of perceived exertion. However, White females had significantly lower BMI and significantly higher maximal oxygen uptake, compared to African American females.

3.6. Smoking

3.6.1. Cross-sectional data—Four clinical studies, all reporting cross-sectional data, assessed the prevalence of current tobacco smoking among psychiatric in- and out-patients by race/ethnicity. One found no significant differences in smoking prevalence by race/ethnicity among patients with bipolar disorder [86]. However, the proportion of non-White participants was only 7%, limiting the power for cross-racial/ethnic comparisons. The second study, of patients in a state hospital with mixed psychiatric diagnoses, found no significant difference in smoking status by race/ethnicity, but higher odds among White

smokers of meeting a heavy smoking threshold than among non-White smokers [85]. The remaining studies found no significant difference in the prevalence of smoking between Whites and African-Americans in a sample of inpatients with psychosis, including SSD [87], and a sample of overweight/obese outpatients with SMI [51].

4. Discussion

Our critical literature review examined articles published between 1986 and 2013 that document the prevalence of CVD risk factors in patients with SSD and BD across racial/ethnic groups. The aim of this review was to assess the state of the science regarding the possible double burden of risk for CVD and its risk factors among racial/ethnic minorities with SMI, relative to non-Hispanic Whites with SMI. The variety of study methodologies and sampling approaches limits our ability to draw definitive conclusions. We outline the limitations of the research, highlight key findings, and discuss implications for research and clinical interventions.

4.1. Methodological limitations

Varied recruitment strategies, sample sizes, study designs, psychiatric disorder inclusion criteria, and categorization of participants by race and/or ethnicity limit our ability to make definitive statements about the prevalence of CVD risk factors. Study designs ranged from randomized clinical trials testing the likelihood of developing obesity in the context of antipsychotic pharmacotherapy to case-control studies of large public databases. Most studies recruited non-random, convenience samples from psychiatric clinics. Sample sizes were frequently small: 30% (N = 12) of studies had fewer than 100 participants.

Both small and large studies often included nonrepresentative samples with low proportions of minority participants— as low as 8% of the study sample (n = 4), with which prevalence could not be reliably estimated [58]. Prevalence estimates of CVD risk factors were further limited as many studies did not conduct separate analyses by sex, possibly biasing results by not incorporating this important effect modifier. Finally, we noted inconsistencies in the definition and assignment of participants to racial and ethnic categories. In some cases, investigators assigned all participants who were not White to one category, likely obscuring effects considerably, as African Americans, Hispanics of diverse races, Asian Americans, and American Indians are genetically, socially, economically, and culturally varied groups with disparate rates of CVD risk factors and access to medical care [27,90,91]. Hispanic ethnicity was rarely refined by country of origin, even though rates of CVD risk factors may vary widely by Hispanic subgroup and racial background [92–94]. Lastly, few studies included American Indian and Asian American participants.

4.2. Key findings

Findings about racial/ethnic differences in overall CVD morbidity and mortality were inconclusive. However, some signals of differences emerged for specific risk factors. As in the general population, African Americans with SMI appear to be at greater risk for obesity and weight gain, especially among women. Among the larger studies in our review, elevated rates of obesity among women with SMI were notable [54,56,57] and tended to exceed

obesity prevalence in women from the general population in national samples [51,95], possibly implying an additive risk for CVD among women with SMI across racial/ethnic subgroups. However, most studies reviewed here did not stratify their comparisons by sex, which may explain why some found no significant differences in weight or weight gain by race/ethnicity. Future studies should stratify by race, ethnicity, and sex; conduct longitudinal assessments (both observational and following medication initiation); and analyze possible pathways, since the pattern of higher rates of obesity among African Americans was not consistent across studies, implying the existence of a more complex association.

Similarly, African Americans and possibly Hispanics with SMI appear to be at particular risk for DM, especially in the context of treatment with atypical antipsychotics. These findings mirror rates from the general U.S. population, where African Americans and Mexican Americans have approximately twice the prevalence of diabetes mellitus as Whites [95]. There was also some evidence for a double burden of risk, as prevalence of DM among people with SMI was consistently higher than general population estimates within each racial/ethnic sub-group [51,67], indicating that SMI may exacerbate pre-existing disparities in DM disease rates for racial/ethnic minorities. The studies on DM in this review highlight the importance of stratifying analyses by sex and examining Hispanic sub-groups in future studies.

Our findings indicate that Whites and Hispanics may be at higher risk for MS among people with SMI. However, several studies in the present review showed statistically non-significant relationships between race/ethnicity and risk of MS, likely because men and women were combined in the analysis. The two MS studies with the largest sample sizes in this review were from the CATIE trial, and both presented results stratified by sex [52,78]. Prevalence of MS was higher among women than men within all racial/ethnic groups in this sample. For all race/sex subgroups, prevalence was higher in the CATIE sample than in the general U.S. population, although among men prevalence was very similar between participants with schizophrenia and the national average for all racial/ethnic groups [9]. Again, this may indicate a double burden of disease, in that the groups with elevated rates of MS in the general population consistently have even higher rates of disease among the population of people with SMI. Of particular note was that almost three-quarters of Hispanic women with schizophrenia in the fasting CATIE sample had MS [52]. Sex therefore emerged as likely a more significant risk factor than race/ethnicity for risk of CVD due to MS. More studies are needed, again which stratify by sex and Hispanic sub-group, as well as psychiatric diagnosis and medication type.

In the general U.S. population, African Americans and Hispanics are statistically significantly more prone to physical inactivity than Whites, according to national data [96]. Results from the present review were inconsistent and limited, so patterns cannot be compared. For smoking, rates are similar between African Americans and non-Hispanic Whites, and slightly lower among Hispanics in the general population, though variation is found within racial and ethnic subgroups [24]. Among psychiatric patients, an estimated 80% of patients with schizophrenia and 60% with BD smoke tobacco; thus, irrespective of racial/ethnic differences in prevalence, tobacco use is a commonly acknowledged serious

public health issue in this population [85,97]. However, the data we found are so limited that firm conclusions are impossible.

4.3. Conclusion

Results of this review, though inconclusive, indicate that risk for some CVD risk factors may vary by racial/ethnic subgroup and sex. Racial and ethnic categories should be clearly defined in future studies. Investigation of differential risk due to both biological sex and gender status is also warranted, in order to discriminate between biological (e.g., hormonal) risk factors versus sociocultural factors related to eating, physical activity, and physical appearance [29]. Researchers who study the health of racial/ethnic minority populations emphasize the importance of assessing the interactive effects of race, ethnicity, sex, gender, socioeconomic status, place, and culture over the life course, in order to more fully capture the axes through which differences and disparities occur [35].

The goals of these analyses are to clearly identify where disparities exist and what factors perpetuate them, and to lay the groundwork for research that informs interventions to reduce them. To this aim, we encourage programs of research to: (a) systematically sample adequate numbers of racially and ethnically diverse participants with SSD, BD and other SMI to enable analyses with adequate power; (b) assess the relationships among race/ethnicity, sex, gender, diagnosis, and CVD risk factors; (c) employ methods of longitudinal research to track the progression of disease within these populations; (d) evaluate the extent to which health services variables (e.g., provider behavior, access to medical care, availability of appropriate preventive and treatment interventions, engagement in care, quality of care) contribute to the burden of CVD risk factors among racial/ethnic minorities with SMI; and (e) identify the various contributions of individual (genetic, hormonal), social/environmental (cultural, economic, social), and service-related factors on CVD outcomes among people with SSD, BD and other SMI in order to intervene effectively. In this review, aside from some studies which used large national datasets, none of the above conditions were met.

With respect to clinical practice, healthcare professionals should be mindful of the potentially differential adverse effects of psychiatric medications on patients based on race/ethnicity and sex. Patients naïve to psychotropic medications should be monitored carefully for changes in weight, metabolic symptoms, and other symptoms of cardiovascular disease; and clinicians should be prepared for potentially differential outcomes in minority patients. Clinicians should attempt to prescribe medications with less risk for developing obesity and diabetes to those at greater risk, and monitor these patients closely for potential adverse effects [98]. Clinical practice should be informed by existing treatment guidelines for prescribing and monitoring the physical health of people with SMI [10,99], with extra consideration given to racial/ethnic minority patients who may be at greater risk of developing adverse effects from treatment.

Ultimately, mental health service systems must consider the physical health outcomes of people with SMI as key measures of their success; collaborate with other health professionals to reduce adverse CVD outcomes; begin to integrate care guidelines for cardiovascular disease and diabetes prevention, monitoring, and referral into routine

practice, as recommended by expert panels [99]; and implement existing models of care that have been found to reduce CVD and improve the physical health of people with SMI [100–105]. Healthcare professionals must also work to change health policies and insurance practices that create barriers to integrated care systems and deny reimbursements to preventive services for people with SMI. Although the poor physical health of people with SMI has received increasing attention from the public health community, the specific health issues faced by racial/ethnic minorities with SMI are an important point to consider as part of the overall effort to reduce health disparities in the United States and integrate systems of care.

Acknowledgments

The authors wish to thank Andel Nicasio, Elizabeth Siantz, Madeline Tavarez, and Ron Turner for administrative and review support for this manuscript. This work was supported in part by the New York State Office of Mental Health (HC, PYC, LJC, RLF), by institutional support from the New York State Psychiatric Institute (RLF), and NIH grants L60 MD001850 (LJC), K01 MH09118 (LJC), and by the Implementation Research Institute (IRI) at the George Warren Brown School of Social Work, Washington University in St. Louis through an award from the National Institute of Mental Health (R25 MH080916) and the Department of Veterans Affairs, Health Services Research & Development, Quality Enhancement Research Initiative (QUERI). Dr. Lewis-Fernández receives research support from Eli Lilly & Co. The views expressed in this article do not necessarily represent the views of the NIMH or the federal government.

References

1. Druss BG, Zhao L, Von Esenwein S, Morrato EH, Marcus SC. Understanding excess mortality in persons with mental illness: 17-year follow up of a nationally representative US survey. *Med Care*. 2011; 49(6):599–604. [PubMed: 21577183]
2. Office of Applied Studies. Summary of national findings. Rockville, MD: Substance Abuse and Mental Health Services Administration; 2002. Results from the 2001 National Household Survey on Drug Abuse: volume 1. Contract No.: DHHS Publication No. SMA 02-3758
3. Parks, J.; Svendsen, D.; Singer, P.; Foti, ME.; Mauer, B. Morbidity and mortality in people with serious mental illness. Alexandria, VA: National Association of State Mental Health Program Directors (NASMHPD) Medical Directors Council; 2006 Oct. 2006. Report No
4. Colton CW, Manderscheid RW. Congruencies in increased mortality rates, years of potential life lost, and causes of death among public mental health clients in eight states. *Prev Chronic Dis*. 2006; 3(2):A42. [PubMed: 16539783]
5. Druss BG, Bradford WD, Rosenheck RA, Radford MJ, Krumholz HM. Quality of medical care and excess mortality in older patients with mental disorders. *Arch Gen Psychiatry*. 2001; 58(6):565–72. [PubMed: 11386985]
6. Newcomer JW, Hennekens CH. Severe mental illness and risk of cardiovascular disease. *JAMA*. 2007; 298(15):1794–6. [PubMed: 17940236]
7. Centers for Disease Control and Prevention (CDC). Racial/ethnic and socioeconomic disparities in multiple risk factors for heart disease and stroke—United States, 2003. *MMWR Morb Mortal Wkly Rep*. 2005 Feb 11; 54(5):113–7. [PubMed: 15703691]
8. Centers for Disease Control and Prevention (CDC). Prevalence of heart disease—United States, 2005. *MMWR Morb Mortal Wkly Rep*. 2007 Feb 16; 56(6):113–8. [PubMed: 17301730]
9. Ervin RB. Prevalence of metabolic syndrome among adults 20 years of age and over, by sex, age, race and ethnicity, and body mass index: United States, 2003–2006. *National health statistics reports*. 2009; 5(13):1–7. [PubMed: 19634296]
10. Care Diabetes. American Diabetes Association APA, American Association of Clinical Endocrinologists, and North American Association for the Study of Obesity. Consensus Development Conference on Antipsychotic Drugs and Obesity and Diabetes. 2004; 27(2):596–601.

11. Druss BG, Bornemann TH. Improving health and health care for persons with serious mental illness: the window for US federal policy change. *Jama*. 2010 May 19; 303(19):1972–3. [PubMed: 20483975]
12. Kreyenbuhl J, Dickerson FB, Medoff DR, Brown CH, Goldberg RW, Fang L, et al. Extent and management of cardiovascular risk factors in patients with type 2 diabetes and serious mental illness. *J Nerv Ment Dis*. 2006 Jun; 194(6):404–10. [PubMed: 16772856]
13. Compton MT, Daumit GL, Druss BG. Cigarette smoking and overweight/obesity among individuals with serious mental illnesses: a preventive perspective. *Harv Rev Psychiatry*. 2006; 14(4):212–22. [PubMed: 16912007]
14. Vreeland B. Bridging the gap between mental and physical health: a multidisciplinary approach. *J Clin Psychiatry*. 2007; 68(Suppl 4):26–33. [PubMed: 17539697]
15. Morden NE, Mistler LA, Weeks WB, Bartels SJ. Health care for patients with serious mental illness: family medicine's role. *J Am Board Fam Med*. 2009; 22(2):187–95. [PubMed: 19264942]
16. Brown S, Inskip H, Barraclough B. Causes of the excess mortality of schizophrenia. *Br J Psychiatry*. 2000; 177:212–7. [PubMed: 11040880]
17. Goff DC. Integrating general health care in private community psychiatry practice. *J Clin Psychiatry*. 2007; 68(Suppl 4):49–54. [PubMed: 17539701]
18. Himelhoch S, Brown CH, Walkup J, Chander G, Korthius PT, Afful J, et al. HIV patients with psychiatric disorders are less likely to discontinue HAART. *AIDS*. 2009; 23(13):1735–42. [PubMed: 19617816]
19. Kreyenbuhl J, Dixon LB, McCarthy JF, Soliman S, Ignacio RV, Valenstein M. Does adherence to medications for type 2 diabetes differ between individuals with vs without schizophrenia? *Schizophr Bull*. 2010; 36(2):428–35. [PubMed: 18718883]
20. Pleis JR, Lucas JW. Summary health statistics for U.S. adults: National Health Interview Survey, 2007. *Vital Health Stat*. 2009 May; 10(240):1–159.
21. Mensah GA, Mokdad AH, Ford ES, Greenlund KJ, Croft JB. State of disparities in cardiovascular health in the United States. *Circulation*. 2005; 111(10):1233–41. [PubMed: 15769763]
22. Keppel KG, Percy JN, Wagener DK. Trends in racial and ethnic-specific rates for the health status indicators: United States, 1990–98. *Healthy People 2000 Stat Notes*. 2002 Jan;(23):1–16. [PubMed: 11808619]
23. Centers for Disease Control and Prevention. Health, United States, 2005 with chartbook on trends in the health of Americans. Hyattsville, Maryland: National Center for Health Statistics; 2005. Report No
24. Centers for Disease Control and Prevention (CDC). Prevalence of cigarette use among 14 racial/ethnic populations—United States, 1999–2001. *MMWR Morb Mortal Wkly Rep*. 2004; 53(3):49–52. [PubMed: 14749612]
25. Centers for Disease Control, Prevention (CDC). Differences in prevalence of obesity among black, white, and Hispanic adults — United States, 2006–2008. *MMWR Morb Mortal Wkly Rep*. 2009; 58(27):740–4. [PubMed: 19609247]
26. Centers for Disease Control and Prevention. Health, United States, 2009 with chartbook on trends in the health of Americans. Hyattsville, Maryland: National Center for Health Statistics; 2009. Report No
27. Centers for Disease Control, Prevention (CDC). Prevalence of diabetes among Hispanics—selected areas, 1998–2002. *MMWR Morb Mortal Wkly Rep*. 2004; 53(40):941–4. [PubMed: 15483527]
28. The Henry J. Kaiser Family Foundation. Overweight and obesity rates for adults by race/ethnicity. The Henry J. Kaiser Family Foundation; [[cited 2010 January 25]. Available from:], <http://www.statehealthfacts.org/comparebar.jsp?ind=91&cat=22008>
29. Krieger N. Genders, sexes, and health: what are the connections—and why does it matter? *International Journal of Epidemiology*. 2003; 32(4):652–7. [PubMed: 12913047]
30. Burchard EG, Ziv E, Coyle N, Gomez SL, Tang H, Karter AJ, et al. The importance of race and ethnic background in biomedical research and clinical practice. *N Engl J Med*. 2003; 348(12):1170–5. [PubMed: 12646676]

31. Kuzawa CW, Sweet E. Epigenetics and the embodiment of race: developmental origins of US racial disparities in cardiovascular health. *Am J Hum Biol.* 2009; 21(1):2–15. [PubMed: 18925573]
32. Risch N, Burchard E, Ziv E, Tang H. Categorization of humans in biomedical research: genes, race and disease. *Genome Biol.* 2002; 3(7):1–12.
33. Mendelson T, Thurston RC, Kubzansky LD. Affective and cardiovascular effects of experimentally-induced social status. *Health Psychol.* 2008; 27(4):482–9. [PubMed: 18643006]
34. Lewis TT, Everson-Rose SA, Powell LH, Matthews KA, Brown C, Karavolos K, et al. Chronic exposure to everyday discrimination and coronary artery calcification in African-American women: the SWAN Heart Study. *Psychosom Med.* 2006; 68(3):362–8. [PubMed: 16738065]
35. Williams DR, Mohammed SA, Leavell J, Collins C. Race, socioeconomic status, and health: complexities, ongoing challenges, and research opportunities. *Ann N Y Acad Sci.* 2010 Feb. 1186:69–101. [PubMed: 20201869]
36. Wong MD, Shapiro MF, Boscardin WJ, Ettner SL. Contribution of major diseases to disparities in mortality. *N Engl J Med.* 2002; 347(20):1585–92. [PubMed: 12432046]
37. Johnson RL, Saha S, Arbelaez JJ, Beach MC, Cooper LA. Racial and ethnic differences in patient perceptions of bias and cultural competence in health care. *J Gen Intern Med.* 2004 Feb; 19(2): 101–10. [PubMed: 15009789]
38. Dressler WW, Oths KS, Gravlee CC. Race and ethnicity in public health research: models to explain health disparities. *Annu Rev Anthropol.* 2005; 34(1):231–52.
39. Williams DR, Collins C. Racial residential segregation: a fundamental cause of racial disparities in health. *Public Health Rep.* 2001; 116(5):404–16. [PubMed: 12042604]
40. Krieger N. Embodying inequality: a review of concepts, measures, and methods for studying health consequences of discrimination. *Int J Health Serv.* 1999; 29(2):295–352. [PubMed: 10379455]
41. Nettleton JA, Polak JF, Tracy R, Burke GL, Jacobs DR Jr. Dietary patterns and incident cardiovascular disease in the Multi-Ethnic Study of Atherosclerosis. *Am J Clin Nutr.* 2009 Sep; 90(3):647–54. [PubMed: 19625679]
42. Ader M, Garvey WT, Phillips LS, Nemeroff CB, Gharabawi G, Mahmoud R, et al. Ethnic heterogeneity in glucoregulatory function during treatment with atypical antipsychotics in patients with schizophrenia. *J Psychiatr Res.* 2008; 42(13):1076–85. [PubMed: 18295798]
43. U.S. Department of Health and Human Services. Mental health: culture, race, and ethnicity—a supplement to mental health: a report of the Surgeon General. Rockville, MD: U.S Department of Health and Human Services, Substance Abuse and Mental Health Services Administration, Center for Mental Health Services; 2001.
44. Cabassa LJ, Blanco C, Lopez-Castroman J, Lin KH, Lui SM, Lewis-Fernandez R. Racial and ethnic differences in diabetes mellitus among people with and without psychiatric disorders: results from the National Epidemiologic Survey on Alcohol and Related Conditions. *Gen Hosp Psychiatry.* 2011 Mar-Apr;33(2):107–15. [PubMed: 21596203]
45. Centers for Disease Control and Prevention (CDC). Behavioral risk factor surveillance system survey data. Atlanta, Georgia: U.S. Department of Health and Human Services, Centers for Disease Control and Prevention; 1986.
46. Stroup DF, Berlin JA, Morton SC, Olkin I, Williamson GD, Rennie D, et al. Meta-analysis of observational studies in epidemiology: a proposal for reporting. Meta-analysis Of Observational Studies in Epidemiology (MOOSE) group. *JAMA.* 2000; 283(15):2008–12. [PubMed: 10789670]
47. von Elm E, Altman DG, Egger M, Pocock SJ, Gøtzsche PC, Vandenbroucke JP, et al. The Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) statement: guidelines for reporting observational studies. *Prev Med.* 2007; 45(4):247–51. [PubMed: 17950122]
48. Higgins, J.; Green, S. *Cochrane handbook for systematic reviews of interventions*, version 5.1.0. 2011 Mar. 2011. Report No
49. Henderson DC, Nguyen DD, Copeland PM, Hayden DL, Borba CP, Louie PM, et al. Clozapine, diabetes mellitus, hyperlipidemia, and cardiovascular risks and mortality: results of a 10-year naturalistic study. *J Clin Psychiatry.* 2005; 66(9):1116–21. [PubMed: 16187768]

50. Kelly DL, McMahon RP, Liu F, Love RC, Wehring HJ, Shim JC, et al. Cardiovascular disease mortality in patients with chronic schizophrenia treated with clozapine: a retrospective cohort study. *J Clin Psychiatry*. 2010; 71(3):304–11. [PubMed: 20079332]
51. Keenan TE, Yu A, Cooper LA, Appel LJ, Guallar E, Gennusa JV 3rd, et al. Racial patterns of cardiovascular disease risk factors in serious mental illness and the overall U.S. population. *Schizophr Res*. 2013; 150(1):211–6. [PubMed: 23916188]
52. McEvoy JP, Meyer JM, Goff DC, Nasrallah HA, Davis SM, Sullivan L, et al. 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]
53. Schneiderhan ME, Batscha CL, Rosen C. Assessment of a point-of-care metabolic risk screening program in outpatients receiving antipsychotic agents. *Pharmacotherapy*. 2009; 29(8):975–87. [PubMed: 19637951]
54. Strassnig M, Brar JS, Ganguli R. Nutritional assessment of patients with schizophrenia: a preliminary study. *Schizophr Bull*. 2003; 29(2):393–7. [PubMed: 14552512]
55. 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]
56. Vieweg WV, Thomas M, Janisko M, Booth M, Fernandez A, Pandurangi A, et al. Patient and direct-care staff body mass index in a state mental hospital: implications for management. *Acta Psychiatr Scand*. 2004; 110(1):69–72. [PubMed: 15180782]
57. Daumit GL, Clark JM, Steinwachs DM, Graham CM, Lehman A, Ford DE. Prevalence and correlates of obesity in a community sample of individuals with severe and persistent mental illness. *J Nerv Ment Dis*. 2003; 191(12):799–805. [PubMed: 14671456]
58. Fagiolini A, Frank E, Houck PR, Mallinger AG, Swartz HA, Buysse DJ, et al. Prevalence of obesity and weight change during treatment in patients with bipolar I disorder. *J Clin Psychiatry*. 2002; 63(6):528–33. [PubMed: 12088166]
59. Fagiolini A, Kupfer DJ, Houck PR, Novick DM, Frank E. Obesity as a correlate of outcome in patients with bipolar I disorder. *Am J Psychiatry*. 2003; 160(1):112–7. [PubMed: 12505809]
60. Susce MT, Villanueva N, Diaz FJ, De Leon J. Obesity and associated complications in patients with severe mental illnesses: a crosssectional survey. *J Clin Psychiatry*. 2005; 66(2)
61. Kemp, DE.; Sylvia, LG.; Calabrese, JR.; Nierenberg, AA.; Thase, ME.; Reilly-Harrington, NA., et al. General medical burden in bipolar disorder: findings from the LiTMUS comparative effectiveness trial. *Acta Psychiatr Scand*. 2013. <http://dx.doi.org/10.1111/acps.12101>
62. Basson BR, Kinon BJ, Taylor CC, Szymanski KA, Gilmore JA, Tollefson GD. Factors influencing acute weight change in patients with schizophrenia treated with olanzapine, haloperidol, or risperidone. *J Clin Psychiatry*. 2001; 62(4):231–8. [PubMed: 11379836]
63. de Leon J, Diaz FJ, Josiassen RC, Cooper TB, Simpson GM. Weight gain during a double-blind multidose clozapine study. *J Clin Psychopharmacol*. 2007; 27(1):22–7. [PubMed: 17224708]
64. Henderson DC, Cagliero E, Gray C, Nasrallah RA, Hayden DL, Schoenfeld DA, et al. Clozapine, diabetes mellitus, weight gain, and lipid abnormalities: a five-year naturalistic study. *Am J Psychiatr*. 2000; 157(6):975–81. [PubMed: 10831479]
65. Littrell KH, Hilligoss NM, Kirshner CD, Petty RG, Johnson CG. The effects of an educational intervention on antipsychotic-induced weight gain. *J Nurs Scholarsh*. 2003; 35(3):237–41. [PubMed: 14562491]
66. Cassidy F, Ahearn E, Carroll BJ. Elevated frequency of diabetes mellitus in hospitalized manic-depressive patients. *Am J Psychiatry*. 1999; 156(9):1417–20. [PubMed: 10484954]
67. Dixon L, Weiden P, Delahanty J, Goldberg R, Postrado L, Lucksted A, et al. Prevalence and correlates of diabetes in national schizophrenia samples. *Schizophr Bull*. 2000; 26(4):903–12. [PubMed: 11087022]
68. Lambert BL, Chou CH, Chang KY, Tafesse E, Carson W. Antipsychotic exposure and type 2 diabetes among patients with schizophrenia: a matched case-control study of California Medicaid claims. *Pharmacoepidemiol Drug Saf*. 2005; 14(6):417–25. [PubMed: 15786516]

69. Ramaswamy K, Kozma CM, Nasrallah H. Risk of diabetic ketoacidosis after exposure to risperidone or olanzapine. *Drug Saf.* 2007; 30(7):589–99. [PubMed: 17604410]
70. Regenold WT, Thapar RK, Marano C, Gavirneni S, Kondapavuluru PV. Increased prevalence of type 2 diabetes mellitus among psychiatric inpatients with bipolar I affective and schizoaffective disorders independent of psychotropic drug use. *J Affect Disord.* 2002; 70(1):19–26. [PubMed: 12113916]
71. Sernyak MJ, Gulanski B, Leslie DL, Rosenheck R. Undiagnosed hyperglycemia in clozapine-treated patients with schizophrenia. *J Clin Psychiatry.* 2003; 64(5):605–8. [PubMed: 12755666]
72. Basu A, Meltzer HY. Differential trends in prevalence of diabetes and unrelated general medical illness for schizophrenia patients before and after the a typical antipsychotic era. *Schizophr Res.* 2006; 86(1–3):99–109. [PubMed: 16753284]
73. Basu R, Brar JS, Chengappa KN, John V, Parepally H, Gershon S, et al. The prevalence of the metabolic syndrome in patients with schizoaffective disorder–bipolar subtype. *Bipolar Disord.* 2004; 6(4):314–8. [PubMed: 15225149]
74. Bermudes RA, Keck PE Jr, Welge JA. The prevalence of the metabolic syndrome in psychiatric inpatients with primary psychotic and mood disorders. *Psychosomatics.* 2006; 47(6):491–7. [PubMed: 17116950]
75. Cardenas J, Frye MA, Marusak SL, Levander EM, Chirichigno JW, Lewis S, et al. Modal subcomponents of metabolic syndrome in patients with bipolar disorder. *J Affect Disord.* 2008; 106(1–2):91–7. [PubMed: 17628693]
76. Kato MM, Currier MB, Gomez CM, Hall L, Gonzalez-Blanco M. Prevalence of metabolic syndrome in Hispanic and non-Hispanic patients with schizophrenia. *Prim Care Companion J Clin Psychiatry.* 2004; 6(2):74–7. [PubMed: 15254600]
77. Lamberti JS, Olson D, Crilly JF, Olivares T, Williams GC, Tu X, et al. Prevalence of the metabolic syndrome among patients receiving clozapine. *Am J Psychiatry.* 2006; 163(7):1273–6. [PubMed: 16816234]
78. Meyer JM, Nasrallah HA, McEvoy JP, Goff DC, Davis SM, Chakos M, et al. The Clinical Antipsychotic Trials Of Intervention Effectiveness (CATIE) Schizophrenia Trial: clinical comparison of subgroups with and without the metabolic syndrome. *Schizophr Res.* 2005; 80(1): 9–18. [PubMed: 16125372]
79. Meyer JM, Pandina G, Bossie CA, Turkoz I, Greenspan A. Effects of switching from olanzapine to risperidone on the prevalence of the metabolic syndrome in overweight or obese patients with schizophrenia or schizoaffective disorder: analysis of a multicenter, raterblinded, open-label study. *Clin Ther.* 2005; 27(12):1930–41. [PubMed: 16507379]
80. Meyer JM, Rosenblatt LC, Kim E, Baker RA, Whitehead R. The moderating impact of ethnicity on metabolic outcomes during treatment with olanzapine and aripiprazole in patients with schizophrenia. *J Clin Psychiatry.* 2009; 70(3):318–25. [PubMed: 19192469]
81. Rivas-Vazquez RA, Bello I, Sarria M, Fernandez ND, Rey GJ. Prevalence of metabolic syndrome in a predominantly Cuban, psychiatrically ill, and homeless population. *Prim Care Companion CNS Disord.* 2011; 13(3) <http://dx.doi.org/10.4088/PCC.10m01002>.
82. Straker D, Correll CU, Kramer-Ginsberg E, Abdulhamid N, Koshy F, Rubens E, et al. Cost-effective screening for the metabolic syndrome in patients treated with second-generation antipsychotic medications. *Am J Psychiatry.* 2005; 162(6):1217–21. [PubMed: 15930076]
83. Patel JK, Buckley PF, Woolson S, Hamer RM, McEvoy JP, Perkins DO, et al. Metabolic profiles of second-generation antipsychotics in early psychosis: findings from the CAFE study. *Schizophr Res.* 2009; 111(1–3):9–16. [PubMed: 19398192]
84. Daumit GL, Goldberg RW, Anthony C, Dickerson F, Brown CH, Kreyenbuhl J, et al. Physical activity patterns in adults with severe mental illness. *J Nerv Ment Dis.* 2005; 193(10):641–6. [PubMed: 16208158]
85. de Leon J, Dadvand M, Canuso C, White AO, Stanilla JK, Simpson GM. Schizophrenia and smoking: an epidemiological survey in a state hospital. *Am J Psychiatry.* 1995; 152(3):453–5. [PubMed: 7864277]
86. Waxmonsky JA, Thomas MR, Miklowitz DJ, Allen MH, Wisniewski SR, Zhang H, et al. Prevalence and correlates of tobacco use in bipolar disorder: data from the first 2000 participants

- in the Systematic Treatment Enhancement Program. *Gen Hosp Psychiatry*. 2005; 27(5):321–8. [PubMed: 16168792]
87. Kelly DL, McMahon RP, Wehring HJ, Liu F, Mackowick KM, Boggs DL, et al. Cigarette smoking and mortality risk in people with schizophrenia. *Schizophr Bull*. 2009; 37(4):832–8. [PubMed: 20019128]
 88. Expert Panel of Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults. Executive summary of the third report of the National Cholesterol Education Program (NCEP) Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults (Adult Treatment Panel III). *Jama*. 2001; 285(19):2486–97. [PubMed: 11368702]
 89. Grundy SM, Brewer HB Jr, Cleeman JI, Smith SC Jr, Lenfant C. Definition of metabolic syndrome: report of the National Heart, Lung, and Blood Institute/American Heart Association conference on scientific issues related to definition. *Circulation*. 2004; 109(3):433–8. [PubMed: 14744958]
 90. Singh GK, Siahpush M. Ethnic-immigrant differentials in health behaviors, morbidity, and cause-specific mortality in the United States: an analysis of two national data bases. *Hum Biol*. 2002; 74(1):83–109. [PubMed: 11931581]
 91. Hummer RA, Rogers RG, Nam CB, LeClere FB. Race/ethnicity, nativity, and US adult mortality. *Soc Sci Q*. 1999; 80(1):136–53.
 92. Ortega AN, Feldman JM, Canino G, Steinman K, Alegria M. Cooccurrence of mental and physical illness in US Latinos. *Social psychiatry and psychiatric epidemiology*. 2006; 41(12):927–34. [PubMed: 17013767]
 93. Caballero A. Diabetes in the Hispanic or Latino population: genes, environment, culture, and more. *Current Diabetes Reports*. 2005; 5(3):217–25. [PubMed: 15929869]
 94. Daviglius MI, Talavera GA, Avilés-Santa M, et al. Prevalence of major cardiovascular risk factors and cardiovascular diseases among Hispanic/Latino individuals of diverse backgrounds in the United States. *JAMA*. 2012; 308(17):1775–84. [PubMed: 23117778]
 95. National Center for Health Statistics. Health, United States, 2011: with special feature on socioeconomic status and health. Hyattsville, Maryland: 2012.
 96. Schoenborn, CA.; Adams, PF. Health behaviors of adults: United States, 2005–2007. National Center for Health Statistics; 2010. Vital Health Stat 10(245)Contract No.: Vital Health Stat 10(245)
 97. Lasser, K.; Boyd, JW.; Woolhandler, S.; Himmelstein, DU.; McCormick, D.; Bor, DH. Smoking and mental illness: a population-based prevalence study. 2000. p. 2606–10.
 98. Ananth J, Kolli S, Gunatilake S, Brown S. Atypical antipsychotic drugs, diabetes and ethnicity. *Expert Opin Drug Saf*. 2005; 4(6):1111–24. [PubMed: 16255668]
 99. Marder SR, Essock SM, Miller AL, Buchanan RW, Casey DE, Davis JM, et al. Physical health monitoring of patients with schizophrenia. *Am J Psychiatry*. 2004; 161(8):1334–49. [PubMed: 15285957]
 100. Druss BG, Rohrbach RM, Levinson CM, Rosenheck RA. Integrated medical care for patients with serious psychiatric illness: a randomized trial. *Arch Gen Psychiatry*. 2001 Sep; 58(9):861–8. [PubMed: 11545670]
 101. Kilbourne AM, Post EP, Nosssek A, Drill L, Cooley S, Bauer MS. Improving medical and psychiatric outcomes among individuals with bipolar disorder: a randomized controlled trial. *Psychiatr Serv*. 2008; 59(7):760–8. [PubMed: 18586993]
 102. Brar JS, Ganguli R, Pandina G, Turkoz I, Berry S, Mahmoud R. Effects of behavioral therapy on weight loss in overweight and obese patients with schizophrenia or schizoaffective disorder. *J Clin Psychiatry*. 2005; 66(2):205–12. [PubMed: 15705006]
 103. McKibbin CL, Patterson TL, Norman G, Patrick K, Jin H, Roesch S, et al. A lifestyle intervention for older schizophrenia patients with diabetes mellitus: a randomized controlled trial. *Schizophr Res*. 2006; 86(1–3):36–44. [PubMed: 16842977]
 104. Van Citters AD, Pratt SI, Jue K, Williams G, Miller PT, Xie H, et al. A pilot evaluation of the In SHAPE individualized health promotion intervention for adults with mental illness. *Community Ment Health J*. 2010 Dec; 46(6):540–52. [PubMed: 20012197]

105. Druss BG, von Esenwein SA, Compton MT, Rask KJ, Zhao L, Parker RM. A randomized trial of medical care management for community mental health settings: the Primary Care Access, Referral, and Evaluation (PCARE) study. *Am J Psychiatry*. 2010; 167(2):151–9. [PubMed: 20008945]

Summary of results.

Table 1

Author	Psychiatric diagnosis	Sample size (N)	Proportion of racial/Ethnic minorities	% Female	Type of sample	Data type	Measure	Group with more adverse outcome(s)
<i>OVERALL CVD MORTALITY</i>								
Henderson et al., 2005	Sch, SzAD	96	6% African American 4% Hispanic 1% Asian American	28%	Outpatient clinic sample	Longitudinal treatment study of clozapine	CVD Mortality risk Incident DM	African Americans and Hispanics
Keenan et al., 2013	Sch, SzAD, BD, MDD, OD	274 with SMI 1653 from NHANES	40.5% African American	50.3%	Overweight/obese outpatient clinic sample from psychiatric rehabilitation programs in Maryland	Cross-sectional baseline data from a behavioral weight loss intervention clinical trial	Mean weight, BMI, waist circumference, fasting glucose, systolic BP, diastolic BP, total cholesterol, triglycerides, HDL-C, LDL-C, and overall 10-year global CVD risk score Prevalence and risk of smoking, obesity, diabetes, hypertension, and metabolic syndrome	African Americans (mean weight, blood pressure) Whites (triglycerides, HDL-C, high cholesterol, MS) No significant differences: 10-year CVD risk and other risk factors
Kelly et al., 2010	Sch, SzAD, PDNOS	1686	Clozapine group: 33.2% African American, 3.9% Other Risperidone group: 49.7% African American, 2.5% Other	~38%	Maryland administrative database samples of antipsychotics users	Retrospective cohort longitudinal treatment study of clozapine and risperidone	CVD Mortality risk	Whites
<i>OVERWEIGHT, OBESITY, DIET</i>								
Daumit et al., 2003	SPMI: Sch, BD, OD	223	~50% African American	58%	Random sample of Medicaid recipients	Cross-sectional	Prevalence of obesity	NS
Fagioliini et al., 2003	BD	175	9.7% Non-White	57%	Clinic sample	Cross-sectional	Prevalence of obesity	NS
Kemp et al., 2013	BD	264	17.4% African American 4.5% Asian American 3.8% "Other"	56%	Outpatient clinic sample (LiTMUS Study)	Multisite cross-sectional	Prevalence of overweight and obesity	African Americans
Schneiderhan et al., 2009	Sch, SzAD, BD, MDD	92	53% African American 17% Hispanic 7% Asian American	53%	Outpatient clinic sample	Cross-sectional	Prevalence of obesity, abdominal obesity and hypertension	African Americans
Strassnig et al., 2003	Sch, SzAD, PDNOS	146	45.9% African American	47%	Outpatient clinic sample	Cross-sectional	Mean BMI	NT
Susce et al., 2005	Sch, SzAD, BD, MDD	560	17% African American 2% Other	46%	In- and out-patient clinical sample	Cross-sectional	Odds of obesity Odds of hyperlipidemia	African Americans with mood disorders (obesity) Whites (hyperlipidemia) NOTE: Race was not a significant predictor of HTN or DM.
Vieweg et al., 2004	Sch, SzAD, MMD, OD	95	61% African American 1% Other	23%	Inpatient clinic sample	Longitudinal observational	Mean BMI	NT
<i>WEIGHT GAIN</i>								

Author	Psychiatric diagnosis	Sample size (N)	Proportion of racial/Ethnic minorities	% Female	Type of sample	Data type	Measure	Group with more adverse outcome(s)
Basson et al., 2001	Scz, SzAD, SzPh	2335	19%–24% Non-White	36%	Clinic sample	Longitudinal, retrospective RCTs of olanzapine,	Mean weight change over 6 weeks	Non-Whites
de Leon et al., 2007	Scz, SzAD	50	12% African American	Not reported	Inpatient clinic sample	Longitudinal treatment study of clozapine	Mean weight gain	African Americans
Fagiolini et al., 2002	BD	50	8% Non-White	56%	Clinical sample	Longitudinal treatment study	Prevalence of overweight/obesity Weight gain	NT
Littrell et al., 2003	Scz, SzAD	70	26% African American	61%	Outpatient clinic sample	Longitudinal treatment study of olanzapine	Mean weight gain	NS
DIABETES MELLITUS								
Basu et al., 2006	SSD	1,784,142 (25,898 with SSD)	SSD: 33% African American General Pop: 19% African American	SSD: 49% General: 65%	Probability sample of nationally-representative National Hospital Discharge Survey	Longitudinal study of atypical antipsychotic use and DM prevalence	Prevalence of DM Change in DM over time	African American females
Cassidy et al., 1999	BD	345	40% African American	52%	inpatients Inpatient clinic sample	Cross-sectional	Prevalence of DM	NS
Dixon et al., 2000	SSD	Medicare: 14,182 Medicaid: 6066 Field trial: 719	Medicare: 20% African American 6% Other Medicaid: 59% African American 11% Other Field trial: 41% African American 7% Other	Medicare: 46% Medicaid: 64% Field trial: 37%	Random sample of Medicare recipients Random sample of Medicaid recipients Random sample of inand out-patients in public, private, and VA care	Cross-sectional	Prevalence of DM	African Americans and Other Non-Whites
Henderson et al., 2000	Scz, SzAD	82	9% Non-White	27%	Outpatient clinic sample	Longitudinal treatment study of clozapine	Incident DM Weight gain	NT
Kreyenbuhl et al., 2006	SSD, MMD	143	42.8% Non-White	48%	Public, private, and VA outpatient clinic samples	Cross-sectional	Prevalence of DM	Non-Whites with schizophrenia Whites with MMD
Lambert et al., 2005	Scz	Cases: 3663 Controls: 14,523	16%–21% African American 1%–2% Hispanic 1% Other 25%–29% Unknown	an 53%	California Medicaid recipients	Longitudinal treatment study of atypical and conventional antipsychotics	Odds of incident DM	African Americans and Hispanics
Ramaswamy et al., 2007	Scz, BD, MDD, OD	140,951	15% African American 8%–9% Other 21%–26% missing data	53%	California Medicaid recipients	Longitudinal treatment study of atypical antipsychotics	Odds of incident diabetic ketoacidosis	African Americans
Regenold et al., 2002	MDD, BD, Scz, SzAD, OD	243	51% African American	~65%	Inpatient clinic sample	Cross-sectional	Prevalence of DM	NT
Sernyak et al., 2003	Scz	121	10% African American	5%	VA clinic sample	Longitudinal treatment study of clozapine	Prevalence of undiagnosed hyperglycemia or DM	NS
METABOLIC SYNDROME								
Basu et al., 2004	SzAD – B subtype	D 33	36% African American 3% Other	49%	Clinic sample	Cross-sectional	Prevalence of MS	NS

Author	Psychiatric diagnosis	Sample size (N)	Proportion of racial/Ethnic minorities	% Female	Type of sample	Data type	Measure	Group with more adverse outcome(s)
Bermudes et al., 2006	Sez, SzAD, PDNOS, BD, MDD	102	49% African American	50%	Inpatient clinic sample	Cross-sectional	Prevalence of MS	Whites
Cardenas et al., 2008	BD	98	27.6% African American 7.1% Hispanic 4.1% Asian	8%	VA clinic sample	Cross-sectional	Prevalence of MS	NS
Kato et al., 2004	Sez	48	64.6% Hispanic 10.4% African American	50%	Outpatient clinic	Cross-sectional	Prevalence of MS	Hispanics
Lamberti et al., 2006	Sez, SzAD	93	20.4% Non-White	33%	Outpatient clinic	Cross-sectional	Prevalence of MS	NS
McEvoy et al., 2005	Sez	689	35.2% African American 11.6% Hispanic	26%	National, multisite clinic sample (CATIE)	Cross-sectional	Mean BMI Prevalence of MS	NT
Meyer, Rosenblatt et al., 2009	Acute Sez	314	27.4% African American 16.2% Hispanic	~30%	Randomized clinic sample	Longitudinal treatment study of aripiprazole and olanzapine	Prevalence of MS Least squares mean change in MS	NS (baseline prevalence) African Americans/Hispanics (mean change)
Meyer, Nasrallah et al., 2005a	Sez	1231	41.3% Non-White	26%	National, multisite clinic sample (CATIE)	Cross-sectional	Prevalence of MS	Whites (vs. African Americans) (No difference by Hispanic ethnicity)
Meyer, Pandina et al., 2005b	Sez, SzAD	121	45.5% Non-White	51%	National, multisite clinic sample	Cross-sectional	Prevalence of MS	Whites
Patel et al., 2009	Sez, SzAD, SzPh	400	43% African American 6% “Other”	27%	English-speaking inpatient, outpatient, and emergency department sample (CAFE study)	Multisite longitudinal randomized clinical trial of antipsychotic medication treatment for early psychosis	Treatment-emergent MS	Whites and people of “Other” race (vs. African Americans)
Rivas-Vazquez et al., 2011	Sez, MDD, BP, OD	122	79.5% Hispanic (of which 95% were Cuban-American) 8.2% African American 4.1% “Other”	18%	Homeless clinic consecutive sample	Cross-sectional	Prevalence of MS	Cuban–Americans
Straker et al., 2005	SSD, MMD, OD	89	28.1% African American 12. % Hispanic 7.9% “Other”	50%	Inpatient clinic sample	Cross-sectional	Prevalence of MS	NS
PHYSICAL ACTIVITY								
Daumit et al., 2005	Sez, SzAD, BD, MDD	185	40% Non-White	52%	Outpatient clinic sample	Cross-sectional	Prevalence and odds of physical activity and inactivity	NS
Strassnig et al., 2011	Sez, SzAD, PDNOS	117	54% Non-White	59%	Outpatient clinic sample	Cross-sectional	Mean BMI, maximum minutes spent exercising, maximal oxygen uptake, and rating of perceived exertion	African American females (BMI) African American males (min exercise) All else NS
SMOKING								
de Leon et al., 1995	Sez, SzAD, MMD, OD	360	16% African American	38%	Inpatient clinic census	Cross-sectional	Odds of smoking and heavy smoking	Whites for heavy smoking only

Author	Psychiatric diagnosis	Sample size (N)	Proportion of racial/Ethnic minorities	% Female	Type of sample	Data type	Measure	Group with more adverse outcome(s)
Kelly et al., 2009	Scz, SzAD, PDNOS	1213	~40% African American	38%	Maryland inpatient hospitals	Retrospective longitudinal chart review	Prevalence of smoking	NS
Waxmonsky et al., 2005	BD	1904	7% Non-White	58%	Multisite clinical sample	Cross-sectional	Prevalence of smoking	NS

Scz = Schizophrenia, SSD = Schizophrenia-Spectrum Disorders, SzAD = Schizoaffective Disorder, SzPh = Schizophreniform Disorder, BD = Bipolar Disorder, MMD = Major Mood Disorder, MDD = Major Depressive Disorder, OD = Other Disorder, PDNOS = Psychotic Disorder Not Otherwise Specified, SPMI = Serious and Persistent Mental Illness, BMI = Body Mass Index, CVD = Cardiovascular Disease, DM = Diabetes Mellitus, HTN = Hypertension, MS = Metabolic Syndrome, NS = Not significant at $\alpha = 0.05$; NT = Not Tested for statistical significance.

Data Type refers to the data presented in this review. While a study may have been longitudinal, if the data on outcome by race/ethnicity were point prevalences, the study was referred to as cross-sectional in this table.

Some studies report findings for more than one CVD risk factor. Articles are listed under their main outcome and other findings are included in the same row.